INTRODUCTION TO NEUROLOGY.

Nerve cells have the ability to perceive, conduct and transmit nerve impulses. They synthesize the mediators involved in their conduct (neurotransmitters): acetylcholine, catecholamines, indolamines, as well as lipids, carbohydrates and proteins. Some specialized nerve cells have the ability to neurocrinia (synthesize protein products - octapeptides, such as antidiuretic hormone, vasopressin, oxytocin in the cells of the supraoptic and paraventricular nuclei of the hypothalamus). Other neurons entering into the structure of the basal parts of the hypothalamus, they develop the so-called releasing factors, which affect the function of the adenohypophysis.

All neurons are characterized by a high metabolic rate, so they need a constant supply of oxygen, glucose and other substances.

The body of the nerve cell has its own structural features, which are due to the specificity of their function.

The body of the neuron, in addition to the outer shell, has a three-layer cytoplasmic a membrane consisting of two layers of phospholipids and proteins. The membrane performs a barrier function, protecting the cell from the intake of foreign substances, and transport, providing the intake of substances necessary for its vital activity into the cell. Distinguish between passive and active transport of substances and ions across the membrane. Passive transport is the transfer of substances in the direction of decreasing the electrochemical potential, along the concentration gradient (free diffusion through the lipid bilayer, facilitated diffusion - transport of substances through membrane). Active transport - transport of substances against an electrochemical gradient potential using ion pumps. Pitosis is also distinguished - a mechanism for the transfer of substances through membrane of the cell, which is accompanied by reversible changes in the structure of the membrane. Across the plasma membrane is not only regulated by the intake and output of substances, but also information is exchanged between the cell and the extracellular environment. Nerve membranes cells contain many receptors, the activation of which leads to an increase intracellular concentration of cyclic adenosine monophosphate (cAMP) and cyclic guanosine monophosphate (cGMP), which regulate cell metabolism.

The neuron nucleus is the largest of the cellular structures visible under light microscopy. In most neurons, the nucleus is located in the center of the cell body. In the plasma of the cell chromatin granules are located, representing a complex of deoxyribonucleic acid (DNA) with the simplest proteins (histones), non-histone proteins (nucleoproteins), protamines, lipids, etc. Chromosomes become visible only during mitosis. In the center of the core the nucleolus is located, containing a significant amount of RNA and proteins, it forms ribosomal RNA (rRNA).

The genetic information contained in chromatin DNA is transcribed in messenger RNA (mRNA). Then mRNA molecules penetrate through the pores of the nuclear membrane and enter the ribosomes and polyribosomes of the granular endoplasmic reticulum. There protein molecules are synthesized; in this case, amino acids are used, brought special transport RNAs (tRNAs). This process is called broadcast. Some substances (cAMP, hormones, etc.) can increase the rate of transcription and translation.

The nuclear envelope consists of two membranes - inner and outer. The pores through which

exchange between nucleoplasm and cytoplasm takes place, occupy 10% of the nuclear surface shell. In addition, the outer nuclear membrane forms protrusions, from which arise cords of the endoplasmic reticulum with ribosomes attached to them (granular reticulum). The nuclear membrane and the membrane of the endoplasmic reticulum are morphologically close to each other.

In the bodies and large dendrites of nerve cells with light microscopy are clearly visible lumps of basophilic substance (substance or substance of Nissl). With electron microscopy revealed that the basophilic substance is a part of the cytoplasm saturated flattened cisterns of the granular endoplasmic reticulum containing numerous free ribosomes and polyribosomes attached to membranes. Abundance rRNA in ribosomes determines the basophilic color of this part of the cytoplasm, visible when light microscopy. Therefore, the basophilic substance is identified with the granular endoplasmic reticulum (ribosomes containing rRNA). The size of the basophilic lumps granularity and their distribution in neurons of different types are different. It depends on the state impulse activity of neurons. In large motor neurons, lumps of basophilic

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the substances are large and the tanks are located in it compactly. In granular endoplasmic reticulum in ribosomes containing rRNA, new proteins of the cytoplasm are continuously synthesized.

These proteins include proteins involved in the construction and repair of cell membranes, metabolic enzymes, specific proteins involved in synaptic conduction, and enzymes that inactivate this process. Proteins newly synthesized in the cytoplasm of a neuron enter the axon (as well as the dendrites) to replace the spent proteins.

If the axon of the nerve cell is not cut too close to the perikaryon (so as not cause irreversible damage), then there is a redistribution, reduction and temporary the disappearance of the basophilic substance (chromatolysis), and the nucleus moves to the side. At regeneration of the axon in the body of the neuron, there is a movement of the basophilic substance along towards the axon, the amount of granular endoplasmic reticulum increases and the number of mitochondria, protein synthesis is enhanced and at the proximal end of the cut axon, the appearance of processes is possible.

Lamellar complex (Golgi apparatus) - a system of intracellular membranes, each of which is a series of flattened cisterns and secretory vesicles. This system cytoplasmic membranes are called agranular reticulum due to the absence attached to her cisterns and vesicles of ribosomes. The plate complex takes part in the transport of certain substances from the cell, in particular proteins and polysaccharides. Significant part of the proteins synthesized in ribosomes on the membranes of granular endoplasmic reticulum. entering the lamellar complex, it turns into glycoproteins, which unpacked into secretory vesicles, and then released into the extracellular environment. it indicates the presence of a close connection between the lamellar complex and the membranes of the granular endoplasmic reticulum.

Neurofilaments can be found in most large neurons where they are located in the basophilic substance, as well as in myelinated axons and dendrites. Neurofilaments for their structure are fibrillar proteins with not fully understood function.

Neurotubules are visible only with electron microscopy. Their role is maintaining the shape of the neuron, especially its processes, and participation in axoplasmic transport

substances along the axon.
Lysosomes are vesicles bounded by a simple membrane and
cells providing phagocytosis. They contain a set of hydrolytic enzymes capable of
hydrolyze substances trapped in the cell. In case of cell death, the lysosomal membrane
breaks and the process of autolysis begins - hydrolases released into the cytoplasm break down
proteins, nucleic acids and polysaccharides. A normally functioning cell is reliably protected
protected by a lysosomal membrane from the action of hydrolases contained in lysosomes.
Mitochondria are structures in which enzymes of oxidative
phosphorylation. Mitochondria have outer and inner membranes and are located along
throughout the cytoplasm of the neuron, forming clusters in the terminal synaptic
extensions. They are
Xia peculiar energy stations of cells in which synthesized
adenosine triphosphate (ATP) is the main source of energy in a living organism. Thanks to
mitochondria in the body carry out the process of cellular respiration. Components of tissue
the respiratory chain, as well as the ATP synthesis system, are localized in the inner membrane
mitochondria.
Among other various cytoplasmic inclusions (vacuoles, glycogen, crystalloids,
iron granules, etc.) there are also some pigments of black or dark brown
colors similar to melanin (in the cells of the substantia nigra, blue spot, dorsal
motor nucleus of the vagus nerve, etc.). The role of pigments has not yet been fully
elucidated. but
it is known that a decrease in the number of pigmented cells in the substantia nigra is associated
with a decrease
the decrease in the content of dopamine in its cells and caudate nucleus, which leads to
parkinsonism syndrome.
The axons of nerve cells are enclosed in a lipoprotein sheath, which begins at
some distance from the cell body and ends at a distance of 2 microns from the synaptic
endings. The membrane is located outside of the axon border membrane (axolemma). She like
and the cell body shell, consists of two electron-dense layers, separated by less electronically
a dense layer. Nerve fibers surrounded by such lipoprotein sheaths are called
myelinated. With light microscopy, it was not always possible to see such
An “insulating” layer around many of the peripheral nerve fibers that have been

classified as non-myelinated (non-myelinated). However, electron microscopic
studies have shown that these fibers are also enclosed in a thin myelin
(lipoprotein) membrane (thin myelinated fibers).
Myelin sheaths contain cholesterol, phospholipids, some cerebrosides and
fatty acids, as well as protein substances intertwined in the form of a network (neurokeratin).
The chemical nature of myelin of peripheral nerve fibers and myelin of the central nervous
the system is somewhat different. This is due to the fact that in the central nervous system,
myelin
is formed by oligodendroglial cells , and in the peripheral - by lemmocytes. These two types of
myelin
also have various antigenic properties, which is revealed in infectious-allergic
the nature of the disease. The myelin sheaths of nerve fibers are not continuous, but are
interrupted along
fibers at intervals, which are called knot interceptions (Ranvier interceptions). Such
interceptions exist in nerve fibers of both the central and peripheral nervous systems,
although their structure and frequency in different parts of the nervous system are
different. Branching out

from the nerve fiber usually falls on the site of interception of the node, which corresponds to the place of lavage
Cania of two lemmocytes. At the end of the myelin sheath at the level of interception of the node there is a slight narrowing of the axon, the diameter of which decreases by 1/3.
Myelination of the peripheral nerve fiber is carried out by lemmocytes. These
cells form a process of the cytoplasmic membrane, which spirals around
nerve fiber. Up to 100 spiral layers of myelin can form in the correct layout.
stranded structure. In the process of wrapping around the axon, the cytoplasm of the lemmocyte is displaced to
its core; this ensures the convergence and close contact of adjacent membranes. Electronic microscopically, the myelin formed by the shell consists of dense plates thick
about 0.25 nm, which are repeated in the radial direction with a period of 1.2 nm. Between them there is a light zone, divided in two by a less dense intermediate plate, which has
irregular outlines. The light zone is a highly water-saturated space.
between the two components of the bimolecular lipid layer. This space is available for
circulation of ions. "Non-fleshy" so-called non-myelinated fibers of the vegetative
of the nervous system are covered with a single spiral of the lemmocyte membrane.
The myelin sheath provides an isolated, non-decremental (no fall
amplitude of potential) and faster conduction of excitation along the nerve fiber. There is direct relationship between the thickness of this shell and the speed of the impulses. Fiber with a thick layer of myelin conduct impulses at a speed of 70-140 m / s, while the conductors with a thin myelin sheath at a speed of about 1 m / s and even slower - “fleshy” fibers (0.3-0.5 m / s).
The myelin sheaths around axons in the central nervous system are also multi-layered.
and are formed by processes of oligodendrocytes. The mechanism of their development in the central nervous system
similar to the formation of myelin sheaths in the periphery.
In the cytoplasm of the axon (axoplasm) there are many filamentous mitochondria,
axoplasmic vesicles, neurofilaments and neurotubules. Ribosomes in axoplasm
are very rare. The granular endoplasmic reticulum is absent. This leads
to the fact that the body of the neuron supplies the axon with proteins; therefore glycoproteins and a number
macromolecular substances, as well as some organelles such as mitochondria and various bubbles must move along the axon from the cell body. This process is called axonal. or axoplasmic transport.
Certain cytoplasmic proteins and organelles move along the axon in two
streams at different speeds. One is a slow stream moving along an axon at a speed of 1-3 mm / day, moves lysosomes and some enzymes necessary for the synthesis of neurotransmitters
at the endings of the axons. Another stream - fast, also directed from the cell body, but its speed is 5-10 mm / h (about 100 times the slow flow rate). This stream
transports components necessary for synaptic function (glycoproteins,
phospholipids, mitochondria, dopamine hydroxylase for adrenaline synthesis). Mechanism axoplasmic current is not fully understood.
Dendrites are usually much shorter than axons. In contrast to the axon, dendrites are dichotomous
branch out. In the central nervous system, dendrites do not have a myelin sheath. Large dendrites differ from
axon also in that they contain ribosomes and cisterns of granular endoplasmic
reticulum (basophilic substance); there are also many neurotubules, neurofilaments and

mitochondria. Thus, dendrites have the same set of organelles as the body of a nerve cell. The surface of the dendrites is significantly increased due to small outgrowths (spines), which serve as sites of synaptic contact.

The parenchyma of brain tissue includes not only nerve cells (neurons) and their processes, but also neuroglia and elements of the vascular system.

Light microscopy reveals several types of neuroglial cells lying next to with neurons and their processes. Glia of ectodermal origin consists of oligodendrocytes, fibrous and plasma astrocytes and ependyma cubic cells. The latter line ventricles and central canal of the brain and spinal cord. Smaller glial cells forming microglia, are of mesodermal origin and can turn into phagocytes.

Neuroglia is of great importance in ensuring the normal functioning of neurons. She is in close metabolic relationship with neurons, taking part in synthesis protein and nucleic acids and information storage. In addition, neuroglial cells are internal support for the neurons of the central nervous system - they support the body processes neurons, ensuring their proper relationship.

Special functions are also attributed to certain types of glia. Oligodendrogliocytes participate in the formation and maintenance of myelin sheaths. In the nuclei of these cells, one can clearly see lumps of chromatin, and in the cytoplasm there are many granular endoplasmic reticulum and mitochondria. Oligodendrogliocytes are located mainly around neurons. Astrocytes practically do not contain granular endoplasmic reticulum and have little mitochondria. These cells are usually located between capillaries and neurons, as well as between capillaries and ependyma cells. Astrocytes are credited with an important role in metabolism between neurons and the circulatory system. In most parts of the brain, surface membranes the bodies of nerve cells and their processes (axons and dendrites) do not touch the walls blood vessels or cerebrospinal fluid of the ventricles, central canal and subarachnoid space. The exchange of substances between these components, as a rule, is carried out through the so-called blood-brain barrier. Blood-borne substances must pass primarily through the cytoplasm of the vascular endothelium. This barrier is no different from the endothelial cell barrier in general. Then they need to pass through the basement membrane capillary, a layer of astrocytic clay and, finally, through the surface membranes of the neurons themselves.

The latter two structures are believed to be the main components of the blood-brain barrier.

In other organs, brain tissue cells are in direct contact with the basal capillary membranes, and the intermediate layer, similar to the astrocytic cytoplasm layer glia, absent. Large astrocytes, which play an important role in the rapid intracellular transfer of metabolites to and from neurons and ensure the selective nature of this renosa probably constitute the main morphological substrate of hematoencephalic barrier. In certain structures of the brain - neurohypophysis, pineal gland, gray tubercle, supraoptic, subforonic and other areas - the metabolism is very quickly. It is assumed that the blood-brain barrier in these brain structures is not is functioning.

Microglia are small cells scattered throughout the white and gray matter of the nervous system. She performs a protective function, taking part in a variety of reactions in response to damaging factors. In this case, microglial cells first increase in volume, then divide mitotically. Astrocytes and oligodendrocytes replace destroyed neurons in the form glial scar.

Ependymal cells are also often referred to as glial cells. They have a core
lamellar complex and granular reticulum. The processes of ependymal cells are located in direct contact with the processes of neurons and glial cells in the gray and white matter of the brain.
Ependymal cells perform a proliferative support function, are involved in the formation choroid plexuses of the ventricles of the brain. On the surface of the ventricles of the brain, the nerve elements are
separated from the cerebrospinal fluid only by the ependyma layer (except in some places). V the choroid plexus also separates the ependyma layer from the cerebrospinal fluid capillaries.
Ependymal
cells
ventricles
brain
perform
function
blood-brain barrier.
Nerve cells connect to each other only through contact - synapse (Greek synapsis

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- contact, seizure, connection). Synapses can be classified by their location
on the surface of the postsynaptic neuron. Distinguish axodendritic synapses - axon ends in a dendrite; axosomatic synapses - contact is formed between the axon and the body neuron; axoaxonal - contact is established between axons. In this case, the axon can synapse only on the unmyelinated part of another axon. It is possible either in
the proximal part of the axon, or in the area of the terminal button of the axon, since in these places
the myelin sheath is absent. There are other variants of synapses: dendrodendritic and dendrosomatic. About half of the entire surface of the neuron body and almost the entire surface its dendrites are littered with synaptic connections from other neurons. However, not all synapses transmit nerve impulses. Some of them inhibit the reactions of the neuron with which they are associated.
(inhibitory synapses), while others located on the same neuron excite it (excitatory synapses). The total action of both types of synapses on one neuron leads to each given moment to balance between two opposite kinds of synaptic effects.
Excitatory and inhibitory synapses have the same structure. Their opposite action due to the release of various chemical neurotransmitters in synaptic endings, having different ability to change the permeability of the synaptic membrane for ions of potassium, sodium and chlorine. In addition, excitatory synapses are more likely to form axodendritic
contacts, and inhibitory - axosomatic and axoaxonal.
The section of a neuron through which impulses enter the synapse is called presynaptic ending, and the site that receives impulses - postsynaptic ending. V
the cytoplasm of the presynaptic terminal contains many mitochondria and synaptic vesicles containing a neurotransmitter. The axlemma of the presynaptic portion of the axon, which
closely approaches the postsynaptic neuron, in the synapse forms a presynaptic membrane. The site of the plasma membrane of the postsynaptic neuron, closely adjacent to the presynaptic membrane is called the postsynaptic membrane. Intercellular
the space between the pre- and postsynaptic membranes is called the synaptic cleft.
The structure of the bodies of neurons and their processes is very diverse and depends on their functions.

Distinguish between receptor neurons (sensitive, autonomic), effector (motor, vegetative) and combination (associative). From a chain of such neurons, reflex arcs. Each reflex is based on the perception of stimuli, its processing and transfer to the reacting body is the executor. The collection of neurons required for exercise reflex is called a reflex arc . Its structure can be both simple and very complex, including both afferent and efferent systems.

Afferent systems - represent the ascending conductors of the spinal and head the brain, which conduct impulses from all tissues and organs. A system that includes specific receptors, conductors from them and their projection in the cerebral cortex, is defined as an analyzer. He performs the functions of analysis and synthesis of stimuli, i.e., the primary decomposition of the whole into parts, units and then the gradual addition of the segmental apparatus of the spinal cord). Numerous descending conductors from these formations of the brain approach the neurons of the segmental apparatus of the spinal cord and then follow to the executive organs: striated muscles, endocrine glands, blood vessels, internal organs and skin.

REFLEXES AND THEIR CHANGES.

The functioning of the nervous system manifests itself in the form of a continuously arising scary responses to irritating factors of the external and internal environment. The functional unit of nervous activity is a **reflex** as a response the reaction of the nervous system to irritation. Reflexes are subdivided into **unconditional and conditional.**

Unconditioned reflexes are inherited, they are inherent in everyone biological species; their arcs are formed by the time of birth and are normal persist throughout life. However, they can change under the influence illness.

Conditioned reflexes arise during individual development and the accumulation of new skills. The development of new temporary connections depends on changing conditions Wednesday. Conditioned reflexes are formed on the basis of unconditioned and with the participation higher parts of the brain.

Arcs of unconditioned reflexes are closed in the segmental apparatus of the spinal cord and trunk brain, but they can be closed and higher, for example, in the subcortical ganglia or in the cortex. Reflector-

Naya arc of segmental reflexes usually consists of 2-3 nerve cells. If the arch of the spinal reflex is formed by two neurons, the first of them is represented by a cell of the spinal ganglion, and the second - by the motor cell (motoneuron) of the anterior horn of the spinal cord. Dendrite the cells of the spinal ganglion have a considerable length, it follows to the periphery in the composition sensitive fibers of the nerve trunks. The dendrite ends with a special device for perception of irritation - by the receptor. The axon of the spinal ganglion cell is part of back spine; this fiber reaches the motoneuron of the anterior horn and with the help of the synapse establishes contact with the cell body or with one of its dendrites. The axon of this neuron enters

the composition of the anterior root, then the corresponding motor nerve and ends a motor plaque in the muscle.
The cell of the spinal ganglion with its processes is called the receptor cell, otherwise afferent, or centripetal, part of the reflex arc, and the motor neuron of the anterior horn - the effector, or centrifugal, part of it. If the reflex arc has 3 neurons, then the third the neuron is interposed between the receptor and effector neurons.
For example, the knee reflex is triggered when a neurological hammer strikes lig.patellae. In response, the quadriceps muscle of the thigh contracts and the lower limbs in the knee joint. The arc of this unconditioned reflex consists of two neurons. She closes at the level Lp - Liv. The arcs of other deep reflexes are closed at the level of various segments of the spinal cord.
A person has a large number of innate connections, that is, unconditioned reflexes, carried out through the segments of the spinal cord and other parts of the central nervous system. V
so-called tonic reflexes play an important role in ensuring the function of movement.
The muscle and outside the perception of the impulse of active movement is in a state of tension, which is indicated by tone. When a muscle is stretched, its resistance arises as a result upcoming stress. This phenomenon is called the myotatic reflex (gr. Tu- muscle, tasis-tension). Another term has also been proposed - "proprioceptive reflex" (lat. - own), since irritation of the spiral receptor and response reflex contractions are within the same muscle.
New data on electrophysiology have been obtained in experiments on animals motor function, in particular on the issue of muscle tone. It turned out that the structure the motor cells of the anterior horns of the spinal cord are not the same. The largest of them designated as large and small *a* -motoneurons, in addition there are also γ-motoneurons. Large α-motoneurons innervate white muscle fibers capable of performing fast contractions (phasic). Small α-motoneurons innervate the red muscle fibers that play an important role in maintaining tone and posture (tonic).
About 1 /3 cells of the anterior horns make γ -motoneyrony. Axons of *a* - and γ -motoneurons go to the periphery in the anterior roots and peripheral nerves. Axon of the *α*- motoneuron ends with end plates on muscle fibers (α-muscle, or extrafusal

muscle fiber). The axon of the γ-motor neuron approaches the muscle spindles. This is how receptor apparatuses, the functional significance of which was elucidated relatively recently. Thin muscle fibers (γ-muscle, or intrafusal) are laid at both ends of the spindle, the axons of γ-motor neurons end on them. In the middle (equatorial) part of the spindle, the spiral receptor of the spinal ganglion cell is developed. The γ-motor neuron impulse causes contraction of the muscle elements of the spindle. This leads to a stretching of its equatorial the area and irritation of the receptor fibers located here - the endings of the cell dendrites spinal ganglia - fibers 1a. The excitation is transferred to the α-motor neuron, and there is tonic muscle tension.
There are central (suprasegmental) connections with the γ-motoneurons of the spinal cord. It is assumed that they somehow regulate the formation of the stretch reflex. Probably such connections begin in the network formation of the brain stem, in the cerebellum, in the ganglia extrapyramidal system. The possibility is not excluded that such a role can be played by pyramidal fibers. Part of the dendrites of the nerve cells of the spinal ganglia - fibers 1b - ends not in the muscle spindle, but in the tendon receptors (Golgi tendon organs). They are receptors for conducting impulses that inhibit the activity of α-motoneurons. The axons of these sensory neurons end at intercalary cells that contact with *a* -motoneurons.
The force created by the tensing muscle stimulates these receptors.

The latter have a high threshold and are excited only when significant
muscle effort. The resulting action potentials are conducted into the spinal cord and
cause inhibition of α-motoneurons. Inhibition of motoneurons of the anterior horns
accompanied by relaxation of synergistic muscles, protecting them from excessive
overexertion, and simultaneous contraction of antagonist muscles.
Neurophysiologists and neurohistologists have received new
data on the structure and function of the so-called proprioneurons (interneurons) of the spinal cord,
that is, nerve cells that are not involved in the formation of the anterior roots. Renshaw described the special
the nerve cells now called after him are Renshaw cells . These cells are located in front of
them in the horns of the spinal cord and have an inhibitory and sometimes facilitating effect on β-
motor neuron. Before leaving the spinal cord, the axon of the *α*- motoneuron gives a return collateral
to the Renshaw cage. With excessive excitation of the α-motoneuron, the Renshaw cell exerts on it
braking action (so-called return braking). Regarding the role of the mentioned
relief in relation to the α-motor neuron, then it changes the interaction of the muscles
agonists and antagonists, it weakens the action of the antagonist. Intrafusal muscle
fibers can contract under the influence of γ-motoneurons. This increases the excitability of sleep
spindle receptor and increases the muscle stretch reflex. Influenced by cells
Renshaw changes the excitability of α-motor neurons, which can also affect the degree of expression
stretch reflex. Both of these phenomena can affect muscle tone, which
when the peripheral α-motor neuron is damaged, it decreases. Obviously it depends on the violation
the extension reflex arc, since the *α*- motoneuron is simultaneously an effector
part of the arc of this reflex. Muscle tone also decreases with damage to the posterior roots, i.e.
at a break in any part of the arc of the segmental reflex.

In clinical practice, reflexes are divided according to the location of the receptor moat into superficial (skin, from mucous membranes) and deep (reflexes stretching the muscles).

Deep reflexes were initially mistaken for a response to direct stimulation
muscles when hitting a tendon with a hammer. Later, the point of view about
the reflex nature of this phenomenon.
It was found that tendon reflexes are inherently identical to reflexes to
stretching. When struck with a hammer, not only the tendon is stretched, but also the
muscles. It is in response to stretching that a reflex occurs. Muscle fiber lengthening by
a few microns are enough to induce it. If stretching is done slowly, the reflex
acquires a tonic character. The degree of muscle tension occurring at this moment
is a criterion for measuring its tone. For short and abrupt irritation with a hammer

the reflex becomes "phasic", the response movement has the character of a single short contraction
muscle schenia.
Currently, there is a need to take "tendon" reflexes for one of the
varieties of stretch reflexes. The same applies to the so-called periosteal, or
periosteal, reflexes. These reflexes are reproduced by the same segmental arc,
consisting of two neurons - cells of the spinal ganglion and α-motoneuron. The main excite
lem "tendon" reflex, according to modern concepts, is the stretching of muscle

spindle. This stretching leads to irritation of the spindle in the bag.
spiral receptor cells of the spinal ganglion. Therefore, such reflexes should be called
not tendon, but myotatic, or deep. With the formation of a stretch reflex
muscle, there is a conjugated action of α- and γ-neurons of the anterior horns of the spinal
cord. Under
the influence of suprasegmental drives, γ-neurons can trigger intrafusal
contractile elements, which facilitates the formation of a reflex.

The following deep reflexes are examined on the upper limbs.

Reflex from the tendon of the biceps brachii (biceps reflex) causes
It occurs when a hammer strikes the tendon of this muscle above the elbow
joint. Have
the examined person's upper limb is slightly bent in this joint. In response to the blow
muscle contraction and slight flexion of the upper limb in the elbow occur
joint. This reflex is called elbow flexion. Its arc closes on
the level of Cv-Cvi segments of the spinal cord, afferent and efferent fibers of the arc
reflexes are part of the musculocutaneous nerve.

Reflex from the tendon of the triceps brachii muscle (triceps reflex) causes
is struck by a hammer blow on the tendon of this muscle 1-1.5 cm above the ulnar
process of the ulna, muscle contraction and extension of the upper
limbs in the elbow joint (extensor-elbow reflex). The ways
evocation: 1) the upper limb of the subject is supported in the elbow joint
with the examiner's brush, the forearm hangs down freely; 2) examiner
supports the bent arm of the examinee by the elbow region. Reflex arc
- sensory and motor fibers of the radial nerve, closes at Cvp-Cvsh.

Carpal-ray (carporadial reflex). When struck with a hammer on
the styloid process of the radius occurs flexion in the elbow joint and
pronation of the forearm. Starting position: the upper limb is bent in
elbow joint at an angle of about 100 °, the hand is held by the examiner on average
position between pronation and supination. This reflex can be explored in
the position of the patient lying on his back. The reflex arc closes at the Cv-Cvin
level,
fibers are part of the median, radial and musculocutaneous nerves. Carpal
the ray reflex (it is mistakenly called the metacarpal-ray reflex) is referred to as
periosteal reflexes. It is believed to be caused by irritation of the receptors,
embedded in the periosteum, but, most likely, this reflex refers to
myotatic and arises from stretching of the brachioradial, round pronator and
biceps brachii.

Deep abdominal reflexes are triggered by tapping with a hammer on
the pubic region 1-1.5 cm to the right and left of the midline; in response it turns out
contraction of the muscles of the corresponding side of the abdominal wall. Reflex arc
closes in the segments T VP - Txp.

Knee reflex - extension of the lower limb in the knee joint with
hitting the tendon of the quadriceps femoris muscle below the patella. Su-
There are several ways to study the knee reflex:
the patient must put the lower limb on the other or sit in such a way

so that his legs hang freely and the angle of flexion of the lower limbs at the knee joints was 90 °. You can sit the subject so that the feet are free
leaned on the floor, and the lower limbs were bent at the knee joints under the blunt angle. In the position of the patient lying on his back, the doctor brings his left hand under
knee joints of the subject and position them so that the angle of flexion in
the knee joints were blunt, and the heels rested freely on the bed.
Knee reflexes in some healthy people turn out to be
inhibited and difficult to evoke. In such cases, they resort to
Endrashika: the patient is offered to clasp the fingers of both hands and pull the hands with force
to the side. The Endrashik effect is explained by the activating effect of γ- neurons on intrafusal muscle fibers.
To facilitate the induction of knee reflexes, the patient's attention is distracted by:
he is asked questions, offered to count in his mind, take deep breaths, etc.
The arch of the knee reflex: sensory and motor fibers of the femoral
nerve, spinal cord segments L P -Liv.
Achilles reflex - contraction of the calf muscles and plantar flexion
feet in response to a hammer blow to the heel (Achilles) tendon. For
examining the patient in the supine position examining the left hand
captures the foot, bends the lower limb at the knee and hip
joints, produces dorsiflexion of the foot. In the position of the patient lying on
abdomen, his lower limbs are bent at right angles at the knees and
ankle joints; with one hand the examiner holds the foot, with the other
hits the heel tendon. The subject kneels on a chair or
the couch so that his feet hang freely, and in this position, a blow is made
a hammer on the heel tendon. Reflex arc: sensitive and
motor fibers of the tibial nerve, spinal cord segments Si - Sp.
In addition to deep reflexes, patients are examined for superficial (skin) reflexes.
Abdominal reflexes: the upper one is caused by streaky skin irritation
abdomen parallel to the costal arch, middle - the same irritation in
horizontal direction at the level of the navel, lower - parallel to the groin
fold. In response, the abdominal muscles contract on the side of the same name.
The examinee lies on his back with freely extended lower limbs. At
laxity of the skin in the abdomen in multiparous women, with obesity, in persons in in old age, when examining abdominal reflexes, it is recommended to tighten
abdominal skin. The reflex arc runs through the following spinal segments:
upper abdominal reflex - Tvi-Tvsh; medium - Tix-Tx; lower - Txi-Txp.
Abdominal reflexes appear in children from 5-6 months (in newborns these
reflexes are not triggered).
Cremasterny (testicular) reflex - contraction of the muscle that lifts
testicle, with streak irritation of the skin of the inner surface of the thigh. Arc
the reflex is closed in Li-Lp; sensory and motor fibers are included in
the composition of the femoral genital nerve. This reflex appears in boys aged 4
5 months

Plantar reflex - plantar flexion of the toes in response to
streak irritation of the outer edge of the sole. The spinal arch of this reflex
closes in the Lv-Sp segments and passes through the sciatic nerve. This reflex

begins to be caused only in children over the age of 2 years; it appears in connection with
the ability to maintain body position while standing and walking.
Anal reflex. When applying injections near the anus, reduce
its circular muscle (external sphincter of the anus). Arc diagram
anal reflex Siv-Sv, n. aposoccygei, n. pudendus.
**In addition to skin surface reflexes, it is quite informative
a study of reflexes from mucous membranes (corneal,
conjunctival, palatine and pharyngeal).** When evoking reflexes, it is necessary
to achieve from the patient the fullest possible relaxation of the investigated
limbs. Hammer blows should be applied with equal force. When evaluating
reflexes pay attention to their severity and symmetry. That's why
it is necessary to study the same reflexes on the right and on the left and immediately carry out them
comparison. The severity of reflexes in healthy people can individually
fluctuate, in particular a symmetrical position or revitalization of reflexes when
complete preservation of muscle strength can be regarded as a variant of the norm.
The following changes in reflexes are observed: decrease or loss (with
damage to the reflex arc), increase and perversion (with damage
pyramidal system and disinhibition of the segmental apparatus of the spinal cord).

ARBITRARY MOVEMENTS AND THEIR VIOLATIONS

Movement is a universal manifestation of life, providing
the possibility of active interaction of both the constituent parts of the body and the whole
organism with the environment. All human movements can be divided into
reflex and voluntary.
Reflex motor reactions are unconditioned and arise in response to painful,
light, sound and other irritations, including muscle strains. Besides such simple
reflex motor reactions, there are also complex reactions in the form of a series of consecutive
purposeful movements. Reflex mechanisms play an important role in ensuring
motor functions and regulation of muscle tone. These mechanisms are based on
The simplest stretch reflex is the myotatic reflex.
Arbitrary movements arise as a result of the implementation of programs that are formed in
motor functional systems of the central nervous system. These movements are carried out when reducing
muscle agonists and synergists and the simultaneous relaxation of antagonists. In this way
not only movements of the limbs are provided, but also more complex motor acts:
walking, exercise, speaking and writing, etc.
The effector divisions of voluntary motor systems are represented by many
anatomical structures. The most direct path from the cortex to the periphery consists of two

nerve cells. The body of the first neuron is located in the cortex of the precentral gyrus. Its accepted
called the central (upper) motor neuron. Its axon is headed for education
synapse with the second - peripheral (lower) motor - neuron . This two-neural
pathway connecting the cerebral cortex with the skeletal (striated)
musculature, clinicians call cortical-muscular. The totality of all central movements
body neurons are called the pyramidal system . The sum of the elements of the second link, i.e.
peripheral neurons, constitutes the motor effector part of the segmental apparatus
brain stem and spinal cord. The pyramidal system by means of a segmental apparatus and
muscle activates the CNS program. When the program is repeated, an arbitrary
movement can become stereotyped and turn into automatic, switching from
pyramidal system to extrapyramidal.
In phylogenetic terms, the pyramidal system is a young formation that
especially developed in humans. The segmental apparatus of the spinal cord in the process
evolution appears early, when the brain is just beginning to develop, and the cortex of large
hemispheres are not yet formed. The pyramidal system means a complex of nervous
cells with their axons, through which the connection of the cortex with the segmental apparatus
is formed. Body
these cells are located in the V layer of the precentral gyrus and in the paracentral lobule
(cytoarchitectonic field 4), are large (40-120 microns) and triangular in shape.
They were first described by the Kiev anatomist V.A. Bets in 1874, they are called
giant pyramidal neurons (Betz cells). There is a clear somatic ra-
the definition of these cells. Located in the upper part of the precentral gyrus and in
paracentral lobule cells innervate the lower limb and trunk, located in
its middle part is the upper limb. At the bottom of this gyrus are neurons,
sending impulses to the linden, tongue, pharynx, larynx, masticatory muscles. Effective
the cortical centers of the muscles of the limbs, face and neck are located in the area of the
precentral gyrus
in the reverse order of the body diagram, that is, the cells that control the movements of the head
are shown below,
face, above - the upper limbs, and in the upper and medial parts - the lower limbs.
Another feature of the motor areas of the cortex is that the area of each of them
depends not on muscle mass, but on the complexity and subtlety of the function
performed. Especially great
the area of the motor area of the hand and fingers of the upper limb, in particular the large, and
lips, tongue.
Recently, it has been proven that giant pyramidal neurons are present not only in
precentral gyrus (field 4), but also in the posterior parts of the three frontal gyrus (field 6), as
well as in
other fields of the cerebral cortex. The axons of all these nerve cells go down and inward,
approaching each other. These nerve fibers make up the corona radiata. Then
pyramidal conductors are collected in a compact bundle that forms part of the inner capsule
(capsula interna). This is the name of the narrow plate of white matter, which is located in front
between the head of the caudate and the lenticular nucleus (anterior leg of the inner capsule) . A
place
connection of these two legs at an angle, open laterally, makes up a sleek internal
capsules. The fibers of the pyramidal system form the knee and the adjacent part of the back
legs. The knee is formed by fibers directed to the motor nuclei of the cranial
nerves (cortical-nuclear), the posterior leg - with bundles of fibers to the spinal segmental
apparatus

(cortical-spinal), in front of the bundle lies for the upper limb, and behind - for the lower limb. From
the inner bursa axons of giant pyramidal neurons pass into the base of the brain stem, occupying the middle part of it. Cortical-nuclear fibers are located medially, cortical-spinal - laterally. In the pons of the brain, the pyramidal tract also runs at its base, dividing into separate bundles. Within the brain stem, part of the cortical-nuclear fibers goes to the opposite side, after which they form synapses with motor neurons the body nuclei of the corresponding cranial nerves.
Another part of the cortical-nuclear fibers remains on their side, forming synaptic connections with the cells of the nuclei on the same side. Thus, a bilateral cortical innerve for the oculomotor, masticatory muscles, for the upper facial muscles, for the muscles of the pharynx
and larynx. Corticonuclear fibers for the muscles of the lower half of the face and for the muscles of the tongue are almost
completely move to the opposite side (these two muscle groups receive
innervation only from the cortex of the opposite hemisphere).
Cortical-spinal fibers of the pyramidal system at the level of the caudal pons the brain converge and on the ventral part of the medulla oblongata form two visible macroscopically roller (pyramid of the medulla oblongata). Hence the designation "Pyramid system". On the border of the medulla oblongata with the dorsal fibers of the pyramidal bundle
go to the opposite side and a cross is formed
pyramids (decussatio
pyramidum). Most of the fibers that have passed to the opposite side go down into lateral cord of the spinal cord, forming a lateral or crossed pyramidal bundle.
A small part of the pyramidal fibers (about 20%) remains on their side and passes down into anterior cord (straight or non-crossed pyramidal bundle). Quantitative
the ratio of crossed and uncrossed fibers for different parts of the body is not the same. V the upper extremities are sharply dominated by cross innervation.
In the spinal cord, the diameter of both pyramidal bundles gradually decreases. All over along the length of them, fibers extend to the segmental apparatus (to the *a*- motoneurons of the anterior horns
and to intercalary neurons). Peripheral motor neurons for the upper limbs are located in the cervical thickening of the spinal cord, for the lower ones - in the lumbar thickening. In the thoracic region
there are cells for the muscles of the trunk. The axons of the spinal cord motor neurons are directed to
the corresponding muscles in the anterior roots, then the spinal nerves, plexuses and, finally, peripheral nerve trunks. Each motoneuron innervates several we cervical fibers that form a "motor unit".
So, the cortical-nuclear fibers of the pyramidal system move to the opposite side at different levels of the brain stem, and cortical-spinal - on the border of the medulla oblongata with the spinal cord. As a result, each of the cerebral hemispheres controls
the opposite half of the segmental apparatus, and therefore the opposite half muscular system. As mentioned, some of the pyramidal fibers do not participate in the intersections and
comes into contact with peripheral neurons of the side of the same name. Such peripheral neurons receive impulses from both hemispheres. However, bilateral innervation is not the same presented in various muscle groups. It is more pronounced in the muscles innervated cranial nerves, with the exception of the lower half of the facial muscles and tongue. Two-sided The innervation is preserved in the axial muscles (neck, trunk), to a lesser extent - in distal extremities. This long cortical-muscular pathway with various

diseases can be interrupted at any site. This leads to the loss of arbitrary movements in certain muscle groups. Complete loss of voluntary movements is called paralysis (paralysis), weakening - paresis (paresis). Paralysis or paresis occurs with defeat both central and peripheral neurons, however, the signs of paralysis will be different.

Clinical study of voluntary movements includes a number of methods - technical receptions. First, it is ascertained whether the subject can actively produce flexion of the limbs in all joints, whether these movements are performed in full volume. When determining the restriction, the doctor performs passive movements, to exclude local lesions of the osteoarticular apparatus (ankylosis, contractures, etc.). This kind of immobilization does not belong to the category of paralysis.

or paresis. Finding paralysis is not difficult. When identifying paresis pay attention to a decrease in the amount of active movement, for example when flexing and extending a limb in one or another joint. However, such the method of research is only indicative and does not determine degree of paresis. Another way to detect paresis is to study the strength of contraction

different muscle groups. In this way, you can get an idea of the degree paresis and the formula for its distribution. This technique is widely used in clinic.

There are various instruments with which you can quantify to measure the force of contraction of certain muscle groups, but usually they use dynamometer, which measures the force of compression of the hand (in kilograms) when straightening sweet hand. **Approximate force of contraction of various muscle groups can be determined in the so-called manual way.** Counteracting some any elementary voluntary movement performed by the patient, the investigator determines the effort sufficient to stop this movement.

There are two modifications of this technique. At the first, the doctor provides an obstacle to the patient during active movement in various parts of the body and limbs in a certain direction. The examiner's task is determination of the resistance force that can stop the movement, for example when flexing the upper limb at the elbow joint. In most cases use a different modification. The examinee is asked to perform the given active movement and hold the limb with full strength in this new position. The examiner tries to move in the opposite direction and reverses attention to the degree of effort required to do this. For example flexor strength the forearms are determined with full active flexion at the elbow joint. Sick are asked to resist while actively flexing the upper limb. About- the next one clasps the lower part of the forearm with his right hand and, resting left hand in the middle of the patient's shoulder, trying to straighten the upper limb in elbow joint.

The research results are assessed using a six-point system:

muscle strength in full - 5 points;

slight decrease in strength (compliance) - 4 points;

moderate decrease in strength (active movements in full volume during action

gravity on a limb) -3 points;
the ability to move in full only after the elimination of gravity
(the limb is placed on a support) - 2 points;
preservation of movement (with barely noticeable muscle contraction) - 1 point.
In the absence of active movement, if you do not take into account the weight of the limb, strength
the studied muscle group is taken equal to zero. With muscle strength of 4
points speak about mild paresis, 3 points - about moderate, 2-1 - about deep.
When examining muscle strength manually, subjective
evaluating the results. Therefore, with unilateral paresis, one should compare
indicators of symmetrical muscle groups. With the defeat of the upper limbs
use a wrist dynamometer (if possible, a reversible dynamometer,
allows you to accurately measure the strength of the flexor and extensor muscles of the forearm and
shins).
The study of muscle strength is usually performed in the following sequence:
head and cervical spine (head tilts forward, backward, right, left,
side turns), upper and lower limbs (from proximal to
distal), trunk muscles. Muscle Strength Study Results
the upper limb are recorded in the medical history in a form according to which it is easy
navigate the degree and prevalence of movement disorders.
In some cases, more research is needed: definition
electrical excitability of muscles and nerves, electromyography, etc. Determination of strength
individual muscle groups is supplemented by monitoring the patient's motor skills in general (walking, going from a lying position to a sitting position, getting up from a chair and
etc.).

When paralysis or paresis of certain muscle groups is detected
the question arises about the topical diagnosis of the lesion. When determining the lo-
the pathological focus uses the criteria for the distribution of paresis and
concomitant muscle condition. With central and peripheral paralysis
they change unevenly - in the opposite direction.
Evaluation of the phenomena accompanying paralysis begins with muscle tone.
It is determined by palpating the muscles and identifying the degree of their elasticity. except
In addition, the muscle resistance arising during passive movements in
the corresponding joints, the degree of tonic muscle tension is assessed. At
In normal tone, this tension is not great, but clearly noticeable. On palpation
muscle is determined by light elasticity. With severe muscle hypotension
passive movements are performed without resistance. Their volume is increasing.
For example, with a sharp hypotonia of the muscles, the lower limb in the hip
the joint can be bent until the thigh touches the anterior abdominal wall; at

flexion of the upper limb at the elbow joint, the wrist and hand can be
brought to the shoulder joint. On palpation, the muscle is perceived to be hypotonic.
With an increase in muscle tone, passive movements meet
significant resistance, sometimes even formidable. It is expressed only
at the beginning of passive flexion and extension, then the obstacle seems to be removed,
and the limb moves freely (jackknife symptom). This is especially clear
it is determined if passive movements are made quickly. With spastic
paralysis, the lower limbs are often straightened, they can be bent only when
great effort. Paralyzed muscles are palpated more dense.
In the studies of some physiologists, it has been shown that the pyramidal bundle contains
a large number of axons not of giant pyramidal neurons, but of other neurons of the cortex, and
subcortex. The authors associate an increase in muscle tone with a lesion not actually
pyramidal fibers, and their satellites, in particular cortical-reticular fibers (they pass from
cells of the premotor cortex to the reticular formation of the brainstem and further to γ-motor neurons
the segmental apparatus of the spinal cord). Therefore, the term "pyramidal hypertension" is
inaccurate, obviously, it is more correct to call the increase in tone while muscle spasticity.
For "pyramidal" hypertension, an increase in tone is characteristic mainly in certain
muscle groups. On the upper limbs, these are pronators and flexors of the forearm, hand,
fingers, on the lower extremities - the extensors of the lower leg, flexors of the foot. Due to the increase
muscle tone with unilateral paralysis of the limbs, a typical posture occurs with a characteristic
appearance - the Wernicke-Mann pose.
Changes in muscle tone are also observed with lesions of extrapyramidal and
cerebellar systems.
In maintaining muscle hypertonicity in central paralysis, an important role is played by
neuropeptides (oligopeptides), which are called postural asymmetry factor. If the liquor
a patient with hemiplegia (due to vascular, traumatic, tumor lesions of one
cerebral hemisphere) enter an endolumbally healthy experimental animal, then
this recipient animal rapidly develops a movement disorder in the extremities of the same
and the patient's side. Neuropeptides apparently act on the synaptic membrane of motor
neurons of the segmental apparatus of the spinal cord, acting as a synapse modifier.
Improvement in synaptic transmission may result from increased sensitivity
receptors to the mediator or changes in the metabolism of the mediator itself. The dynamics is revealed
the activity of these neuropeptides during the course of the disease - they are detected 1-3 days after
the development of a pathological focus in the brain. This activity persists for 1-3
weeks and then decreases.
So, neuropeptides are involved in the formation of neurological syndromes. Activity
neuropeptides of cerebrospinal fluid can be reduced, which is of great promising importance in the development of
methods of treating neurological patients.
Paralysis (plegia) is a complete lack of motor activity, and **paresis**
- This is a partial violation of motor activity.
With paralysis or paresis, muscle atrophy may occur, which is characteristic of
dysfunction of the peripheral motor neuron. Central
the motor neuron affects muscle trophism to a much lesser extent, when it

lesion, muscle atrophy usually does not occur or is poorly expressed.
Essential in recognizing the type of paralysis (peripheral or
central) has a state of reflexes.
Symptoms of damage to the peripheral motor neuron. For couples
lich, depending on the lesion of the peripheral motor neuron, is characterized by
decrease in muscle tone, decrease or complete disappearance of deep re-
flexes, the appearance of muscle atrophy. Such a symptom complex is called sluggish,
or atrophic, paralysis. With peripheral paralysis, changes
electrical excitability of nerves and muscles, as well as electromyographic
characteristic
theristics; sometimes the mechanical excitability of the muscles increases (in response
to a blow
the muscle contracts with a hammer). In atrophic muscles, fast
rhythmic contractions of fibers or their bundles, which is indicated by fasciculations,
and
their presence indicates damage to the neurons of the anterior horns of the spinal cord.
Symptoms of the lesion of the central motor neuron. Pyramid
the system transmits motion programs. Therefore, to defeat the central
the neuron is characterized by paralysis not of individual muscles, but of whole
groups. Also typical
is a symptom complex of disinhibition of deep reflexes. Among them
increased muscle tone (the occurrence of spastic phenomena).
Therefore, this paralysis is called spastic.
Deep reflexes increase, their reflexogenic zones expand.
The extreme increase in these reflexes is manifested by clonuses.
Patella clonus is caused in a patient lying on his back with a
straight lower limbs. The examiner I and II grips with his fingers
the top of the patient's patella, together with the skin pushes it up, then displaces
down and holds it in that position. Quadriceps femoris tendon
stretches, rhythmic muscle contractions and rapid oscillations occur
patella up and down. The clonus is held until the examiner
will stop stretching the tendons. This phenomenon indicates a high degree of
increase the stretch reflex.
The clonus of the foot is called in the patient in the supine position. Doctor flexes
the lower limb of the patient in the hip and knee joints, holds it
with his hand for the lower third of the thigh, the other grabs the foot and after
maximal plantar flexion with a strong jolt
produces extension of it, trying to maintain this position. In response,
rhythmic contraction of the gastrocnemius muscle and clonus of the foot throughout
the entire time,
while the heel tendon continues to stretch. At the heart of this phenomenon
there is not a tonic, but an increased repeated phasic stretch reflex.
With central paresis of the lower extremities, the clonus of the foot sometimes appears
as if
spontaneously, but in fact it appears as a result of stretching the tendon in the

the time when the patient, sitting on a chair, rests his toes on the floor, as well as when trying to get up.
With central paralysis, skin reflexes (abdominal, cremasteric, plantar) fall or fall out. This is probably due to the fact that segmental superficial reflexes appear only if the facilitated of the pulses arriving along the pyramidal tracts to the segmental apparatus spinal cord. The defeat of the latter is accompanied by the disappearance of the superficial reflexes.
Pathological reflexes are fairly constant and practically very important signs of central motor neuron.
Pathological reflexes on the lower extremities are divided into 2 groups - extensor (extensor) and flexor (flexor).
Among the extensor reflexes, the most famous and diagnostic

the **Babinsky reflex** acquired importance **.** It belongs to the number of skin reflexes, preserving preschool age. At 2-2.2 years, this reflex disappears, which coincides with myelination of the pyramidal system. Obviously, by this time it begins to function and inhibits this reflex. Instead, the plantar flexor begins to be called reflex. The Babinsky reflex becomes pathological only after 2-2.2 years of life. He is it is one of the most important symptoms indicating a lesion of the central motor neuron. In response to intense streak irritation of the external part of the plantar surface of the foot, there is a slow tonic extension of the 1st finger, often this coincides with the fan-shaped divergence of the rest fingers.
Other pathological reflexes of the extensor type are expressed in the same motor effect - extension of the 1st finger. They differ in character and the place of application of the stimulus.
Oppenheim reflex - extension of the first toe in response to holding with by pressing the pad of the first finger of the examiner on the anterior surface of the lower leg.
The movement is performed along the inner edge of the bony bone from top to bottom. V normally, with such irritation, the fingers bend in the interphalangeal joints (less often - flexion in the ankle joint).
Gordon reflex - extension of the first finger or all fingers of the lower extremity when compressed by a brush examining the gastrocnemius muscle.
Schaefer reflex - a similar extension of the thumb in response to compression of the heel tendon.
Cheddock reflex - extension of the first toe of the foot during stroke stimulation the skin of the outer ankle in the direction from the heel to the back of the foot.
Grossman's reflex - extension of the first toe with compression of the distal phalanxes of the V toe.
Flexion pathological reflexes include the following re-

flexes.
Rossolimo reflex - rapid plantar flexion of all toes in
response to abrupt blows to the distal phalanges of the fingers. Normally observed sometimes just a shake of the fingers. From a modern perspective, the Rossolimo reflex
should be considered as an increased phasic reflex to stretch the muscles - flexors, due to the lack of pyramidal inhibition in case of damage
central motor neuron.
Ankylosing spondylitis-Mendel reflex - rapid plantar flexion of the II-V fingers when tapping with a hammer on the back of the foot in the region of the III-IV metatarsal bones. V
normally, the fingers of the lower extremity do dorsiflexion or remain motionless.
Zhukovsky-Kornilov reflex - rapid plantar flexion II-V
toes when struck with a hammer on the plantar side of the foot closer to fingers.
On the upper limbs, with damage to the central neuron, they can also pathological reflexes occur. More often than others, the **upper reflex** is called **Rossolimo -** flexion of the distal phalanx of the 1st finger of the hand in the interphalangeal joint
with a short blow to the tips of the II-V fingers with a freely hanging hand. Often, the distal phalanges in the interphalangeal joints can bend synchronously and other fingers. A successful modification of the technique is E.L. Venderovich - with a supinated hand of the subject, a blow is applied to distal phalanges of fingers II-V slightly bent in the interphalangeal joints (reflex Rossolimo - Venderovich).
Less persistent pathological reflexes on the hand are:
ankylosing spondylitis - a fast nodding movement of the II-V fingers upon impact a hammer on the back of the hand in the area of II-IV metacarpal bones;

Zhukovsky reflex - flexion of the II-V fingers in response to a blow with a hammer on
palmar surface of the hand in the region of III-IV metacarpal bones.
Hoffmann reflex - flexion movement of the fingers in response to a pinch irritation of the nail plate of the third finger of the passively hanging hand;
Klschel-Weyl reflex - flexion of the first finger of the hand with passive extension II-V fingers;
Jacobson-Lask symptom - palmar flexion of the fingers on impact a hammer along the lateral part of the carpal-radial articulation. Apparently he reflects an increase in the carpal-ray reflex. The same movements can occur when the biceps reflex is triggered.
From the above, it follows that pathological reflexes have a different genesis. but whether or not they are revitalizing an inhibited reflex (Babinsky reflex) or enhancement of the normal stretching reflex (Rassolimo reflex), all of them are of great diagnostic value and indicate a lesion of the central motor neuron.

Protective reflexes (reflexes of spinal automatism) are also characteristic of central paralysis. They represent involuntary tonic synergistic movements in the paralized limbs, arising in response to intense irritation of skin receptors or deep-lying tissues. As an irritant, you can use injections, tweaks, application to the skin of ether drops. Protective reflexes can be triggered by sharp passive bending in any the joint of the paralyzed limb.

The protective (shortening) reflex of ankylosing spondylitis-Marie-Foix consists in a synergistic triple flexion of the lower limb: in the hip, knee and ankle joints (dorsiflexion of the foot). This reflex can be triggered by a superficial or deep dredging. One technique for inducing a reflex is enhanced passive plantar flexion. fingers or feet.

Remak's protective femoral reflex is detected by dashed stimulation with a handle neurological malleus of the skin of the upper third of the anterior region of the thigh. Plantar flexion of the foot, I-III toes and extension of the lower limb in the knee joint.

The reflex arc of this reflex closes at the level of the lower lumbar - upper sacral segments. The appearance of a reflex indicates damage to the spinal cord above the lumbar thickening.

Protective shortening (lengthening) reflex of the upper limb: in response to irritation of the upper half of the body, the upper limb is brought to the trunk and bends in elbow and wrist joints (shortening reflex) or upper limb unbends in these joints (lengthening reflex).

The Davidenkov shortening reflex is manifested in case of shrikhovy irritation of the sole paralyzed limb or when trying to produce plantar flexion of the foot: occurs flexion in the hip and knee joints and extension - in the ankle joint (triple shortening).

Establishing the segmental level to which defensive reflexes are evoked has diagnostic value. They can be used to judge the lower border of the pathological focus in the spin-
brain.

In addition to the above research methods, with a defeat central motor neuron to detect mild paresis there are some pretty visual tests. These include, for example, a test for the presence of pyramidal insufficiency **(Barre's test): the** patient in the supine position on the stomach, passively bend both lower limbs at the knee joint at an angle about 45 ° and suggest holding this position. The paretic leg begins to gradually descend. The Barre test for the upper extremities is checked in a patient sitting with eyes closed. The upper limbs of the patient raise slightly above the horizontal level with touching palmar surfaces; the patient is asked to fix them in this position. Paretic the upper limb soon begins to descend (Barre's upper test). At hands raised above the head, palms up (Buddha pose), descends faster paretic upper limb.

Mingazzini symptom - the examinee is offered with closed eyes under pull both upper limbs up, palms inward, to a horizontal line and

fix them in this position. The paretic limb bends faster in elbow and wrist joints and descends. There is also a modification methods of causing a symptom: the patient should raise the upper

limbs and fix them in this position. The symptom allows you to identify initial signs of paresis of the upper limb.
If the patient, with actively adducted fingers, try to withdraw the V finger from IV, acting on the main phalanges, it is possible very early to reveal unilateral weakness, which is observed with the defeat of the pyramidal tract, **- motor ulnar defect according to Venderovich.**
A sign of damage to the central motor neuron is also the appearance pathological **synkinesis** (accompanying, normally inhibited movements).
So, when the fingers of a healthy hand are squeezed into a fist, the paralyzed hand is synchronously
makes this movement; when coughing, yawning, sneezing may occur involuntary flexion of the upper limb in the elbow joint on the side of paresis; when trying to sit down in a lying patient, flexion of the lower limb is observed in hip joint.
Thus, the main signs of defeat of the pyramidal system are the absence voluntary movements or limitation of their volume with a decrease in muscle strength, an increase
muscle tone (spastic muscle hypertonicity), increased deep (myotatic) reflexes, decrease or absence of cutaneous and the appearance of pathological reflexes, the occurrence of pathological synkinesis. This picture of the defeat of the central motor neuron differs sharply from the clinic of peripheral paralysis.
According to the prevalence of paretic phenomena, the following are distinguished **options for movement disorders:**
monoplegia (monoparesis) - paralysis of one limb;
paraplegia (paraparesis) - paralysis of two either upper or lower limbs, for example, upper (upper paraplegia) or lower (lower paraplegia):
triplegia (triparesis) - paralysis of the muscles of three limbs;
tetraplegia (tetraparesis) - paralysis of both upper and lower extremities;
hemiplegia (hemiparea) - paralysis of the muscles of one half of the body (right or left);
diplegia (diparesis) - paralysis of both halves of the body (two hemiplegias).
If a muscle group innervated by the cells of the anterior horn is paralyzed one segment of the spinal cord (or the anterior spinal root), then this is denoted as paralysis (paresis) of the myotome in the corresponding segment of the dorsal
brain (for example, Lv myotome paralysis on both sides).
Symptom complexes in case of damage to the cortical-muscular pathway on various levels. The defeat of the cerebral cortex. Pulses of voluntary movement in the affected hemispheres are not formed or do not reach the level of the internal capsules. Here the pyramidal bundle is interrupted above the cross. Both its parts are cortical-nuclear and cortical-spinal. Hence paralysis muscle occurs on the opposite half of the body. Due to incompleteness crossover and unequal disturbance of bilateral innervation degree the severity of paresis in individual muscle groups varies. Of the mimic only the lower group and the muscles of half of the tongue are paralyzed. It appears this is because when showing teeth, the upper lip on the side of the paresis rises less, the corner of the mouth is pulled to the unaffected side. Tongue when protruding

deviates to the affected side, it is moved there by all contracting
non-paralyzed muscles of the healthy half of the tongue. Weakness of the muscles of the upper
the inferiority is more pronounced than the lower one. Both on the upper and lower limbs
distal muscle groups are more affected.
Paralysis (paresis) forms on the side opposite to the focus. The border

between the paralyzed and healthy side runs along the midline of the body. Such
the syndrome is called hemiplegia (from the Greek hemi - half, plege - defeat).
Due to damage to the central neuron on the paralyzed side,
signs of disinhibition of the segmental apparatus - increased muscle tone,
deep reflexes revive, pathological and protective reflexes appear
(Babinsky's reflex is found especially early and constantly).
With disinhibition of the segmental apparatus, except for those listed above
symptoms, there are pathological friendly movements - synkinesis.
Pathological synkinesis is usually divided into global, coordinating and
imitative (contralateral).
Global synkinesis - involuntary movements of the paralyzed
extremities arising from strong and relatively prolonged stress
muscles of healthy limbs (for example, with forced compression of the fingers
a healthy hand in a fist), as well as when coughing, sneezing, laughing, crying,
straining.
The nature of global synkinesis is usually determined by a selective increase
tone in paralyzed limbs. Synkinesis on the upper extremities
manifested by flexion of the fingers, flexion and pronation of the forearm, abduction
shoulder (shortening synergy); on the lower limb - by adduction of the thigh,
knee extension, foot flexion, toe flexion
(extension synergy).
Focal synkinesis - involuntary contractions of paretic
muscles when trying to voluntarily contract other functionally related
them muscles. They are usually observed during the recovery period of arbitrary
movements, when it becomes possible to perform some volitional movements, and
synkinesis cannot be actively delayed.
Synkinesis in hemiparesis includes the so-called
tibial Strumpel phenomenon. The patient in the supine position cannot
on the side of the paresis, perform dorsal extension of the foot, but when he bends the lower
nude limb in the knee joint, especially with opposition from the side
examiner, the anterior tibial muscle contracts and is performed
extension in the ankle joint. To the same category of synkinesis with
hemiplegia refers to the Raimist symptom. The patient in the supine position does not
can lead and abduct the thigh on the paralyzed side. But these movements in
paretic muscles of the lower limb appear when the patient produces them
a healthy limb, especially with resistance from the examiner.

Friendly movements with hemiplegia include a symptom of an unproductive free raising of the paralyzed lower limb, when the patient from lying position sits on the bed without the aid of hands **(Babinsky synkenesia).** When bending the trunk forward, the lower limb is on the side of hemiparesis bends involuntarily in the knee joint **(Neri phenomenon)** . Often occurs synkinesis in the form of involuntary flexion of the thumb with passive extension of II-V fingers (Klippel-Weil phenomenon) or fan-shaped breeding fingers of the paralyzed upper limb with yawning.

Simulated synkinesis - involuntary movements of one limb, imitating volitional movements of the other. Fake synkinesias include replacement compensatory movements that the patient makes healthy limb when performing a movement with a paretic limb. Patients with paresis of the upper limb sometimes make it easier for themselves to perform movements with it by means of identical tension of the healthy upper limb.

In the early stages of ontogenesis, human movements are symmetrical and bilateral. due to bilateral irradiation of motor impulses. Subsequently, at complete myelination of pyramidal and extrapyramidal fibers, as well as with the acquisition of individual motor experience, it becomes possible

perform movements with only one limb. Purchase of motor skills consists in learning not only to quickly produce the necessary movements, but also suppress synkinetic impulses. With the defeat of the pyramidal system at the level of the cortex and basal nuclei, disinhibition of the existing in the early period of development of neural connections in subcortical formations and again imitating synkinesis, which are pathological, are resumed.

Congenital imitative synkinesis is expressed in the propagation of impulses along associative fibers to both hemispheres of the brain, which makes it difficult formation of many motor skills of the upper limb.

So, hemiplegia syndrome in its most complete form is associated with a lesion the inner capsule of one of the cerebral hemispheres. If the pyramid bundle on this level damaged shallowly and temporarily (for example, as a result of edema), violations its functions are reversible. When pyramidal fibers are destroyed, arbitrary movements are lost and hemiplegia is formed. In this phase, patients learn walking. At the same time, the paretic lower limb is brought forward, describing semicircle. This resembles the movement of the scythe when manually cutting grass - "mowing gait". A persistent change in the posture of the limbs with hemiplegia is called by the names of the authors - the Wernicke-Mann contracture.

When the focus is localized along the pyramidal path from the cortex to the inner capsules (precentral gyrus, paracentral lobule, radiant crown) the clinical picture largely depends on the transverse size of the lesion.

If it is extensive and covers the entire area of the beginning of the pyramidal system,

symptom complex, identical to capsular hemiplegia. However, such massive foci are rare. Usually part of the central neurons is involved or their axons. Those of them that are related to the regulation of traffic suffer one limb or just the muscles of the head area. Isolated paralysis the lower limb is designated by the term monoplegia cruralis, and the upper limb is sti - monoplegia brachialis. The paralysis will show signs of central neuron. The defeat of the upper limb is often combined with the defeat of the face and language. It turns out a characteristic syndrome - paralysis facio-linguo-brachialis - of its kind of incomplete hemiplegia.

Unilateral damage to the fibers of the pyramidal bundle can occur and below the inner capsule, within the brain stem (brain stem, brain bridge, medulla). In this case, hemiplegia will develop on the on the opposite side to the hearth. At the same time, any of the movement is involved cranial nerves for the corresponding muscles of the side of the same name. WHO- a peculiar clinical syndrome disappears: cranial nerve palsy on the side focus and hemiplegia on the opposite, which is called alternating hemiplegia (from Latin alternans - alternating, alternating).

Spinal cord injury. Along its entire length in the lateral cords pass the pyramidal beams crossed above. Parallel to them in the front horns peripheral motor neurons are located. At the level of each segment from pyramidal bundles, fibers depart to form a synapse with the corresponding neurons. When the pyramidal fibers are interrupted above the cervical thickening (damage to the upper cervical segments) of the spinal cord will be impaired pyramidal beams for the upper and lower extremities. Paralysis of the upper and lower limbs (tetraplegia). Paralysis will also be signs of damage. central neuron (spastic tetraplegia).

What movement disorders will develop with transverse lesion spinal cord at the level of the thoracic segments? Fibers are interrupted pyramidal system for both lower extremities, which will lead to their paralysis. The upper limbs will remain unaffected. Deep reflexes and muscular the tone on the lower extremities will be increased, protective and pathological

reflexes. Skin reflexes below the level of the lesion are extinguished. Such a transverse the distribution of paralysis of the limbs is called paraplegia. Since in this case, only the lower extremities are affected, they speak of lower paraplegia. In connection with an increase in muscle tone (spastic phenomena) such paraplegia is called spastic.

With the defeat of the pyramidal bundle in one lateral cord at the level upper cervical segments of the spinal cord, paralysis of the upper and lower extremities on the side of the focus (spastic hemiplegia). Mimic muscles and tongue at the same time do not suffer. This syndrome is called spinal hemiplegia.

Isolated damage to motoneurons in the spinal cord occurs when

poliomyelitis, tick-borne encephalitis, myeloischemia, etc.
When the cells of the anterior horns are destroyed in the cervical thickening, peripheral paralysis of the upper limbs (upper flaccid paraplegia).
There is damage to motoneurons throughout the spinal cord, which clinically manifested by paralysis of both upper and lower extremities - flaccid tetraplegia.
When the motor neurons of the lumbosacral segments are damaged, lower paraplegia with decreased muscle tone, extinction of deep reflexes on the lower limbs. Muscle atrophy occurs after a few weeks (flaccid atrophic lower paraplegia).
The lesion in the anterior horns of the spinal cord may be limited to one or two segments. The corresponding muscle groups will be affected (segmental type paralysis or paralysis of the myotome). Innervation of muscles located below and above hearth will remain preserved.
Now consider paralysis when a peripheral neuron is interrupted outside proper medulla. In these cases, the roots may suffer or peripheral nerve trunks. Important for diagnosis distribution of movement disorders. In one case, a group of victims muscles coincides with the innervation of some root, in the other - a nerve. Erb-Duchenne palsy is a classic example. Arises unilateral paralysis of the peripheral type of deltoid muscle (axillary nerve), biceps brachii and brachialis (musculocutaneous nerve), brachioradial muscles (radial nerve). It is difficult to allow the simultaneous defeat of three nerve trunks, it is more likely to assume the defeat of the roots of Cv-Cvi. Defeat spinal roots can be confirmed by the distribution of disorders sensitivity.
If a muscle group has signs of peripheral paralysis and these muscles coincide with the zone of innervation of one nerve, this is called the neural type of paralysis distribution.
With multiple lesions of the nerve trunks (polyneuritis) or with non- which hereditary diseases of the nervous system occurs in a peculiar symptom complex: flaccid tetraparesis, in which muscle weakness is more expressed in the distal extremities. This pattern is called distal or polyneuritic distribution of paralysis.
With hereditary muscular dystrophies, atrophy and paresis are localized more in the muscles of the pelvic and shoulder girdle, as well as the proximal regions limbs; distal muscles remain relatively intact (myopathic type of paralysis distribution). Central neuron (dendrite and body) may be subject to excessive irritation (irritation).
The accumulation of subthreshold irritative impulses leads to periodically the coming stormy discharges in the form of a stream of impulses along the axons of the central neurons. The peripheral motor neuron is instantly activated, muscles contracting convulsively, corresponding to the focus of irritation in the cortex

brain. These cramps of individual muscle groups are predominantly clonic character. In one or another joint, jerky sequential flexion and extension or adduction and abduction. Attack lasts several minutes and can be repeated later. Consciousness at the patient is saved. Such paroxysms of local seizures are called cortical, or Jacksonian, epilepsy. Convulsions usually occur in those groups muscles that a person uses more during voluntary movements. For example measures, muscle spasm of the 1st finger of the hand occurs more often than the V (a large area the cortical zone for the 1st finger). A cramp starting in the muscles of the thumb, often spreads in this order: other fingers, hand, entire upper limb, face; transition to the lower limb is possible. This is consistent with the spread excitation along adjacent motor centers in the precentral gyrus.

In some cases, local seizures are generalized, there is a general epileptic seizure with loss of consciousness. The appearance of epileptic seizures, in particular attacks of cortical epilepsy, always arouses suspicion of intracranial volumetric process (tumor, cyst, arachnoiditis).

There is another type of cortical epilepsy. It is characterized by the fact that local convulsions do not occur in seizures, but persist constantly. Periodically convulsions intensify, generalize, and the patient has a general epileptic seizure. This form of the disease has been described by a domestic neuropathologist A. Ya. Kozhevnikov in 1894 and is called Kozhevnikovskaya epilepsy. This syndrome often occurs with chronic tick-borne encephalitis.

It should be noted that there are various options for paralysis (paresis), such as organic, reflex and functional.

Organic paralysis (paresis) develops with changes in structure central or peripheral motor neuron due to various causes (trauma, vascular, tumor, inflammatory, degenerative and other diseases). Organic paralysis options: central, peripheral and mixed. Mixed paralysis (paresis) is said to be when at the same time in the muscles of one limb, signs of damage are revealed peripheral (muscle atrophy, hypotension, fascicular twitching) and central motor neurons (revitalization of deep reflexes, pathological signs). The most common mixed paresis occurs with lateral amyotrophic sclerosis.

Reflex paresis is characterized by revitalization of deep reflexes with the presence of pathological signs with sufficient preservation of muscle strength. it happens with partial damage to the cortical-muscular pathway or with dislocation impact on the pyramidal system in the case of an extensive, adjacent pathological focus.

Functional paralysis (paresis) is associated with the effects of psychogenic

factors that lead to neurodynamic disorders of the central
nervous system and are found mainly in hysteria. With such paralysis
trophism and muscle tone do not change, deep reflexes are preserved, there are no
pathological hand and foot signs.

GENERAL SENSITIVITY AND ITS DISTURBANCES

Sensitivity - the ability of a living organism to perceive irritations,
emanating from the environment or from one's own tissues and organs, and respond to them by differentiated forms of reactions.

The body is constantly exposed to various stimuli: mechanical,
chemical, temperature, etc. All external agents primarily affect the integument
body. These irritations are perceived by a large number of nerve fibers, which are
distal areas of the dendrites of the cells of the spinal nodes. For the most part, this is so
called free nerve endings; there are so many of them that they form whole plexuses.
Some fibers end up in epithelial structures, in the form of a bulb, disk or
bulbs. These terminal devices of dendrites are receptors. Free ends of fibers and
fibers entering special receptors perceive irritation and transform it into
nerve impulses.

In case of skin irritation, the cell of the intervertebral ganglion (first neuron) or homologous
to him, the ganglion of the cranial nerve directs the impulses perceived and processed by it not only in
an effector neuron for the formation of a segmental reflex. At the same time, she transmits
information to a second sensitive neuron located in the spinal cord and stem
formations; the third neuron (in the visual hillock) transmits impulses to the cerebral cortex. Here
a huge complex of cortical nerve cells comes into play, and a nerve impulse enters
consciousness - a sensation arises. This is how the classic idea of the formation
sensations as a result of stimuli acting on the body. All perceptions of external influences and
internal environment in physiology, it is customary to combine the concept of
"reception". However, not all that
it is perceived by nerve receptors, it is felt, that is, it enters consciousness. The concept of reception is broader,
than the concept of sensitivity. An example is the signals coming from the reference motor.
body apparatus into the cerebellum. They regulate muscle tone and are involved in coordination
movements, but impulses from them do not lead to sensations.

Analyzers are functional associations of structures of peripheral and
central nervous system, carrying out the perception and analysis of information about phenomena occurring both in the environment and in the internal environment of the body.

Analyzers are divided into 2 groups. External (exteroceptive) analyzers carry out
analysis of information about phenomena occurring in the environment or inside the body. To them
include visual, olfactory, auditory, tactile, etc. Internal (interoceptive)
analyzers process information about changes in the internal
the environment of the body, for example, information about the state of the gastrointestinal tract, cardiovascular
that system, lungs and other internal organs. One of the main internal analyzers
is a movement analyzer that informs the brain about the state of the musculo-articular apparatus
rata. The muscular system is not only an executive motor apparatus, but also an organ
proprioceptive sensitivity. Even I. M. Sechenov in 1863 showed that “the dark

muscle sense ”has an important role in the mechanisms of movement regulation.
An intermediate position between external and internal analyzers is
vestibular analyzer. The receptor is located inside the body (semicircular canals), but excited by external factors (acceleration and deceleration of rotational and rectilinear movements).
Each analyzer consists of a peripheral (receptor) section, a conductive part and cortical department.
Peripheral analyzer
represented by specialized receptors,
converting certain types of energy (light, sound, heat) into nerve impulses.
Due to the specialization of receptors, the primary analysis of external stimuli is carried out. V the brain differentiates the meaning of these signals. This is due to the fact that the signals in the receptor portions are encoded. In addition to pulse-code communication, and specific electrotonic functional connections with different areas of the brain.
The conductive part of the analyzers is represented not only by neurons of the thalamic nuclei and their
projections to the corresponding areas of the cerebral cortex, but also such formations as the reticular
formation, structures of the limbic system, cerebellum. It has been established that the afferent signal,
coming even along one fiber, is transmitted to many neurons in specific, associative and nonspecific nuclei of the thalamus, which, in turn, switch each impulse to another more cortical neurons.
The cortical section of the analyzer has a certain localization, for example, the visual - mainly in the occipital region, auditory - in the temporal, motor - in the parietal region

cerebral cortex. The boundaries of these analytic zones are indistinct. In the cortical regions the analyzer contains neurons that respond only to certain sensory stimuli. it
specific projection neurons (the nucleus of the cortical end of the analyzer). Next to them are nonspecific nerve cells that respond to various sensory stimuli, i.e.
possessing multisensory convergence. There are especially many such neurons in the associative areas of the cerebral cortex. Due to the convergence of excitations on the cortical neuron, it is possible
interaction between many analyzers. Based on the analysis of signals entering the brain from external and internal receptors, afferent synthesis of information is carried out, followed by the formation of a program of behavior and an apparatus for evaluating the results of action - an acceptor of results
actions.
The analyzer's activity is not limited to the analysis of external and internal information, but includes the reverse influence of the higher sections on the receptor and conductive parts of the analyzer.
The sensitivity of the receptors (the receiving part) and the functional state of the transmission the relay (conductive part of the analyzer) is determined by the descending influences of the cerebral cortex,
which allows the body to actively select from many stimuli the most adequate for a given moment sensory information. This is expressed in the form of peering, squinting, listening and is physiologically explained by a decrease in the threshold for visual or auditory stimuli.
Receptors, depending on their location, are conditionally subdivided into exteroreceptors (pain, temperature, tactile receptors), proprioceptors
(located in muscles, tendons, ligaments, joints, receptors that give

information about the position of the limbs and trunk in space, the degree of reduction muscles), interoreceptors (located in the internal organs of the baro- and chemoreceptors).
In clinical practice, various receptors are investigated when applying co-corresponding irritations; the sensations arising in the subject are indicated, as a general sensitivity.
Sensations from irritation of exteroreceptors are called **superficial (exteroceptive) sensitivity** .
Allocate the following shape exteroceptive sensitivity - pain, heat, cold and tactile.
A sense of the position of the trunk and limbs in space (muscular-articular feeling), feeling of pressure and body weight, vibrational, kinesthetic sensitivity, two-dimensional-spatial sense refers to a **deep sensitivity (bathyesthesia).**
There are also **complex types of sensitivity** due to the combined activity of different types of receptors and cortical parts of the analyzers (feeling localization, recognition of objects by touch - stereognosis, etc.).
Interoceptive sensitivity is the sensation arising from irritation. internal organs, walls of blood vessels. To a large extent, they are associated with the sphere vegetative innervation. Under normal conditions, impulses from internal organs are almost not are realized. However, with irritation of the interoceptors, there are sensations of discomfort, feelings severity, pain of varying intensity. This kind of sensation is not strictly localized, in others in cases they are, to varying degrees, localized and associated with a specific organ.
In addition to general sensitivity, a special sensitivity is distinguished that arises in response to irritation from outside of special senses. This sensitivity includes: sight, hearing, smell, taste, and sight, hearing and smell are also called distant, that is, perceiving irritation at a distance, in contrast to contact, in which the stimulus is directly in contact with skin or mucous membranes. Contact receptors include tactile receptors sensitivity (sensation of touch), partly also pain and temperature receptors.
Pain and temperature sensitivity conductors. Conductor travel pain and temperature sensitivity differs from that of deep sensitivity.
The first neuron of the conductors of pain and temperature feelings, as well as other tracts of the general sensitivity, represented by the nerve cell of the spinal ganglion with its T-shaped dividing dendraxon (a process in which the beginning of the dendrite and axon are in close contact and there is the impression of their merger). The peripheral process of this cell as part of the spinal nerve,

plexus, peripheral nerve trunk goes to the corresponding dermatome (dermatome call the area of innervation of the skin from one spinal ganglion and the corresponding segment spinal cord). Dendrites that perceive cold irritations contain receptors in the form encapsulated sensitive endings (Krause bulbs), and thermal fibers in the form non-encapsulated nerve endings (Ruffini endings), Axons of spinal cells

the ganglion form the spinal nerve and the posterior root, entering the substance of the spinal cord, it
the fiber passes the marginal zone (Lissauer zone), then a gelatinous substance and forms at the base
the posterior horn is a synapse with a second neuron in the sensory pathway. The cells of the second neuron make up
the so-called own nuclei - a column of nerve cells that runs along the spinal cord. Yet
before the formation of the synapse, the axon of the neuron of the spinal ganglion gives up the collateral branch for the arch
the corresponding segmental reflex. The axon of the second neuron then passes through the anterior
soldering to the opposite side into the lateral cord, however, the fiber does not pass strictly horizontally, and obliquely and upward. The transition is carried out 1-2 segments higher. This anatomical
the feature is important in determining the level of spinal cord injury.
Entering the lateral cord on the opposite side, the axon of the second neuron goes up
together with similar fibers entering the lateral cord below. A bundle is formed
passing through the entire spinal cord and brain stem. In the medulla oblongata, it occupies the position
slightly dorsal to the lower olive, in the pons and midbrain from the dorsal side adjacent to lemniscus medialis, ends in the ventrolateral nucleus of the thalamus.
At the place of origin (spinal cord) and end (ventrolateral nuclei of the thalamus), this path received
the name of the spinal thalamic. The fibers in this bundle are distributed in a very peculiar way. From
dermatomes located below, the fibers lie in bundles outside, and from located more
high - inside. As a result, at the height of the upper cervical segments in the dorsal-thalamic bundle
the fibers are located most laterally from the lower limb, medial - from the trunk, and
more inwards - from the upper limb. Such a pattern in the arrangement of long conductors, or the law of the eccentric arrangement of long conductors is important for the topical diagnostics; this especially applies to the diagnosis of spinal tumors. With extramedullary tumor, the area of disorder of surface sensitivity begins from the distal parts of the lower limbs, and with further growth of the tumor, it spreads upward (ascending type of disorder sensitivity). With an intramedullary tumor, the area of sensitivity disorder, on the contrary, spreads from top to bottom (descending type of development of sensitivity disorder) . Part axons of the second neuron ends in the formatio reticularis and in the nonspecific nuclei of the thalamus.
Axons of the third neuron begin in the cells of the dorsoventral nucleus of the thalamus; they head to
the posterior thigh of the inner bag, where they occupy a position behind the pyramidal bundle, forming
thalamocortical bundle. Then the fibers of this bundle fan-out (corona radiata) and reach cortex (postcentral gyrus, adjacent areas of the parietal lobe - cytoarchitectonic fields 3, 1, 2, 5, 7). Here (especially in field 3) there is a somatotopic projection of these conductors with respect to
to certain parts of the body. In the upper part of this area of the cortex, including the paracentral lobule, the sensitivity of the lower limb is presented, below - the trunk, upper limb,
faces. In this case, the area of cortical sensitivity of innervation for the distal parts of the upper and
the lower limbs are larger than for the proximal. It is especially great for the thumb
the upper limb and around the innervation area of the face and head.

So, the features of the conductors of pain and temperature sensitivity are three-neuronal structure, intersection of their fibers (irritation from the right side of the body perceived by the left hemisphere, and vice versa), crossing the axons of the second neuron on over 1-2 overlying segments of the spinal cord.
Specific differentiation of sensitivity is associated with structural and physiological features of the peripheral nerve fiber. Pulses through sensitive fibers from receptors are carried out at different rates depending on the thickness of the myelin sheath and different frequencies of the observed fluctuations of electrical potentials. Group A fibers with thick myelin sheath conducts impulses faster and provides a deep and tactile sensitivity. Fibers of group B with a thin myelin sheath conduct impulses of more slowly and provide pain, temperature and tactile sensitivity. Fibers with single-layer myelin sheath (non-fleshy) of group C conduct pulses of diffuse unlocalized pain even more slowly.

Pain and temperature sensitivity studies begin with examination of complaints. The most common complaint is pain. During questioning it is necessary to find out the nature of the pain (acute, dull, shooting, aching, stabbing, burning, throbbing, etc.), its localization and prevalence, is it constant or occurs periodically (attacks).
In a similar way, the so-called **paresthesias** can be characterized **.** This term denotes a kind of sensation when patients complain of crawling, tingling, numbness, tightness, cold and warmth, itching and other sensations arising without the application of external stimuli.
Next, the sensitivity is investigated when certain irritations are applied; it turns out how the patient perceives them. When testing skin sensitivity it is necessary to create an appropriate environment that allows the patient to concentrate (peace, silence, fairly comfortable room temperature); at fatigue of the patient should take a break. Tasks are offered to the patient in a clear form; it is shown in advance which research will be carried out, and then a patient with closed eyes should determine the nature of the irritation applied. Suggestive language should be avoided, the patient should describe his own Feel. Irritations are produced at different intervals, sensations are compared on the sick and healthy side. The boundaries of the zone must be defined altered sensitivity.
Research begins with determining pain sensitivity. Injections are not must be too strong and frequent. First you need to find out whether it distinguishes a patient in the investigated area, an injection or touch. For this, alternately, but without in the correct sequence, touch the skin with a blunt or sharp object, and the patient is asked to define "dull" or "acute". The shots should be short they should be produced so as not to cause severe pain. To be sure the boundaries of the zone of altered sensitivity, the study is carried out both from a healthy

plot, and in the opposite direction. The boundaries of the disorder can be noted on the skin.
dermograph. **To study thermal sensitivity** as
irritants use test tubes with hot (+40 ... + 50 ° C) and cold (not higher
+25 "C) water. The water temperature must be sufficient to cause a distinct sensation of warmth and not too high so as not to cause burns. For indicative judgment sometimes you can use metal objects with a high and
low (rubber objects, examiner's finger) thermal conductivity.
First, they find out whether the patient distinguishes between warm and cold (healthy people notice
difference up to 2 ° C). Then the intensity of perception is compared
temperature irritations on different parts of the skin surface, find the border reduced or lost temperature sensitivity. Study
carried out separately for thermal and cold sensitivity (they can
violated to varying degrees). Temperature irritation should be applied in such a pace, like injections, otherwise the patient will not have time to correctly assess their nature and
intensity.
Sufficiently objective information can be obtained by examining the threshold characteristics pain and tactile sensitivity with a graduated set of Frey bristles and hairs
(their thickness and the strength of the skin irritation produced gradually increase from the first to the tenth
numbers in the set). This technique allows you to reveal a hidden decrease in sensitivity - due to a decrease in the number of correct answers or the appearance of sensation only when irritation is more
large hairs or bristles.

It is advisable to study sensitive adaptation: with prolonged and unchanged irritation
(pricks of the skin with a pin or bristle) after a while, the sensation of pain that has arisen fades away,
despite continued irritation (adaptation time). Pathological significance are
shortening of the adaptation time, its lengthening and instability (when comparing the data of the research
different symmetrical areas of the skin).
In the zone of latent hypesthesia, the phenomenon of sensory extinction is revealed: at the same time,
irritation with a double stimulus (prick and touch) of symmetrical skin areas on both sides or two patches of skin on one side. After a short period of time in one of two
irritated areas, despite continued irritation, the sensation fades.
Deep and tactile sensitivity. The first stage of the guides of this path is like
and other types of general sensitivity, represented by the cell of the spinal ganglion with its T-shaped dividing process. Its branch, which plays the role of a dendrite, goes to the periphery. If there is
in view of the deep sensitivity, then this branch ends in the spiral receptor of the tendon.
If the branch perceives tactile stimuli, then it ends in the receptors in the form of bodies Merkel or tactile bodies (Meissner's bodies) in the skin and deep tissues. Cell axon
the spinal ganglion enters the posterior cord of its side, gives up a branch to form an arc segmental reflex, then rises up to the medulla oblongata. The totality of these
ascending fibers forms thin and wedge-shaped bundles (Gaulle and Burdach bundles) .

In the course of the fibers of the posterior cords, there is the following feature. Newly entering fibers, if
look from the bottom up, push back to the midline those who entered from the lower lying spinal ganglia.
Therefore, in the medially located thin bundle, fibers for the lower extremities pass, and in wedge-shaped beam - for the trunk and upper limbs. The axons of the first neurons end in
the nucleus of a thin bundle - the nucleus of Gaul (nucleus gracilis) and the nucleus of Burdakh - the nucleus of the wedge-shaped bundle (nucleus
cuneatus). Here are the bodies of the second neurons.
The axons of the second neurons form a new bundle that passes to the opposite side
near the inferior olives of the medulla oblongata. After crossing, this fiber complex takes ascending direction, in the bridge of the brain, painful and temperature fibers are attached to it sensitivity.
In the cerebral pedicles, the common sensory pathway is located in the area of the tire, above the black
substance, lateral to the red nucleus and ends in the ventrolateral nucleus of the thalamus. Beam this one has two names; one in the place of its beginning and end - fasciculus bulbothalamicus, the other is old,
descriptive anatomical - medial loop (lemniscus medialis). The outlines of the beam on the transverse
cut resemble a loop. From the third neuron of this path, the body of which is located in the thalamus, on-
the axon is directed to the cerebral cortex. This fiber tract is called thalamo-corticalis.
As part of the thalamocortical bundle, axons of third neurons pass through the posterior third of the posterior
the legs of the inner bag, through the radiant crown and end in the postcentral and in the precentral
convolutions (fields 2, 2, 1, 4, 6). As for all types of skin sensitivities, the projection of body parts into
the postcentral gyrus is presented as follows: the sensitivity of the face - below, the trunk and upper
limbs - in the middle, lower limbs - at the top (on the medial surface of the gyrus).
Thus, the pathways of deep and tactile sensitivity also consist of 3 neurons.
The axons of the second neurons cross at the level of the olives of the medulla oblongata. Axons of the third
neurons reach the cells of the cerebral cortex.
The generality in the structure of the conductors of surface and deep sensitivity is manifested in their
three-neuron composition, the location of the first neuron in the spinal ganglion, and the third in ventrolateral nucleus of the thalamus. Second neuron for pain and thermal sensitivity
located along the entire spinal cord in the so-called own nuclei of the spinal cord. Poe-
To this, the transition of the axons of this neuron to the other side is stretched along the entire length of the spinal cord. V
conductors of deep and tactile sensitivity also cross the second neurons, but
crisscrossing fibers are more compact and located within the bottom
medulla oblongata (at the level of the lower edge of the olives).
The end of the afferent pathways in the cerebral cortex is the postcentral gyrus with
adjacent areas of the parietal lobe and precentral gyrus (for a deep sense
somatic sensitive zone I. According to some reports,
the fibers of the thalamocortical bundle also end in the region of the posterior part of the upper lip of the lateral
(Sylvian) sulcus (somatic sensitive zone II).

A study of tactile sensitivity. Tactile sensitivity
check by lightly touching the skin with a cotton swab, soft brush or thin
paper. The contact area should not exceed 1cm ^{2} ; should not be produced
sliding, smearing movements. The patient is asked to close his eyes and when feeling
touches say yes. The same as in the study of pain and temperature
sensitivity, compare the sensations on symmetrical parts of the body. At
absence or decrease in the sensitivity of the boundary of its changes in determining
applied to a special form.

A study of deep sensitivity. Feelings arising in
as a result of excitation of proprioceptors of the musculoskeletal system (muscles,
tendons, joints, periosteum), is called articular-muscular feeling. It
refers to deep sensitivity and forms the basis of a sense of position and
movement (feeling of kinesthesia).

In the study of deep sensitivity, the feeling is checked separately.
passive movements, a sense of position, skin kinesthesia, a feeling of pressure and
weight,
vibration sensitivity.

When examining the feeling of passive movements in the joints, the patient
explain what movements they will produce (up and down, outwards - inwards), then
ask the patient to close his eyes and determine the direction of the movement being
made.

A healthy person is able to distinguish between movement in the joint at an angle of
1-2 °.

The study begins with the distal parts of the limbs (terminal phalanges), then
move to more proximal joints. Passive movements in
joints should be done not too smoothly, but not in jerks. If the patient does not
distinguishes between light movements, their amplitude is increased. You need to
touch the limbs
easy, avoid unnecessary exposure to cutaneous exteroceptors.

The sense of position is investigated as follows : the limbs give a certain
position, and the patient (with closed eyes) is asked to describe in what position
there is a limb. You can suggest giving the same position to a healthy
limbs or open his eyes, look and ask if he imagined
limb position.

Skin **kinesthesia** is checked by displacing a fold of skin, and the patient is asked to
determine
direction of movement.

A deep **feeling of pressure and weight is** also involved.
sensitivity. These sensitivities are rarely tested in the clinic.

The examinee should distinguish pressure from touch and note the difference in
degree
the applied pressure. Roughly, this is checked by squeezing with different strength.
muscle or by pressing on the skin. A more detailed study of the feeling of pressure
and weight

carried out with the help of weights of different masses, placed on certain areas limbs or torso.
The feeling of weight is determined by a set of weights placed on the palm of an outstretched hand
of the subject. Normally, a difference in the weight of the cargo of 10% is detected. Square
the touch should be the same.
Perception of the sensation of vibration (pallesthesia) occurs when arousal deep receptors by fluctuations of a certain frequency and amplitude. In clinical practice use low frequency tuning forks (64-128 Hz). Vibrating foot
the tuning fork must be placed on the bony protrusion. Determine if there is a sensation of vibration,
its duration (in seconds) and intensity. The intensity is found out by comparisons with the sensation in a symmetrical area. When the patient has ceased to feel

vibrations of the tuning fork, the latter is immediately transferred to a known healthy area, where
vibration will still be felt (normally, vibration is felt for 9-11 s).
All the described methods were related to the study of the so-called simple types of sensitivity.
Complex types of sensitivity include: two-dimensional
feeling, feeling of localization, feeling of discrimination, stereognosis.
Exploration of two-dimensional spatial sense
is carried out like this:
the examiner draws with a blunt object on the patient's skin numbers, letters or simple figures (triangle, circle, cross); the patient must recognize them with closed eyes zami. It is necessary to write without much pressure, the sizes of the figures and figures depicted are not
must be very large.
Sense of localization is tested by applying to different parts of the body tactile irritation; the patient must determine the place of touch. Fine
the subject indicates this with an accuracy of 1 cm.
Discriminatory feeling - the ability to distinguish between 2 at the same time applied irritation on closely spaced points of the body surface.
The study is carried out using a Weber compass or Siveking esthesiometer.
You can use a compass for drawing by measuring the distance between its legs millimeter ruler. Bringing the legs of the compass closer and apart and at the same time touching
them of the skin or mucous membrane, find out from the subject whether he distinguishes both
touch or perceives them as one. The minimum distance at which irritation is perceived as double, unequally on different parts of the body (within from 1 mm at the tip of the tongue to 60-70 mm in the scapular region).
Stereognostic feeling is the ability to recognize familiarity by touching

object with closed eyes (coin, key, safety pin, matchbox, etc.).
A healthy person usually recognizes the subject correctly and correctly characterizes its qualities.
(density, softness and other properties).
In primary astereognosis, superficial and deep sensitivity remains;
unrecognition of the subject depends on the violation of analytic-synthetic activity the cortical section of the analyzer. It should be noted, however, that some authors rejected
the existence of a "pure" astereognosis is believed and it is believed that with astereognosis it is always
elementary sensitivity suffers to one degree or another.
To study the sensitivity, special devices are used:
Rudzita algesimeters - for pain sensitivity, thermoesthesiometers, in particular
Roth thermoesthesiometer - for temperature, baresthesiometers - for the study of sens-wa pressure. In addition, Frey's hairs and bristles, Weber's compasses, and others are used.
Pathology of sensitivity. General sensitivity conductors are included in
the composition of almost all parts of the nervous system, therefore, they are often affected.
Symptoms of irritation, loss and perversion of function can be distinguished.
sensitive conductors.
The most common symptom of sensory neuron irritation is pain. The generally accepted there is no definition of pain. **Pain** is a real subjective sensation caused by the applied irritation or pathological changes in body tissues. The pain is peculiar
psychophysiological state of a person arising from the impact of superstrong
irritants that cause changes in the body at an organic or functional level.
Pain is a signal of danger, in connection with which protective reactions come into play. O the nature of pain can only be judged by the complaints of the subject. This subjectivity assessment of pain sensations led to the need to introduce the concept of nociceptive reactions (lat.

sege - harm) in response to stimuli that can cause damage to the body.
Pain is accompanied by a number of objective changes in various functional systems.
organism, providing respiration, blood circulation, statics and kinetics, vegetative components, behavior, emotions, EEG, etc.
A double sensation of pain is known. Pain occurs immediately after application of the nociceptive
irritation and is a short-term emergency signal of irritation. After 1-2 sec pain
becomes more intense, diffused and long lasting. The initial sensation of pain is the so-called primary (epicritical) pain - due to the conduction of a pain signal through thick, quickly conductive fibers of type A. Subsequent sensation - secondary (protopathic) pain - occurs after a certain interval after the first phase, it is more diffuse and is due to
by passing the excitation through thinner, slower-conducting type C fibers.
The pain impulse is formed by the first, receptor, neuron (the cell of the spinal ganglion). However, the transmission of this impulse to the second neurons of the posterior horns of the spinal cord is controlled in
gelatinous substance by feedback mechanisms (theory of control of afferent flow at the entrance).

Excitation reaches the ventrobasal and posterolateral groups along the spinal thalamic tract. nuclei of the visual hillock. This is where the sensation of pain begins to form. An important role in
the processing of information entering the brain belongs to the reticular formation and limbic system. The reticular formation activates the cerebral cortex, and the limbic the system has a lot to do with memory, motivation and emotion.
Awareness of pain and its localization in relation to a specific area of the body are carried out when
mandatory participation of the sensorimotor area of the cerebral cortex. For differential diagnosis localization of the source of pain is extremely important knowledge of the so-called law of projection of sensations.
According to this law, a painful sensation that forms in the higher parts of the central nervous system, always refers to the initial receptor zone of a certain sensory pathway independently from where the irritation is applied. For example, for any irritation of the fibers ulnar nerve in different parts of the upper limb of a person always there is a feeling of pain in IV-V fingers of the hand, the sensitive innervation of which is provided by these fibers.
The intensity of pain depends on a number of factors: the type of higher nervous activity patient, emotional mood, etc. It is known that strong motivation, efforts of the will the patient, switching attention to any type of intellectual activity and other circumstances can help reduce or even suppress the sensation of pain. Occasionally there is a congenital absence of a feeling of pain (analgia). For short-term or long-term mental disorders (some forms of schizophrenia, extensive damage to the frontal lobes of the brain,
alcoholic intoxication), a sharp change in pain sensitivity and even painless treatment of serious diseases (myocardial infarction, stomach ulcer, etc.).
Pain sensations, according to patients, can be sharp, dull, cutting, stabbing, burning, etc. In case of damage to nerve fibers that carry out predominantly somatic innervation, somatalgia occurs, localized in the branching zone of the spinal roots or peripheral nerves. Such pains are paroxysmal or persistent, usually not accompanied by disorders of autonomic-effector innervation.
With the involvement of fibers in the pathological process, predominantly vegetative sensitive innervation - of the sympathetic nervous system, sympathetic (vegetalgia) develops. Such pains diffuse, deep, pressing, burning, can be constant or paroxysmal. Often they accompanied by spasm of peripheral vessels, goose bumps, impaired sweating, trophic disorders.
A careful analysis is required for the differential diagnosis of somatalgia and sympathetic the nature of the pain syndrome (localization, intensity, duration of pain, frequency seizures, the so-called pain pattern and its coincidence with the nerve innervation zone or spinal root, persistence or paroxysmal pain), finding out the presence or absence of concomitant disorders of autonomic-effector innervation, efficiency medications that affect different levels of pain integration, etc.
Pains are distinguished by local (local), projection, radiating and reflected (reflex).
Local pain occurs in the area of existing painful irritation. The reason for its appearance is often there are diseases of bones, joints, ligaments, accessory nasal cavities, etc.
Local pain during percussion of the skull, spine may indicate damage to the brain and its shells. Often, the localization of pain does not coincide with the site of the existing irritation. This is often
observed with radiculitis (lumbosacral, cervical, chest), when, for example, the degeneratively altered intervertebral disc compresses the fibers of the first sensory neuron (most often the posterior root suffers). As a result, there is acute pain in the innervation zone.

this spine. The pain is shooting, “lancing” (tearing) character. Since localization
pain does not coincide with the site of the existing irritation, such pain is called projection. An example
can serve as pain in case of injury of the elbow joint - irritation of the ulnar nerve passing into the sulcus
ulnaris, causes pain in the IV-V fingers. This also includes the so-called phantom pain in persons amputated: irritation in the stump of the severed nerves causes a sensation of pain in
missing parts of the limb (fingers, hand, foot, heel). The existence of phantom pain
proves that the cerebral cortex is involved in the formation of pain sensation (occurs
excitation of its areas associated with the amputated part of the limb). Pain can be
irradiating, that is, spreading from one branch of the nerve, irritated by the pathological
process, to another, free from immediate irritation. For example, with pulpitis only
one tooth, irritation of the corresponding branch of the trigeminal nerve occurs, however, pain can
irradiate to the zone of innervation and other branches. The result of the same irradiation of irritation
are reflected pain in diseases of internal organs. Pathological impulses from
receptors of internal organs, entering the posterior horn of the spinal cord, excite conductors
pain sensitivity of the corresponding dermatomes, where the pain spreads. This is called
viscero-sensory phenomenon, and the area of the skin where the pain is projected is the Zakharyin-Ged zone. In these
zones can be determined not only pain, but also hypersensitivity. Reflected pains have
important in the diagnosis of diseases of internal organs.
Causalgia - paroxysmal burning pains that worsen with light
touch, breath of air, negative emotions and are localized in the affected area
peripheral nerve. The symptom of a "wet rag" is characteristic - patients experience relief from applying a damp cloth to the painful area. Causalgia often occurs with partial damage
denii of the trunks of the median and tibial nerves.
Pain can occur in response to compression or tension on a nerve or root. These pains are called reactive. These include clinical symptoms such as Valle's pain points. Easier to identify them
in places where the nerve trunks are located superficially or close to the bone (for example, paravertebral at the transverse processes, in the supraclavicular fossa, in the middle third of the internal
surface of the shoulder, along the sciatic nerve). The study of pain points should be carried out with
closed eyes of the patient. Pain associated with peripheral nervous system disease, usually intensified with active movements due to the tension of the nerve trunks. So, the forced slope
head can lead to pain in the area of innervation of the affected spinal root (symptom
Neri), the same occurs when coughing, sneezing, straining. Flexion in the hip joint straight
a lazy lower limb with lumbosacral radiculitis causes pain in the lower back and
the back of the thigh and lower leg - the first phase of the Lasegue symptom, flexion in the knee joint
eliminates the arising pain, and further lifting of the lower limb becomes possible -
second phase of Lasegue symptom. When raising the lower limb, pain occurs along the anterior the surface of the thigh and in the groin area - Wasserman's symptom; the same pain occurs when bending
in the knee joint - a symptom of Matskevich.
In addition to the pain arising from irritation of the receptors and conductors of pain sensitivity, there may be spontaneously arising unpleasant sensations. To them include **paresthesia** (tingling sensation, crawling, burning sensation, etc.).

Paresthesias are usually short-term, often appear at night in patients with compression-ischemic lesions of the nerves of the extremities (tunnel syndromes). Certain tests can provoke the appearance of paresthesia.
Wrist extension test : passive maximum wrist extension in wrist joint for 1 min. In the presence of tunnel syndrome, such extension of the hand causes tactile paresthesias in I-IV fingers of the hand. it due to increased compression of the contents of the carpal tunnel between the flexor retainer and the base of this canal. As a result of this compression ischemia of the fibers of the median nerve deepens.
Wrist flexion test: maximum passive flexion of the wrist at the wrist joint for 1 min). Patients with carpal tunnel syndrome develop paresthesia in the I-IV fingers of the hand. This is due to compression of the median nerve of the tendon - mi of the flexors of the fingers and the holder of the flexors between which this nerve.

Raised upper limb test. The patient is in a lying position on back or sitting; he raises the extended upper limbs up and holds them in within 1 min. The early appearance of paresthesia in the fingers of the hand (after 10-15 s) indicates on high extracellular (tissue) pressure due to venous stasis and lymphostasis and a significant degree of compression of the corresponding nerve in a particular channel. At this position of the upper limbs decreases the hydrostatic pressure in the arteries their distal section, which leads to a violation of microcirculation in the trunk of the of the female nerve and the occurrence of paresthesia.
Cuff test. A tonometer cuff is applied to the patient's shoulder, in which maintain a pressure of 10-15 mm Hg. Art. above the maximum pressure in brachial artery. The sample is considered positive if, within one minute there are paresthesias in the fingers of the hand. The stopwatch measures the time from the beginning compression until paresthesia appears. Clinical observations show that the early onset of paresthesia is a sign of pronounced compression ischemic nerve damage. As the disease progresses, a detectable The "latent" period of time is gradually reduced, and after adequate treatment lengthens. Some researchers recommend cuff compression lasting up to 5 minutes. In this case, not only paresthesias occur, but also hypoesthesia in the fingers of the diseased hand.
Tinel's test: percussion with a hammer on the palmar surface of the hand at the level of the flexor retinaculum. The percussion is repeated 5-6 times with increasing impact, as when evoking deep reflexes. Paresthesias with such percussion occur disappear instantly and usually quickly. Sometimes after percussion for some the feeling of numbness persists over time. Paresthesias are of diagnostic value, appearing in the zone of innervation of the median nerve on the hand (fingers I-III).

Excessive irritation of the sensitive conductors may occur, and then
the injections applied during the examination are perceived as very intense. Such
cases speak of **hyperesthesia,** with pain sensitivity - **hyperalgesia** .
Sometimes even when cutting a peripheral nerve in the corresponding area of the skin
sensitivity to painful irritations may be lost, however, in the anesthesia zone
there may be a sensation of pain - the so-called anesthesia dolorosa. Irritation
the central section of the nerve transmits excitation to certain parts of the brain, which
often perceived as pain in the corresponding skin area.
When the sensitivity conductors are interrupted (damaged), symptoms appear
loss - loss of sensitivity (any kind of superficial and deep), which
is called **anesthesia** , and lowering it is **hypesthesia** . Loss of pain sensitivity
denote by the term analgesia, lowering - **hypalgesia.**
In the clinic, you often have to meet with a peculiar form of the disorder.
pain sensitivity - **hyperpathy** . It is characterized by an increase in the threshold
perception. The patient does not feel single injections, but a series of injections (5-6 or
more)
causes intense and painful pain that occurs through some latent
period as if suddenly. The patient cannot indicate the place of the injection. Solitary
irritations are perceived as multiple, the zone of these sensations expands.
The perception of sensations remains after the cessation of irritation.
(aftereffect). Sometimes the shots are perceived as hot or cold sensations
(temperature dysesthesia). Hyperpathy occurs when various links are affected
skin analyzer from the peripheral region to the thalamus and cerebral cortex.
Were identified 2 types of sensitivity: the more primitive "protopathic",

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caused by gross irritations, and "epicritical", providing perception
more subtle and differentiated influences. On the modern interpretation of these
types of sensitivity discussed above.
Pain sensitivity disorder is often accompanied by a violation
temperature sensitivity. Its complete loss is denoted by the term **thermo-
anesthesia** , lowering - **thermal hypesthesia.** Perversion may occur occasionally
perception of heat and cold - **thermodysesthesia** .
Sometimes the perception of cold and warmth can be upset separately.
Anesthesia, hypesthesia and hyperesthesia can occur in disorders of such
strong sensitivity.
Qualitative violations of surface sensitivity are associated with
perversion of the content of the received information and are clinically manifested
bifurcation of pain (when pricked with a needle, the subject first feels
touch and only after a while pain); **polyesthesia** (single
irritation is perceived as multiple); **allocheiria** (irritation of the patient
localizes not where it is applied, but on the opposite half of the body, usually in
symmetrical section); **synesthesia** (a feeling of irritation not only in its place
application, but in any other area); **dysesthesia** (distorted perception
“The receptor accessory of the stimulus, for example, heat is perceived as
cold or vice versa, an injection - like a touch of hot, etc.). Violation of all kinds

deep sensitivity is called **bathyanesthesia** and is accompanied by sensory ataxia.
There are also partial loss, for example, with multiple sclerosis sometimes only vibration sensitivity is upset. Loss of ability recognizing familiar objects by feeling is called astereognosis.
What signs of sensitivity disorder should be used in topical diagnosis?
First of all, one must take into account the localization of the zone of altered sensitivity. it refers to the phenomena of not only loss, but also irritation (pain, paresthesia). Anesthesia area or another type of sensitivity disorder can always be attributed to a certain level disturbed afferent pathway. This is the basis for topical diagnostics according to these disorders. sensitivity. All types of sensitivity do not always suffer; some of them can be dropped, others persist. Violation of some types of sensitivity, while others are preserved, is called dissociated disorders. Dissociated anesthesia indicates a lesion in an area of the brain where conductors of different modality pass separately. This is especially true for defeat posterior horns and anterior commissures of the spinal cord, as well as foci in the brain stem. An important criterion for topical diagnosis is the coincidence or mismatch of localization movement and sensory disorders. With half-transverse spinal cord injury due to different levels of transition of nerve fibers from one side to the other, there is a peculiar picture: motor loss develops on one side, and sensitive - on the other is the so-called Brown-Séquard syndrome.
It is advisable to record the results of the study of different types of sensitivity graphically. To do this, you can use special forms - schemes of neural and segmental innervation, on which are applied zones of violations of sensitivity.
Variants of the distribution of sensitivity disorders. With damage to the peripheral nerve sensitivity disorders coincide with the zone of his innervation. All types of sensitivities are affected.
nosti. However, the border of anesthesia is usually less than the area of anatomical innervation due to overlapping with adjacent nerves.
The type of distribution of sensitivity disorders with damage to the nerve trunks is called **neural .** These disorders are accompanied by paralysis or paresis of the corresponding muscles.
A peculiar picture of sensitivity disorder is observed with polyneuritis.
Sensitivity is impaired in the ends of the upper and lower extremities. Figuratively such the picture is called "gloves, stocking" anesthesia. The severity of the disorder gradually decreases in the direction from the end of the limb to its root. This type of distribution of sensitivity disorders

called **distal or polyneuric** . Simultaneously with sensitivity disorder and pain for polyneuritis is characterized by paralysis and paresis of the limbs, also with distal distribution.
The defeat of the nerve plexuses (cervical, brachial, lumbar and sacral) is manifested anesthesia or hypesthesia of all types of sensitivity in the area of the projection of the nerve plexus; v pains and paresthesias appear in the same area.
The defeat of the posterior spinal roots leads to a sensory disorder in corresponding dermatomes. When you turn off one spine, no loss of sensitivity is detected as a result of compensation by adjacent roots (dermatomes come one after another, like shingles plates).

On the trunk, dermatomes are located in the form of transverse stripes, on the longitudinal limbs, on
buttock in the form of concentric semicircles. Sensory disorders in defeat
roots correspond to the indicated distribution. For radicular lesions especially
characteristic irritative phenomena in the form of pain and paresthesia in the corresponding dermatomes.
If the spinal ganglion is involved, a rash may appear in the affected dermatome.
vesicles - shingles (herpes zoster).

Sensory disorders with damage to the spinal cord at various levels.

The pathological process (trauma, inflammation, swelling) often leads to transverse damage
spinal cord. In this case, the afferent conductors can be interrupted. All kinds
sensitivity below the level of the lesion turn out to be upset (spinal type
conduction disorder of sensitivity). Such a distribution of sensitivity disorders in
the clinic is called paraanesthesia. In this case, the patient also suffers from efferent systems, in particular
pyramidal bundle, paralysis of the lower extremities develops - lower spastic paraplegia.
A sensitivity study in such patients helps to determine the level of damage (upper
the border of the pathological focus). If, for example, the upper limit of pain disorder
sensitivity is at the level of the navel (Tx segment), then roughly we can say that on
this segment of the spinal cord is also the upper border of the pathological focus. When conducting
topical diagnosis, it is necessary to remember about the peculiarities of the transition of the dorsal-thalamic fibers
in the spinal cord from one side to the other. In this regard, the upper boundary of the focus must be
move 1-2 segments up and in this case consider it to be at the Tix or Tvsh level.
Determination of the focus of the focus is important in the localization of the tumor, when deciding the question of
the level of the operation. It should be borne in mind that there is a mismatch
segments of the spinal cord and vertebrae, which in the lower thoracic and lumbar regions is already 3-4 vertebrae.
Let us consider the clinical picture of the lesion of one half of the spinal cord cross section. V
In this case, on the side of the focus, the articular-muscular feeling is upset (the back
cord), spastic paralysis of the lower limb occurs (break of the crossed pyramidal
beam). On the side opposite to the focus, there is a loss of pain and temperature
sensitivity of the conductive type (the dorsal-thalamic tract is damaged in the lateral
rope). This clinical picture is called Brown-Séquard's palsy.
Conduction disorders of sensitivity are also found with pathological foci in the posterior
ropes. At the same time, the joint-muscular and vibrational feeling on the side of the focus is lost
(turning off the beams of thin and wedge-shaped). Sometimes tactile sensitivity also falls out.
Switching off the posterior cords is observed with tabes dorsalis, or lack of
the accuracy of vitamin B12, or with myeloischemia, which is manifested by sensitive ataxia and paresthesias.
With lesions of the spinal cord, a segmental type of sensitivity disorder occurs .
This happens when the posterior horn and the anterior white commissure of the spinal cord are damaged. In the back horn
the bodies of the second neurons of the pathway are located, the conductive impulse of pain and temperature
sensitivity from the corresponding dermatomes. If the posterior horn collapses over
of several segments, in the corresponding dermatomes of their side, loss of painful
and temperature sensitivity. Tactile and deep sensitivity is preserved, it

the guides find themselves outside the hearth zone, and, entering the Lissauer marginal zone, they immediately go
into the posterior cord. As mentioned, this type of sensitivity disorder is called
dissociated anesthesia. With damage to the anterior spinal adhesions, it also develops
dissociated anesthesia, in this case in several dermatomes on both sides. Genesis of such
distribution of anesthesia will become clear if we remember that the axons of the second neuron, conducting
pain and temperature sensitivity, throughout the entire spinal cord pass from one
side to the other, which leads to bilateral disorder.
With the localization of the focus in the anterior commissure of the spinal cord at the level of the lower cervical and thoracic

segments of the sensitivity disorder develop in the form of a "jacket" - spinal, segmental
type of. Dissociated anesthesia of such localization occurs in syringomyelia (foci of endogenous
the growth of glia, sometimes with decay and the formation of cavities in the gray matter of the spinal cord). That
the same is observed in vascular diseases and with intramedullary tumors.
With cerebral type of sensitivity disorders due to brain damage
conduction anesthesia is always on the opposite side. With the defeat of the right
hemisphere, left-sided hemianesthesia develops and vice versa. Therefore, the border of pathology
and the norm is not a horizontal (transverse) line, but a vertical - the median line of the body.
With organic (destructive) lesions, the border of the anesthesia zone by 2-3 cm does not reach the midline of the body and head due to the overlapping of adjacent sensitive zones one after the other.
As you know, in the posterior leg of the inner capsule, thalamocortical fibers that conduct all types of sensitivity, pass in a compact beam. Lesions in this area of the brain
(softening or hemorrhage) lead to the development of hemianesthesia in the opposite half
body. The distal extremities are more affected. If the rear leg of the inner leg is damaged
bags and the pyramidal bundle are involved. Hemianesthesia on the side opposite to the lesion will
be combined with hemiplegia.
In case of damage to the area of the postcentral gyrus (cytoarchitectonic fields 3, 1, 2)
anesthesia does not cover the entire opposite side of the body, but only the projection zone of the focus. Ras-
construction
sensitivity is limited to the limits of the upper or lower limbs, or
torso. Sometimes anesthesia occupies the distal part of the upper or lower limb from the upper transverse border - anesthesia of the "glove" or "stocking" type. Usually the deepest
sensitivity.
The pathological focus can capture not only the postcentral gyrus, but also the superior and inferior parietal lobules (fields 5, 7). In this case, complex types of sensitivity are upset, astereognosis appears, disorders of discriminatory feelings, unrecognition of those depicted on skin of numbers, other signs, violation of the body scheme (the patient's idea of \ u200b \ u200bthe proportions
your body, the position of the limbs). It may seem to the patient that he has an "extra"
inferiority (pseudomelia), or, conversely, one of the limbs (amelia) is missing. Symptoms lesions of the superior parietal region is also autopagnosia - inability to recognize parts
own body; anosognosia - misunderstanding of one's own defect of the disease, for example, a patient
denies that he has paralysis (usually left-sided).

With a cortical pathological focus (tumor, scar, arachnoid cyst), symptoms are possible not only loss, but also irritation of the afferent conductors. It can manifest attacks of various paresthesias in the corresponding areas of the opposite side of the body (so called sensory type of partial epilepsy). Paresthesias can spread to the entire half of the body and end with general convulsions.
With the defeat of the visual hillock, a whole complex of peculiar sensitive disorders. Heterolateral hemianesthesia appears, often with a seizure of the face. On the side hemianesthesia, excruciating, stitching, burning pains occur, periodically intensifying and bad amenable to cropping. The slightest touch to the skin, pressure, cold increase the pain attack. Pains are poorly localized by patients, usually radiating to the entire half of the body, sometimes
more pronounced at rest and slightly diminished on movement. The study reveals decrease in superficial sensitivity with symptoms of hyperpathy, violation of deep sensitivity is especially pronounced, which leads to sensitive ataxia.
Dorsal
thalamic and bulbothalamic bundles. The defeat of the conductors leads to anesthesia against false half of the body. Dorsal thalamic bundle, which conducts pain and thermal impulses, can be damaged in isolation. This happens with vascular disorders in the basin of the lower and superior cerebellar arteries. In this case, hemianesthesia has a dissociated character - pain and temperature drops out and deep and tactile sensitivity remains.
With a focus in the lateral part of the medulla oblongata, except for the spinal thalamic of the bundle, the spinal tract and the nucleus of the trigeminal nerve are also involved in the process. Defeat them
leads to anesthesia of the face on the side of the same name. There is a kind of distribution sensitivity disorders: anesthesia of the face on the side of the focus and dissociated hemianesthesia on
opposite side. This picture is called alternating hemianesthesia.
With small focal lesions of the reticular formation, especially in the mesencephalic regions the brain, as well as the cerebral hemispheres, various mosaic variants of the spotted hypoesthesia.
Functional (hysterical) hemianesthesia is characterized by loss of all types sensitivity or predominantly painful on one side of the body with a border passing

strictly along the midline.
The upper border with functional paraanesthesia is located more horizontally than corresponding to a dermatome, the border of which on the dorsal side of the trunk is always higher than on
ventral.
So, depending on the localization of the lesion in the nervous system, there are 3 types distribution of sensitivity disorders.
When the spinal roots or peripheral nerves are damaged, one of the variants of the peripheral type of sensitivity disorder: neural - *a* violation of all types sensitivity in the area supplied by the affected nerve; polyneuritic - disorders arise symmetrically in the distal extremities; radicular - violation of all types sensitivity in the area of the corresponding dermatomes.
When the spinal cord is damaged, several variants of the spinal type also develop. disorders of sensitivity: segmental - dissociated disturbance of sensitivity (loss pain and temperature while maintaining tactile and deep) in the same zones as with root damage (in the dermatome zone); conductive - violation of sensitivity on the entire part body below the level of the lesion (paraanesthesia).
With brain damage, a cerebral type of sensitivity disorder develops as well

in the form of several options: conductive - sensitivity disorder on the opposite
half of the body (hemianesthesia, occasionally alternating), cortical - the zone of hypesthesia varies in
depending on the location of the lesion in the postcentral gyrus (usually monoanesthesia).

SYMPTOMS OF SPINAL CORD AND SPINAL ROOTS INJURIES

The spinal cord is the phylogenetically most ancient part of the central nervous system,
located in the spinal canal and surrounded by the meninges. Its upper border is
corresponds to the exit of the first pair of spinal nerve roots and is projected onto the edge of the large
occipital foramen. Above, it passes into the medulla oblongata, and below it ends in the cerebral
a cone at the level of Li - n vertebrae. The length of the spinal cord depends on the height of the person and is 42-
46 cm. Conventionally, 5 sections are distinguished in the spinal cord:
cervical - pars -cervicalis (Ci-Cvш);
chest - pars thoracica (Ti - Tsh);
lumbar-pars lumbalis (Li - Lv);
sacral - pars sacralis (Si - Sv)
coccygeal - pars coccigea (Coi - Sop).
The structure of the spinal cord is metameric, which is manifested in the segmental arrangement of the 31-33rd
pairs of spinal nerve roots. A segment is a segment of the spinal cord corresponding to two
pairs of roots. There are thickenings in the cervical and lumbosacral spinal cord. V
the cervical enlargement includes the lower cervical segments (Cv - Cvsh) and the upper thoracic segment. Part
The lumbosacral enlargement includes all lumbar (Li - Lv) and 2 sacral (Si - Sp) segments.
The cervical thickening is located in the spinal canal at the level of Csh - Cvp, lumbosacral -
at the level of Tx - Txp.
The spinal cord is a cylindrical cord, the diameter of which is
the section is 10 mm, in the area of the cervical thickening - 12-14 mm, in the lumbosacral - 11-13 mm.
In the process of ontogenesis, the spinal cord grows more slowly than the spinal column, and therefore
an adult, its length does not correspond to the length of the spinal column - it ends at the level
Li or the upper edge of the body Lп. At the level of the cervical spine, the difference between the corresponding segments
and vertebrae is 1 vertebra, in the upper thoracic region - 2, and in the lower thoracic - 3 vertebrae. On
the level of the lumbar and sacral vertebrae is the cauda equina, consisting of spinal
spinal roots Lii - Sop and the terminal filament of the spinal cord.
The entire length of the spinal cord is divided into 2 symmetrical halves of the anterior median slit and posterior median sulcus.
On a transverse section of the spinal cord, a gray matter in the shape of a butterfly is distinguished, and
surrounding white matter. The gray matter is divided into anterior and posterior horns. At the seg-

cents Cviii - Liii gray matter forms lateral horns. The ratio of gray to white matter per its different levels are not the same. In the center of the spinal cord is the central canal. In white substance distinguish between paired anterior, lateral and posterior cords, separated on each side the anterior roots of the spinal nerves and the posterior horns of the gray matter.
A segment of the spinal cord is part of the body metamer, which also includes a specific area of skin (dermatome), muscle (myotome), bone (sclerotome) and internal organs (splanchnotom), innervated by one segment.
The gray matter is composed of neurons and glial elements. There are the following nervous cells: motor (motoneurons) of three types - α- large, α- small and γ-neurons. They have in the anterior horns, and from their axons, the anterior roots are formed and then all structures peripheral nervous system (plexus, nerves); sensitive are located in the hind horns and are the second neurons of pain temperature sensitivity, their axons go to the opposite half of the spinal cord in front of the central canal (anterior white commissure) and form dorsal thalamic tract; cerebellar proprioceptor cells are located at the bases of the posterior horn, their axons form the anterior and posterior spinal pathways. Autonomic (sympathetic and parasympathetic neurons) are located mainly in lateral horns and are visceromotor cells; their axons are part of the anterior roots. Associative multipolar small-sized nerve cells are located throughout gray matter and provide intersegmental and intercordial connections of their own and opposite sides.
The white matter of the spinal cord consists of myelinated fibers and is divided into cords in depending on the location in relation to the gray pillars. The posterior cords are located between the back posts, the side posts between the back and front posts, and the front posts between front posts.
The posterior cords are formed by ascending conductors of deep, tactile and vibrational sensitivity. The conductors of deep sensitivity are located medially from the lower limbs (thin bundle), laterally - from the upper limbs (wedge-shaped bundle).
Descending and ascending conductors are located in the lateral cords of the spinal cord. TO descending are the pyramidal (lateral cortical-spinal) pathway, red-nuclear

spinal and reticular-spinal tract. All descending paths end at cells anterior horn of the spinal cord. The ascending paths are located as follows: along the outer the edge of the lateral cord is the spinal anterior and posterior pathways; inward from the front the ascending fibers of superficial sensitivity pass through the spinal cord (lateral dorsal-thalamic tract).
The anterior cords of the spinal cord are formed mainly by descending paths from precentral gyrus, subcortical and stem nuclei to the cells of the anterior horns of the spinal cord (anterior uncrossed pyramidal pathway, vestidoor-spinal, olivospinal, anterior reticular-spinal and teglospinal tract. In addition, in the front a thin sensitive bundle passes through the cords - the anterior dorsal-thalamic pathway.
The clinical picture of focal lesions of the spinal cord is very variable and depends on the prevalence of the pathological process along the long and transverse axes of the spinal cord.
Syndromes of lesion of individual sections of the spinal cord cross section. Syndrome the anterior horn is characterized by peripheral paralysis with muscle atrophy, innervated damaged motor neurons of the corresponding segment - segmental or myotomic paralysis (paresis). Fascicular twitching is often observed in them. Above and below the muscle focus remain unaffected. Knowledge of the segmental innervation of muscles allows quite accurately localize the level of spinal cord injury. Tentatively with the defeat of the cervical thickening the spinal cord affects the upper limbs, and the lumbar - the lower. The efferent part is interrupted reflex arc, and deep reflexes drop out. Selectively, the anterior horns are affected when neuroviral and vascular diseases.
Dorsal horn syndrome is manifested by dissociated sensory impairment (decreased

pain and temperature sensitivity while maintaining the joint-muscular, tactile and vibration) on the side of the lesion, in the area of its dermatome (segmental type of disorder sensitivity). The afferent part of the reflex arc is interrupted, therefore deep reflexes. This syndrome is usually found in syrin-gomyelia.
Anterior gray commissure syndrome is characterized by symmetrical bilateral disorder pain and temperature sensitivity while maintaining the joint-muscular, tactile and vibration sensitivity (dissociated anesthesia) with segmental distribution. Arc deep reflex is not disturbed, reflexes are preserved.
Lateral horn syndrome is manifested by vasomotor and trophic disorders in the area vegetative innervation. With a lesion at the level of Cvsh - Ti, Claude Bernard's syndrome occurs - Horner on the homolateral side.
Thus, for the lesion of the gray matter of the spinal cord, it is characteristic to turn off functions of one or more segments. Cells located above and below the lesions continue function.
The lesions of the white matter, which is a collection of individual bundles of fibers. These fibers are the axons of nerve cells located at a considerable distance from the cell body. If such a bundle of fibers is damaged even on insignificant length and width, measured in millimeters, the ensuing disorder in functions covers a significant area of the body.
Dorsal cord syndrome: joint-muscular feeling is lost, partially reduced tactile and vibration sensitivity, sensitive ataxia and paresthesias appear on the side the focus below the level of the lesion (with damage to a thin beam, these disorders are found in the lower limbs, wedge-shaped bundle - at the top). This syndrome occurs in syphilis of the nervous systems, funicular myelosis, etc.
Lateral cord syndrome: spastic paralysis on the homolateral side, loss pain and temperature sensitivity on the opposite side 2-3 segments below the focus defeat. With bilateral damage to the lateral cords, spastic paraplegia develops or tetraplegia, dissociated conduction para-anesthesia, dysfunction of the pelvic organs of the central type (urinary retention, feces).
Syndrome of lesion of half the diameter of the spinal cord (Brown-Séquard syndrome): on the side lesions develop central paralysis and turn off deep sensitivity (defeat of the pyramidal tract in the lateral cord and thin and wedge-shaped bundles - in the posterior);
disorder of all types of sensitivity of the segmental type; peripheral muscle paresis the corresponding myotome; vegetative-trophic disorders on the side of the focus; conductor dissociated anesthesia on the opposite side (destruction of the spinal thalamic bundle in lateral cord) 2-3 segments below the lesion. Brown-Séquard syndrome occurs when spinal cord injuries, extramedullary tumors, rarely - with ischemic spinal strokes (impaired circulation in the sulco-commissural artery supplying

one half of a cross section of the spinal cord; the posterior cord remains unaffected - ischemic Brown-Séquard syndrome).
The defeat of the ventral half of the spinal cord diameter is characterized by paralysis lower or upper limbs, conductive dissociated para-anesthesia, impairment functions of the pelvic organs. This syndrome usually develops with ischemic spinal stroke in basin of the anterior spinal artery (Preobrazhensky syndrome).
Complete spinal cord injury is characterized by spastic inferior paraplegia or tetraplegia, peripheral paralysis of the corresponding myotome, paraanesthesia of all types, starting with a specific dermatome and below, dysfunction of the pelvic organs, vegetative trophic disorders.
Syndromes of damage along the long axis of the spinal cord. Let's consider the main options lesion syndromes along the long axis of the spinal cord, meaning complete transverse lesion in

every case.
Upper Cervical Segment Syndrome (Ci - Cv): Spastic Tetraplegia
sternocleidomastoid, trapezius muscles (XI pair) and diaphragm, loss of all types
sensitivity below the level of the lesion, impaired urination and defecation in the central
type. With the destruction of the Ci segment, dissociated anesthesia on the face in the posterior
Zelder dermatomes (turning off the lower divisions of the trigeminal nerve nucleus).
Cervical thickening syndrome (Cvi - Ti): peripheral paralysis of the lower extremities
noses, loss of all types of sensitivity from the level of the affected segment, dysfunction
pelvic organs in the central type, bilateral Claude Bernard-Horner syndrome (ptosis, miosis,
enophthalmos).
Thoracic segment lesion syndrome (Ti - Thp): spastic lower paraplegia, loss
all types of sensitivity below the level of the lesion, central pelvic dysfunction
organs, expressed vegetative-trophic disorders in the lower half of the body and lower
limbs.
Lumbar thickening syndrome (Li-Sp): flaccid lower paraplegia, paraanesthesia
on the lower limbs and in the perineal region, central pelvic dysfunction.
Syndrome of lesion of the segments of the epicone of the spinal cord
(Liv-Sп): symmetrical
peripheral paralysis of myotomes L IV - Sp (muscles of the posterior thigh group, muscles of the
lower leg, foot and
gluteal muscles with loss of Achilles reflexes); paraanesthesia of all types of sensitivity to
legs, feet, buttocks and perineum, retention of urine and feces.
Spinal cord cone segment lesion syndrome: anesthesia in the anogenital area
("Saddle" anesthesia), loss of the anal reflex, dysfunction of the pelvic organs by
peripheral type (urinary incontinence, feces), trophic disorders in the sacral region.
Thus, in case of damage to the entire diameter of the spinal cord at any level, the criteria
for topical diagnosis, the prevalence of spastic paralysis (lower paraplegia
or tetraplegia), the upper limit of sensitivity disorders (pain, temperature). Especially
informative (in diagnostic terms) the presence of segmental movement disorders (flaccid paresis
muscles that make up the myotome, segmental anesthesia, segmental autonomic disorders).
The lower limit of the pathological focus in the spinal cord is determined by the state of function
the segmental apparatus of the spinal cord (the presence of deep reflexes, the state of trophism of
muscles and
getative-vascular provision, the level of causing symptoms of spinal automatism, etc.).
The combination of partial lesions of the spinal cord along the transverse and long axis on different
levels are often found in clinical practice. Let's consider the most typical options.
Syndrome of defeat of one half of the transverse section of the segment (Ci-p): subbulbar
alternating hemianalgesia, or Opalsky's syndrome, - a decrease in pain and temperature
sensitivity on the face, symptom of Claude Bernard-Horner, paresis of the limbs and ataxia on the
side
hearth; alternating pain and temperature hypesthesia on the trunk and extremities
the opposite side to the hearth. This syndrome occurs when the branches of the posterior
spinal artery, as well as in the neoplastic process at the level of the craniospinal
transition.
Syndrome of lesion of one half of the diameter of the Cvsh - Ti segments (combination of syndromes
Claude Bernard-Horner and Brown-Séquard): on the side of the lesion - Claude Bernard-Horner
syndrome (ptosis,
miosis, enophthalmos), increased skin temperature on the face, neck, upper limb and upper part
chest, spastic paralysis of the lower limb, prolapse of the articular-muscular,
vibration and tactile sensitivity on the lower limb; contralaterally
conduction anesthesia (loss of pain and temperature sensitivity) with an upper limit of
dermatome Tp-sh.
Syndrome of defeat of the ventral half of the lumbar thickening (Stanilovsky syndrome -

Tanona): lower flaccid paraplegia, dissociated paraane-sesthesia (loss of pain and temperature sensitivity) with the upper limit on the lumbar dermatomes (Li - Lsh), dysfunction central type of pelvic organs: vegetative-vascular disorders of the lower extremities.
This symptom complex develops with thrombosis of the anterior spinal artery or its formative a large radiculomedullary artery (Adamkevich artery) at the level of the lumbar thickening.
Inverted Brown-Séquard syndrome is characterized by a combination of spastic paresis one lower limb (on the same side) and dissociated sensory disorder (loss of pain and temperature) segmental-conductive type. Such a disorder occurs with small focal lesions of the right and left halves of the spinal cord, as well as in violation venous circulation in the lower half of the spinal cord with compression of a large radicular herniated veins of the lumbar intervertebral disc (discogenic-venous myeloischemia).
Syndrome of lesion of the dorsal part of the transverse section of the spinal cord (syndrome Williamson) usually occurs with lesions at the level of the thoracic segments: a violation of the articular muscle sensation and sensitive ataxia in the lower extremities, moderate lower spastic paraparesis with Babinsky's symptom. Possible hypesthesia in the corresponding dermatomes, lungs dysfunction of the pelvic organs. This syndrome is described in posterior spinal artery thrombosis and associated with ischemia of the posterior cords and partially pyramidal tracts in the lateral cords. At the level of cervical segments, an isolated lesion of the wedge-shaped bundle with a violation deep sensitivity in the upper limb on the side of the focus.
Amyotrophic lateral sclerosis (ALS) syndrome is characterized by the gradual development mixed muscle paresis - muscle strength decreases, muscle wasting occurs, fascicular twitching, and deep reflexes with pathological signs increase. This the syndrome occurs when peripheral and central motor neurons are damaged, most often at the level medulla oblongata (bulbar ALS), cervical (cervical ALS) or lumbar enlargement (lumbar variant of ALS). It can be viral, ischemic, or dismetabolic nature.
With damage to the spinal nerve, anterior root and anterior horn of the spinal cord the function of the same muscles that make up the myotome is disrupted. With topical diagnosis in within these structures of the nervous system, the combination of myotome paralysis with sensitive violations. When the process is localized in the anterior horn or along the anterior root of violations there is no sensitivity. Possible only dull indistinct pain in the muscles of the sympathetic nature. The defeat of the spinal nerve leads to paralysis of the myotome and attachment violations of all types of sensitivity in the corresponding dermatome, as well as the appearance of pain in tails character. The area of anesthesia is usually smaller than the area of the entire dermatome due to overlapping areas sensitive innervation by adjacent posterior roots.
The following syndromes are most common.
The syndrome of lesion of the anterior root of the spinal cord is characterized by peripheral paralysis of the muscles of the corresponding myotome; with it, moderate dull pain in paretic muscles (sympathetic myalgia).
The syndrome of lesion of the posterior root of the spinal cord is manifested by intense shooting (lancinating, like "passage of an electric current impulse") pain in the dermatome area; violation- all types of sensitivity in the dermatome zone occur, deep and superficial reflexes, the point of exit of the root from the intervertebral foramen becomes painful, positive symptoms of root tension.
The syndrome of the lesion of the trunk of the spinal nerve includes symptoms of lesion anterior and posterior spinal roots, that is, there is a paresis of the corresponding myotome and destruction of all types of radicular sensitivity.
Cauda equina root lesion syndrome (Sп - Sv) is characterized by severe radicular

pain and anesthesia in the lower extremities, sacral and gluteal regions, perineal region; peripheral paralysis of the lower extremities with extinction of the knee, Achilles and plantar reflexes, dysfunction of the pelvic organs with true incontinence of urine and feces, impotence. With tumors (neurinomas) of the cauda equina roots, there is an exacerbation of pain in the vertical position of the patient (symptom of radicular pain of position - symptom of Dandy-Razdolsky). Differential diagnosis of intra- or extramedullary lesions is determined by the nature of the process of development of neurological disorders (descending or ascending type violations).

EXTRAPYRAMIDAL MOVEMENT DISORDERS

The pyramidal system and peripheral motoneurons provide voluntary muscle contractions. Each completed motor act, no matter how simple it is was, requires the coordinated action of many muscles. The quality of movement does not depend only on the type and number of muscles that implement it. The same muscles are involved in providing various movements. At the same time, the same movement can produce sometimes slower, sometimes faster, with less or more force. For execution movement, it is necessary to connect mechanisms that regulate the sequence, the strength and duration of muscle contractions and regulating the choice of the necessary muscles. In other words, the motor act is formed as a result of sequential, coordinated in strength and duration of the inclusion of individual neurons of the cortical-muscular path, giving orders to muscles, and a large complex of nerve structures outside the pyramidal system, which are combined into an extrapyramidal system, acting reflexively.

The extrapyramidal system includes cell groups of the cortex of large hemispheres (mainly of the frontal lobes), subcortical ganglia (caudate nucleus - nucl, caudatus, shell - putamen, lateral and medial pale balls - globus pallidus, Lewis' subthalamic body), in the brain stem substantia nigra, red nuclei, lamina of the midbrain roof, the nucleus of the medial longitudinal bundle (nucleus Darkshevich), a bluish place in the pons of the brain, a reticular formation with descending and ascending pathways, cerebellum, γ-motoneurons of the spinal cord, etc. Between these formations of the extrapyramidal system, there are numerous single two-way connections (closed neural circles).

Arbitrarily performing any action, a person does not think about what muscle is needed turn on at the right time, does not keep in conscious memory the working sequence of the movement an act. Habitual movements are performed imperceptibly for attention, the change of some muscle abbreviations by others are automated. These motor automatisms contribute the most economical expenditure of muscle energy in the process of performing movements. New, unfamiliar

the motor act is energetically always more wasteful than the habitual, automated one. Improving the quality side of the movement with their transfer to the most automated the economic regime is ensured by the activity of the extrapyramidal system and mainly its basal ganglia.

Morphologically and functionally striopallidal system subdivided into striatal and pallidary.

Pallidary system, phylogenetically older, includes lateral and medial pale balls, black matter, red nucleus, subthalamic nucleus. In both pale balls contains a large number of nerve fibers, large neurons in them relatively Little. The striatal system is phylogenetically “young” and includes the tailed nucleus and shell with many small and large neurons and a relatively small the number of nerve fibers. The striatal system has a somatotopic distribution: in the front sections - the head, in the middle - the upper limb and trunk, in the hindquarters - the lower limb.

The pallidal system in fish and the striopallidal system in birds are the highest motor centers, determining the behavior of these organisms. Striopallidary devices provide diffuse body movements, coordinated work of all skeletal muscles during movement, swimming, flight, etc. Higher animals and humans required a more subtle differentiation of work motor centers. In the process of evolution, a pyramidal system arose, which subjugated striopallidal system.

In human ontogenesis, myelination of striatal conductors ends by the 5th month of life (before the pyramidal system), therefore, in the first months of a child's life, lateral and medial globes pallidus are the highest motor center. Neonatal motor skills are obvious “Pallidary” features: excess, a kind of generosity of movements, rich facial expressions with a smile, etc.

with age, many movements become more and more habitual, automated, energetically calculating. The solidity and gravity of adults are a kind of triumph striopallidal system over pallidary.

When teaching purposeful movements (including professional ones, for example, playing on musical instruments, carpentry, locksmith work, driving a car, etc.) select 2 phases. During the first phase (which is conventionally referred to as pallidary) movement excessive, excessive in strength and duration of muscle contractions. Second phase (pyramidal striatal) is to gradually optimize motion control. They become energetically rational and as efficient as possible and are brought to automatism.

The extrapyramidal system has numerous neural connections between its formations, visual hillock and segmental motor apparatus of the spinal cord.

All afferent systems of the striopallidal system end in the striatum. In these systems include paths from most areas of the cerebral cortex, from the medially located nuclei of the viewer tubercle (in particular, from the medial central and parafascicular nuclei of the thalamus), from

the compact zone of the substantia nigra and the nuclei of the median suture of the midbrain. From the striatum path
go only to the lateral and medial pallidus and the reticular zone of the substantia nigra, from which, in turn, begin the main efferent extrapyramidal systems.
From the medial pallidus, axons go to the nuclei of the thalamus (medial central, ventrolateral and anterior ventrolateral nuclei). The processes of the cells of these nuclei of the thalamus are
They are located on the motor and other areas of the frontal lobe cortex.
Descending systems begin from the black matter and the pallidum, which go to the nuclei lids of the midbrain and brain stem, and from them to the motor neurons of the spinal cord in the composition of the nigro-reticular-spinal and pallido-reticular-spinal tracts.
There are ascending pathways from the substantia nigra to the nuclei of the posterior thalamus and the subthalamic nucleus.
anterior thalamus. The descending and ascending efferent tracts of the substantia nigra provide various effects on human motor activity.
Axons originate from the cells of the brain stem nuclei, which pass in the cords of the spinal cord and end in synapses with cells of the anterior horns at different levels. These include, in addition to of the above-mentioned main nigro- and pallido-reticular-spinal pathways
spinal tract (tr.vestibulospinalis), olive-spinal (tr.olivospinalis), red-nuclear cerebrospinal (tr. rubrospinalis, or Monakov's way), tecto-spinal (tr.tectospinalis), medial longitudinal bundle (fasciculus longitudinalis medialis). The most powerful is reticular-spinal tract. It consists of the axons of the cells of the reticular formation and in the dorsal the brain passes in the anterior cord (ventral part of the tract) and in the lateral cord (medial and its lateral sections). Fibers of the tegmental-spinal tract at the level of the medulla oblongata form synapses with the cells of the reticular formation, and this bundle is part of the reticular the spinal canal, together with which it descends in the anterior cord. The same must be said about passing in the lateral cord of the red-nuclear-spinal bundle. Within the oblong

the brain, a significant number of its fibers pass into the reticular substance and go down in the composition
reticular-spinal tract.
The fibers of the extrapyramidal system, like the pyramidal system, also run along the entire the cerebrospinal axis from the cortex to the lower spinal cord. However, anatomical and histological the structure of the pyramidal and extrapyramidal systems has a significant difference. The bodies of all neurons
pyramidal systems are grouped in the cerebral cortex. Schematically, the pyramidal system is cortical neurons with long axons extending to different segments of the spinal cord.
The extrapyramidal system is a long column of cells with a large number nerve fibers throughout the brain and spinal cord. This column is sharp in places increases in volume (subcortical nodes), at some levels a dense interlacing forms fibers with cell bodies (pallidus, reticular substance of the brain stem, etc.).
The discovery of the functional significance of the extrapyramidal system was facilitated by clinical and
especially clinical and anatomical observations; they identified such forms of movement disorders, which cannot be explained either by a defeat of the pyramidal system, or by a disorder of coordination
movements. Many syndromes of this kind have been described. With one of them, attention is drawn to themselves
slowness and poverty of movements, mask-like face, sluggish facial expressions, rare blinking, general
stiffness, lack of friendly hand movements when walking. Such a picture was called hypokinesis (from the Greek hypo - decrease, failure; kinesis - movement). With another type the disease develops the opposite condition. When examining the patient, they are striking

automatic violent movements. They are called hyperkinesis (from the Greek.hyper - excessive rise and kinesis - movement). And with hypo- and hyperkinesis, a muscle disorder occurs tone, markedly different from that observed with the defeat of the pyramidal system or peripheral motor neuron.
In the 20s of the current century, the concept was put forward that hypokinesis arise due to lesions of a phylogenetically older formation - the globus pallidus, and hyperkinesis arise due to damage to the caudate nucleus and shell. It was believed that the caudate nucleus and shell (new
striatum) inhibit the old striatum (pallidum). However, recently such a mechanism the occurrence of hypo- and hyperkinesis was rejected. It was found that extrapyramidal disorders can occur with damage to both the cerebral cortex and its trunk.
In the 40-60s, new data were obtained on the function of that part of the extrapyramidal system, which
called network formation. Animal experiments have shown that the irritation of this formation of an electric current leads to the activation of the activity of the cerebral cortex. On the electroencephalogram shows that the slow electrical activity of the cortex turns into high-frequency, low-amplitude (desynchronization resynchronization). In addition, in the network-like
formation there are areas, the irritation of which activates the activity of the spinal cord, leads to increased motor spinal reflexes. It relieves the spinal cord transmitted through the reticular-spinal tract. Networked education includes zones, the irritation of which causes inhibition of the cerebral cortex and spinal cord. It was it was found that impulses following the reticular-spinal tract reach not only *a-,* but also γ-motoneurons.
Thus, the final segment of the path along which the pulses from the extrapyramidal systems enter the skeletal muscles. The afferent part of the arc of this tonic reflex represented by fibers of deep muscle sensitivity. At the level of the brain stem from these conductors collateral fibers depart to a network-like formation. Arising in it efferent impulses can be conducted down the reticulospinal tract. Upstream afferent impulses from muscle spindle receptors form 2 streams. One of them passes through the classical path of deep sensitivity, the other - along the conductors of the network-like formation. V
as a result, the activity of the cerebral cortex is activated, in particular - the frontal lobe, which sends impulses not only to various ganglia and nuclei of the extrapyramidal system, but also continuously receives response signals from them (reverse afferentation). Ring-shaped systems, the impulses along which go not only from the frontal lobe to the ganglia and further to the periphery, but
and return through the thalamus back.
Network formation (fonnatio reticularis) is not only part of the extrapyramidal system, but also part of the vegetative-visceral system (limbic-hypothalamic-reticular complex) in frontal lobe. The principle of a neural ring that closes with a feedback channel are currently recognized as the main one in the organization of the activity of the central nervous system.
The function of the basal nuclei has become much clearer in connection with the discovery of the role of cerebral
transmitters. It has been proven that in the implementation of the regulation of motor function of great importance
have dopaminergic systems of the brain. The striatum contains more than 80% of the total

dopamine, and norepinephrine formed from dopamine, which also has a large biological meaning, is found predominantly in the brain stem. On this basis, it was first expressed the assumption that dopamine in the brain has its own functional significance associated with extrapyramidal formations, and different from its role as a precursor of norepinephrine, as

thought before. The introduction of reserpine to an experimental animal leads to a sharp decrease in the content of dopamine in the striatum and the development of akinetic-rigid (parkinson-like) syndrome. Subsequent administration of the dopamine precursor, the levorotatory isomer 3,4-dihydroxyphenylalanine (1-dopa) eliminates this syndrome. It turned out that in patients with the syndrome
parkinsonism, the content of dopamine in the striatum is sharply reduced, and 1-
dopa.
An important achievement in recent years is the discovery and histological identification in the brain main dopaminergic neuronal systems. Already well studied 2 such ascending
systems. The main one is the axons of melanin-containing neurons of the compact zone of the black substances that pass into the tegmental area and the lateral part of the hypothalamus, then enter into the brain stem, then go through the retrolenticular section of the inner capsule, pallidum and end in the striatum with characteristic bead-like nerve terminals. These terminals
contain a large amount of dopamine, as well as its metabolites and synthesizing enzymes. Degeneration of this nigrostriatal dopaminergic tract with a sharp decrease in synthesis and the release of dopamine from its terminals in the striatum are major
histopathological and biochemical signs of parkinsonism. This is the same underlying
the clinical syndrome characteristic of such a disease.
The second ascending dopaminergic system is the mesolimbic tract. It starts
from the cells of the interpeduncular nucleus of the midbrain, located medially from the substantia nigra,
passes laterally from the nigrostriatal tract and ends in phylogenetically older nuclear
formations - nucl. accumbena, nucl. interstitialis striae tennmalis and tuberculum olfactorium, which are combined under the name of the limbic striatum, given their connection with the limbic system (hippocampus, amygdala, olfactory cortex). It is believed that this phylogenetically ancient part of the brain is associated with complex behavioral acts that ensure the safety of the species. From
cells of the limbic striatum axons go to the hypothalamus and the cortex of the frontal lobe - structures,
which are involved in the regulation of emotional reactions and intellectual functions; in particular, the mesolimbic dopaminergic pathway is suggested to be involved in mood control and behavioral reactions. In addition, this system of neurons controls the onset of the motor act.
and motor affective responses (eg, accompanying emotions). The connecting link
between the limbic and extrapyramidal motor systems is nucl. assumbens. This is the core located in the ventromedial part of the anterior striatum and has some common
cytoarchitectonic and biochemical characteristics. as well as general efferent projections in substantia nigra and a pallid ball. In patients with parkinsonism, along with a sharp decrease in the content of dopamine in the striatum, its content in nucl is significantly reduced. accumbens and other limbic formations. Obviously, akinesia and
emotional disorders characteristic of parkinsonism.
In the striatum, in addition to dopamine, 7 more transmitters are secreted: acetylcholine, γ-aminobutyric
acid (GABA), norepinephrine, serotonin, glutamic acid and neuropeptides - substance P and methenkephalin. Participation in the extrapyramidal regulation of angiotensin movements is assumed,
which is also found in striopallidonigral systems. However, the functional meaning
neuropeptides in the extrapyramidal system have not been studied enough. In the development of extrapyramidal
movement pathology is of significant importance, apparently, a violation of the interaction various transmitters with the dopaminergic systems of the brain.
Electron microscopy revealed 9 different types of synapses within the striatum
neurons and their transmitters are determined. More than 90% of the cells of the striatum are subulate interneurons. It is with the spikes of the dendrites of the interneurons of the striatum that
synaptic connection of such tracts as nigrostriatal dopaminergic, corticostriatal
glutamatergic and thalamostriatal cholinergic (transmitter acetylcholine). The main

the transmitter between interneurons is acetylcholine, and in the synapses formed collaterals of axons of projection cells branching inside the striatum - GABA. Thus, convergence occurs at the level of the interneuronal pool of the striatum.

most afferent transmitter systems. An important factor is the establishment of the reverse noisy connection between the striatum and the substantia nigra. Dopaminergic tract melanin-containing cells of the substantia nigra ends on interneurons of the striatum, which are connected through internal synaptic contacts with projection cells. Or these latter axons go back to the substantia nigra. The transmitter of this strionigral the path is GABA.

Large cells in the anterior part of the caudate nucleus produce substance P, which transported and transmits impulses to the neurons of the anterior third of the substantia nigra. At the level of interneurons of the striatum, this closed system has contacts with glutamatergic by the corticostriate pathway and cholinergic receptors from the medial central and parafascicular nuclei of the thalamus. The results of complex processing taking place on interneurons all incoming information is transmitted through synaptic contacts with projection cells on the main efferent systems that control motor functions. In these mechanisms, dopa min has an overwhelming effect, and glutamate has an exciting effect on interneurons of the striatum, in

while the action of acetylcholine depends on its concentration and adapts the incoming impulses to the motor situation necessary at the moment. This system is complicated by synapses (about 20%), formed by the three main afferent tracts of the striatum on large projection cells, in which the same transmitters have the opposite effect: dopamine - exciting, and glutamate - overwhelming.

Communication between nucl. accumbens and substantia nigra, as well as the globus pallidus a GABA transmitter. The inhibitory effect of this tract in its electrophysiological characteristics differs from the influence of striopallidonigral projections.

These data reveal the integrative mechanisms of the striatum that determine its main functional meaning, which consists in focusing attention and emotions on one the most important motor act at the moment. This is achieved by simultaneous inhibition of all other processes that may interfere with its implementation. In carrying out this the role of great importance is attached to the inhibitory GABAergic striopallidonigral pathway, which regulates the activity of efferent systems of various functional significance substantia nigra and globus pallidus and is responsible for focusing attention on the contralateral side. It is assumed that in this case, the caudate nucleus is involved in the control of the mechanisms mental activity and in the implementation of psychomotor actions. Transfer of motivation to action is carried out along the GABA-ergic tract from nucl. accumbens to substantia nigra. In the same time the shell regulates the main motor mechanisms through the main efferent extrapyramidal systems, the so-called function generators - a nigral system that con- trolls the speed of movements, and the pallidary system is the main, most significant locomotor center regulating motor acts and attention.

It should be noted that, unlike the substantia nigra, there is no direct feedback between globus pallidus and striatum. This communication is multi-stage and is carried out through ventrolateral nucleus of the thalamus and neurons of the premotor areas of the cortex.

To understand the need for voluntary movement, its planning and implementation in cortical neurons receive 2 streams of impulses. One stream passes through specific cortical tracts different types of sensitivity, and the other - through the basal ganglia and makes a loop in them. Passing-

the impulses that follow it carry out the preparation of the muscles for movement and its awareness. Fine

the basal ganglia provide an automatic sequence of simple motor programs necessary to carry out the action plan.

Less researched, compared with dopamine, the role in the regulation of movements of other

neurotransmitter - norepinephrine. The main noradrenergic center in the brain is golu-
a rather odd place (locus coeruleus) in the pons of the brain. Its neurons form norepinephrine nerves terminals in all areas of the brain, including the cerebral cortex and cerebellum, in the hippocampus. Thus, one nucleus, through its connections, can influence many structures of the brain.
No other neuronal system of the brain has an innervation of this nature. Paths found
from bluish to substantia nigra, which can be used to control activity
dopaminergic nigrostriatal tract. In patients with parkinsonism, along with pathology
melanin-containing neurons of the substantia nigra, often degenerative changes in the locus coeruleus with a decrease in the content of norepinephrine in their system.
In recent years, data have been obtained on the modulating action of the cerebellum on dopaminergic and
noradrenergic systems of the brainstem and basal ganglia. Known anatomical effects
enzyme connections of the cerebellum with the catecholaminergic nuclei of the tegmental, substantia nigra and

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striatum, as well as with the ventrolateral nucleus of the thalamus. Experimentally revealed connections between
do striatum and cerebellum. Damage to the cerebellum or its connections with catecholaminergic nuclei can affect existing extrapyramidal disorders, as well as participate in the development various hyperkinesis.
It should be noted that in the mechanisms of action of neural systems, an important role is played by functional state of catecholamine receptors, which are located on membranes
postsynaptic and presynaptic formations. Postsynaptic membrane receptors
interact with a neurotransmitter released into the synaptic cleft under the influence of nerve impulses, and transmit excitation to an effector organ or postsynaptic neuron. In contrast to this
presynaptic receptors are located on the outer surface of the membranes of catecholamine terminals. Their main physiological significance lies in the modulated release
mediator into the synaptic cleft.
In the noradrenergic terminals of the peripheral nervous system, 3 types were identified
presynaptic receptors: inhibitory β-receptors (they inhibit the release of norepinephrine),
facilitating ^ -receptors, and inhibitory dopamine receptors. When these receptors are stimulated the release of norepinephrine decreases and noradrenergic transmission is inhibited. Such a me-
khanism underlies the development of muscle hypotension, in particular in the treatment of l-dopa patients with
extrapyramidal movement disorders.
Presynaptic receptors are also found in the central nervous system. They are revealed
and at the terminals of dopaminergic neurons (inhibitory dopamine autoreceptors). In striped
In the body, postsynaptic dopamine receptors are localized on cholinergic interneurons.
The activity of the nigrostriatal dopaminergic pathway is controlled through GABA receptors, located on the dopaminergic neurons of the substantia nigra. In addition, on dopaminergic terminals in the striatum, cholinergic and opiate receptors are found that have
value in the modulated release of the mediator. Postsynaptic hypersensitivity
dopamine receptors in the striatum is an important pathogenetic factor
hyperkinesis.
In recent years, the existence of two types of dopamine receptors has been discovered - D1 and D2, which have different pharmacological properties. Acting on D1 receptors alters the overall locomotor activity, while stimulation of D2 receptors causes dyskinesias.
So, functionally closely related to the pyramidal formations and the system coordination of the extrapyramidal system is involved in the formation of muscle tone and
poses. She kind of prepares the skeletal muscles at every given moment. perceive exciting and inhibitory impulses. Violation in one of the links,

regulating the activity of the extrapyramidal system, can lead to the appearance a special form of increasing muscle tone - rigidity, as well as the development of hypo- or
hyperkinesis.
At research motor functions always have to differentiation to study the component of the activity of the cortical-muscular pathway and extrapyramidal system. The function of the extrapyramidal system is not assessed by the strength of muscle contraction, and by the quality side of the movements. Draw attention to the patient's posture, expressiveness of speech, facial expressions and arbitrary movements, including walking. To assess muscle tone, some tests.
Head tilt test: the patient is in a supine position,
the examiner puts his hand under the occipital region of the patient's head and tilts it, and then quickly transfers the brush lower to the neck. In a healthy person
there is a passive and rapid extension in the cervical spine. At extrapyramidal increase in muscle tone the patient's head for a certain time held in position, then smoothly and slowly returns to
starting position. This test allows you to detect early plastic disorders rigidity. With pronounced muscle rigidity, movements in the cervical spine the spine are limited in the same way as in the meningeal syndrome of rigidity of occlusion
muscles, and therefore bringing the chin to the chest fails. Unlike meningitis with extrapyramidal rigidity no pain in the occipital
muscles during this test.
Upper extremity fall test : in a standing patient, the examiner

raises his relaxed upper limbs to the sides slightly higher
horizontal level. Then, unexpectedly quickly, he lets go of them and his brushes. moves on the subject's torso to determine the time of the fall and the force of the impact
upper extremities of the patient. With a difference in the tone of the muscles of the shoulder girdle on the right, and

on the left, the difference in the speed of the fall and the force of the impact will be revealed.

Upper limb pendulum swing test : in a healthy person with walking, pendulum-like swinging of both upper limbs has the same amplitude that is synchronous with the stepping movement of the contralateral inferior limbs. With extrapyramidal rigidity, there is a slowdown and lag in movement of the upper limb. This is especially noticeable in unilateral lesions.

Pendulum swing test of the lower extremities: the patient sits on a high chair (table) with freely hanging lower limbs. The examiner unbends them in the knee joints and releases them freely. In a healthy person with a relaxed the muscles of the lower limbs of the lower leg symmetrically perform several pendulum movements. On the tonic side, the time is shortened swing and its amplitude decreases.

Noyck-Ganev test: when determining the muscle tone of the upper limb when passing active movements in the elbow or wrist joint, the patient is asked to actively support lower limb. Raising the lower limb reveals an increase plastic hypertension of the muscles of the upper limb. In the case of one-sided lesions with active movement of the lower limb on the side of increasing tone muscle tone in a healthy upper limb does not increase.

Foreman test: in the Romberg position with closed eyes with extrapyramidal stiffness, the tone of the muscles of the upper limbs increases, and in the supine position - goes down.

Posture fixation tests : with extrapyramidal rigidity, the patient retains indefinitely long any pose that is given to him. This is verified with the following tests:

a) extension test in the wrist joint : the doctor performs full extension in the wrist joint and stops it, releasing the hand; in a patient with parkinsonism the hand is in a given position, then slowly bends in the wrist joint;

b) Westphal's foot test: sharp extension in the ankle joint (sliding movement of the doctor's hand along the sole) is accompanied by freezing of the foot in the this position for a while due to tonic tension of the muscles bateurs of the foot (mainly of the tibial muscle), then the foot slowly descends;

c) knee flexion test: the patient is in the supine position on abdomen, the doctor flexes the patient's relaxed lower limbs in the knee joints at right angles and leaves them in that position. In this case, a reflector occurs -

contraction of the flexor muscles of the lower leg, the lower limb bends even more in
knee joint and is held in this position for a long time.
Handwriting assessment: handwriting becomes shallow in patients with parkinsonism
(micrograph).
The main syndromes of extrapyramidal lesions are
amiostatic syndrome (parkinsonism) and various hyperkinesis.
Parkinsonism. This syndrome is characterized by monotonous quiet speech.
(bradilalia) and low physical activity of the patient (oligokinesia). Face
hypomimic, the gaze is motionless, as if directed at one point, the blinking is rare,

sometimes it is absent for several minutes, gestures are poor. Torso
slightly tilted forward (supplicant pose), upper limbs bent at the elbows
joints, pressed to the body. There is a tendency to freeze in any,
even an uncomfortable pose. The patient can lie in bed with the pillow raised above the pillow.
head - a symptom of "air cushion". Active movements are very
slowly (bradykinesia). Difficulty starting the motor act - parkinsonian
"Marking time". The patient walks with small steps, when walking, there is no usual
physiological synkinesia - friendly movement of the upper limbs, they are
walking motionless (acheirokinesis). Sometimes there is another peculiar
symptom - propulsion - the patient begins to move faster and faster on the move, not
may stop and even fall. This is explained by the fact that the displacement of the center of gravity
does not cause the patient to reactively contract the muscles of the back - “he seems to be running for
its center of gravity ”. If the patient is given an initial movement (slightly push into the chest), he begins to move backward (retropulsion). The same forced movement
to the side is called lateropulsion. Sometimes "paradoxical kinesias" are observed
(patients who are almost immobilized during the day, at the time
affective outbursts and emotional tensions can jump, run up
stairs, dance, etc.)
Another component of the described syndrome is muscle stiffness.
- a kind of resistance to passive movements. Muscle stiffness is different from pyramidal spasticity in that it not only appears in the initial phase of movement,

but it is also retained in all subsequent phases of muscle stretching. The limb is like
freezes in the position that is given to her. This state is also denoted as
“Plastic tone”, “wax flexibility” (flexibilitas cerea), etc.
Extrapyramidal rigidity has other features: it is observed during
many muscle groups (agonists and antagonists), but usually predominates in
flexor muscles (“flexor pose”). Typical for pyramidal spasticity
there are no "folding knife" phenomena. The study of passive movements can
detect discontinuity, as it were, the graded resistance of muscles to passive
sprains ("cogwheel" symptom).
Hypokinesia and rigidity can be observed in isolation, but they are often
hyperkinesis is added in the form of a tremor of the fingers of the hand, less
often - of the lower extremities and
chin area. This rhythmic tremor of the fingers of the hands resembles the action
when counting coins or rolling pills. The emerging triad of symptoms
(hypokinesis,
rigidity, rhythmic tremor) is characteristic of a chronic disease described
discovered in 1817 by the English physician J. Ragkinson and named
“trembling
paralysis". A syndrome that is very close clinically to tremor paralysis has been
identified
in patients in the chronic stage of epidemic encephalitis, with vascular
brain lesions, with some exogenous intoxication
(manganese compounds, chlorpromazine, reserpine, etc.). It was named
parkinsonism. The most pronounced hypertonicity and tremor occur with
damage
black matter.
Other symptoms in parkinsonism are characterized by autonomic disorders.
(greasiness of the face, peeling of the skin, hypersalivation, etc.) and disorders
psycho-emotional tone. The latter is manifested in the form of a decrease in
motor
activity, spontaneity in actions. Sometimes it is possible to note the described by
M.I.
Astvatsaturov (1939) the propensity of patients to "molest" (akayriy) - obsessive
the desire to ask the same questions, reapply for insignificant
reasons. Thinking in patients is slowed down (bradypsychia).
Parkinsonism is characterized by the disappearance of all its manifestations in
the period of falling asleep of the patient (the degree of severity of muscle tone
decreases,
the tremor stops).
With long-term treatment of parkinsonism 1-dopa in the form of side symptoms

some psychopathological and neuroendocrine symptoms develop that
is associated with dysfunction of dopamine-containing systems, such as
mesocortical (from the core of the midbrain lining to the cortex of the frontal lobe),
tuberoinfundibular (from neurons agcuatus tuberculum infundibulum of the hypothalamus to
anterior lobe of the pituitary gland).

Extrapyramidal hyperkinesis.

Hyperkinesis are automatic violent excessive movements,
interfering with the performance of arbitrary motor acts. When researching hyperkinesis pay attention to the side, rhythm, character, shape, symmetry, localization of motor manifestations. There are the following main types hyperkinesis: tremor (tremor), myoclonus, choreic hyperkinesis, athetosis, torsion dystonia, hemiballism, etc. Hyperkinesis occurs when
different parts of the extrapyramidal system (mainly the striatal
system). Trembling
- the most common type of hyperkinesis, varied in amplitude, pace and localization.

With neuroses, exogenous and endogenous intoxications, tremors are usually observed
in the fingers of the hands, has a small amplitude, changing rhythm. Tremor with
parkinsonism is rhythmic (4-6 in 1 s), it is also localized in the fingers
hands, but can spread to the lower limbs, head, chin,
torso. The trembling is more pronounced at rest, it decreases or even disappears with
active movements. This distinguishes it from intentional tremor in defeat.
cerebellar systems. Large-sweeping tremor occurs when the red
nuclei ("rubral tremor"). Extrapyramidal tremors are persistent and
disappears only in sleep.

Myoclonus - rapid, usually irregular contractions of individual muscles or their groups. They are visible when examining the limbs, trunk, face. Small amplitude
does not lead to a pronounced locomotor effect. Myoclonus can be
generalized and local (for example, tongue and soft palate - bicycle palatine nystagmus), myoclonus persists at rest and during movement, aggravated with excitement.

They are found in the defeat of red nuclei, black matter, striatum, and serrated kernels and lower olives. The same contractions of part of the muscle (not all)
referred to as myokymia.

Choreic hyperkinesis is characterized by erratic involuntary

movements with a pronounced locomotor effect, occurs in various parts of the body
both at rest and during voluntary motor acts. Movement all the time
replace each other in the most unusual sequence, recalling expedient,
albeit exaggerated, actions. The patient either closes his eyes, then sticks out his tongue,
licks lips, then makes grimaces, etc. Sudden impulsive
changes in the position of the limbs, changes in posture. They are compared to clowning,
dance (Greek choreia - dance). Keep the upper limbs extended forward at rest
or the patient does not succeed with the protruding tongue. These signs are used to identify
weakly expressed forms of choreic hyperkinesis.
Choreic hyperkinesis occurs when the neostriatum system is damaged (with rheumatism, hereditary chorea of Huntington, etc.). With choreic hyperkinesis a decrease in muscle tone is often noted.
Athetosis (Greek athetos - unstable) is a type of hyperkinesis, which is characterized by
slow tonic muscle contractions, which outwardly resembles a slow rhythm
bizarre "worm-like" movements. They arise at rest and during
voluntary movements, amplified by the influence of emotions. These are pretty strong
periodically occurring muscle spasms are most often localized in the distal parts of the upper extremities. They are called spasmusmobilis. You-
straight fingers bend slowly alternately, sometimes especially strongly
unbend in the metacarpophalangeal joints. The brush at this time takes on a bizarre

shape. Athetosis can be bilateral. Athetosis differs from chorea in slowness movement and usually less prevalence. Sometimes in distinguishing between these
hyperkinesis, difficulty arises, then they talk about choreoathetosis. Athetosis develops
in case of damage to large cells of the striatal system with discoordination of function
globus pallidus, subthalamic and red nuclei.
Hyperkinesis, called **torsion dystonia** , should be distinguished from athetosis . Have
these patients, especially with active movements, the wrong
distribution of muscle tone of the trunk and limbs. This leads to
the formation of pathological body postures. Outwardly, this is expressed by the fact that when walking

the trunk and limbs perform corkscrew-like violent movements - equivalents of their rotation around a long axis, which is reflected in the name diseases. Torsion dystonia can stop with various compensatory techniques, for example, when fixing the cervical spine with hands, reinforced turn of the shoulder, etc.

Torsion-dystonic phenomena can be limited to any part muscular system, such as the muscles of the neck (spastic torticollis - torticolis spactica).

The pathogenetic basis of torsion dystonia is muscle spasm antagonists, i.e. involuntary muscle tension that opposes the desired movement. Torsion-dystonic phenomena can occur when different areas of the extrapyramidal system (basal ganglia, brain stem cells).

Convulsive contraction in the fingers of the hand during writing is referred to as writing spasm (graphospasm). Musicians have similar professional spasms. (violinists, pianists, guitarists), typists, etc.

A special form of extrapyramidal hyperkinesis is **"hemiballism"** (from Greek hemi - half, ballismos - bouncing, dancing). This rare variant hyperkinesis, as the name suggests, occurs on one side of the body, suffers more upper limb. Sometimes both sides are involved, then they talk about paraballism.

Hyperkinesis manifests itself with fast, sweeping movements in large volumes, resembling throwing or pushing a ball; elements are possible at the same time rotatory movement of the trunk. This clinical picture has been described with focal defeat of the hypothalamic nucleus (Lewis subthalamic body).

Rapid involuntary muscle contractions (usually the circular muscle of the eye or muscles that twitch the corners of the mouth) is called a **tic.** Unlike functional (neurotic) tics of extrapyramidal genesis differ constancy and stereotype.

Along with local forms, there is a generalized tic with involvement muscles of mimicry, respiratory, limbs and trunk. A special place is occupied by generalized impulsive **tic - Gilles de la Tourette's syndrome** , in which impulsive jumping, squatting, grimacing, vocal phenomena in the form of screaming swear words (coprolalia), screaming, grunting, etc.

With damage to the extrapyramidal system, local hyperkinesis and muscle spasms of the eyeballs and facial muscles **. These include**

tonic spasm of the gaze. The eyeballs are involuntarily retracted upward. Such an attack occurs in a patient suddenly and lasts for several minutes or more. Sometimes there may be an involuntary tonic contraction of the circular muscles both eyes (blepharospasm). In other cases, the spasm covers the facial muscles,

innervated by the facial nerve on one or both sides (facial hemi- or paraspasm}: cramps are accompanied by wrinkling of the forehead skin, raising of the eyebrows,
closing the eyes, pulling the corner of the mouth outward and upward, tension of the subcutaneous
muscles of the neck. With functional facial hemispasm, there will be no paradoxical

synkinesis of the upper mimic muscles, that is, when closing the eye, the eyebrow is not
will rise up, the forehead does not wrinkle.
Sometimes persistent extrapyramidal hyperkinesis is interrupted by general convulsive seizures - this is the so-called **hyperkinesis-epilepsy** . So with Kozhevnikovskaya epilepsy and myoclonus epilepsy
myoclonic hyperkinesis. Combination of choreic hyperkinesis with general convulsive seizures are observed with choreic epilepsy of ankylosing spondylitis.
Detection of the hyperkinesis described above makes it possible to diagnose damage to the extrapyramidal system. However, clinical, anatomical and experimental studies show that with the same localization
of the focus, hyperkinesis of various types can occur, therefore a more accurate definition
the lesion site is difficult. This is also evidenced by the results of surgical treatment of extrapyramidal disorders. In stereotaxic operations, they destroy intact neuronal systems (usually the ventrolateral nucleus of the thalamus). There is a rupture of ring bonds between individual extrapyramidal formations along which impulses flow, causing various motor
disorders (torsion dystonia, parkinsonism). As a result of such operations extrapyramidal disorders undergo significant reverse development.
However, 2-3 years after surgery, extrapyramidal disorders have
recovery trend.
One of the options for tremor is **asterixis (** from the Greek sterigma - not the ability to maintain a fixed pose). With the upper ones extended forward the patient's extremities produce hyperpronation of the hands; in 20-30 s appear propulsive movements of the fingers in the metacarpophalangeal joints in the anteroposterior
direction with a rotator component and with a slower flexion phase than extension. In the origin of asterixis, the role of a disorder of the sense of posture and
violation of afferent information in case of dysfunction of the structures of the upper sections of the trunk

brain and diencephalon involved in the integration of movements and regulation of the we
intestinal tone. Asterixis occurs mainly in dysmetabolic lesions.
nervous system (renal failure, overdose of antiparkinsonian
drugs, exogenous intoxication), less often - with stenosis of the internal carotid artery or
with diapedesic hemorrhage in the brain stem or cerebellum.

Pallidary system

Striatal system

Develops HYPERTONIC-
HYPOKINETIC SYNDROME
(PARKINSONISM).
Develops HYPERKINETIC-
HYPOTINIC SYNDROME.
It develops with the defeat of the globus pallidus or
black matter
The defeat of the caudate nucleus and shell
dopaminergic neuronal
systems
Etiology: hereditary
predisposition, epidemic
encephalitis, atherosclerosis of subcortical nodes,
less often infections, intoxication (manganese,
lead, mercury, antipsychotics, carbon monoxide),
tumors.
Etiology: encephalitis (more often
rheumatic), violation
circulatory, hereditary
diseases, tumors. Symptoms -
a combination of hyperkinesis (i.e.
involuntary movements of the limbs,
trunk) with muscle hypotonia.

Main symptoms: decreased motor
initiative, oligokinesia (small
the patient's motor activity), amimia,
general stiffness, monotonous speech,
quiet *(bradilalia*), slowness
active movements of the patient
(*bradykinesia).* Torso several
tilted forward (*supplicant pose, bent
pose*), the upper limbs are bent at the elbow
joints, pressed to the body. Difficult
the beginning of the motor act
- *parkinsonian "stomping on
place ".* Walks in small steps when walking
there is no normal physiological
synkinesia friendly movement
upper limbs, they are when walking
motionless (*acheirokinesis*). Sometimes
there is a *propulsion* - the patient is on the move

starts to move faster and faster, not
may stop and even fall
(moving the center of gravity does not cause
sick reactive muscle contraction
back). If the patient is given the initial
movement (lightly push into the chest), he
starts to move back
(*retropulsion),* forced movement in
side - *lateropulsion* . Sometimes
" *paradoxical kinesias* " are observed
(patients who are in
almost immobilized, at the moment
affective outbursts and emotional
stresses can jump, dance,
especially at night, etc.). characterized by *muscle*
rigidity , i.e. increased muscle
plastic tone (*symptom*
"Cogwheel "). Small handwriting,
pointed, indistinct (*micrograph*).
Often against the background of general immobility
joins hyperkinesis in the form of *tremor*
fingers of the hand (the phenomenon of "rolling pills "),
less often the feet of the head, lower
jaw. Characteristic of parkinsonism
is the disappearance of all its manifestations in
the period of falling asleep of the patient (decreases
the severity of muscle tone,
the tremor stops). With parkinsonism
vegetative disorders are observed
(greasy face, hypersalivation, peeling
skin).
The emergence of redundant,
uncontrolled movements in
different muscle groups.
There are the following types
hyperkinesis:
-*choreic hyperkinesis* - fast
irregular movements in the distal and
proximal extremities,
face, torso (disorderly
flexion and extension of the arms,
frowning of the forehead, sticking out the tongue);
-*athetosis* - slow artsy
tonic nature of movement,
predominantly in the distal regions
limbs, sometimes in the muscles
face (protruding lips, twisting
mouth, etc.);
-*torsion dystonia* - slow
tonic, mainly
rotational movements of the trunk;
- *tremor* - rhythmic hyperkinesis in
distal extremities, less often in
lower jaw and tongue, amplitude and
the rhythm varies widely;

- *myoclonus* - fast, more often rhythmic
contractions in individual muscles or
muscle fiber groups; in contrast
they are not accompanied by chorea
pronounced motor effect.
May be local
(proximal arms, tongue 6
soft palate, etc.) and
generalized.
A characteristic feature of all hyperkinesis
is their disappearance in sleep.

CEREELLUM AND MOVEMENT COORDINATION DISORDERS

Motor skills
human
characterized by
astonishing
precision
purposeful movements, which is ensured by the proportionate work of many
muscle groups controlled not only arbitrarily, but also in many ways
automatically. Implements this complex multifunctional system
a multi-neuronal coordinating apparatus that controls balance
body, stabilizes the center of gravity, regulates tone and
varied muscle activity. To perform coordination of movements
a clear and continuous reverse afferentation is required, informing about
the relative position of the joints, about the condition of the muscles, about the load on them, control of
trajectory of movement.

The center of coordination of movements is the cerebellum ; in functional
in relation to it, the cerebellar body is distinguished, consisting of two hemispheres, the worm and
three pairs of legs.

The collector of afferent impulses entering the cerebellum according to different
ny ways, is the core of the tent (nucl. fastigii). Having received scattered information
from various sources, this kernel sends it for processing to pear-shaped
neurons (Purkinje cells) of the cerebellar cortex in accordance with the somatic
projection (in the anterior sections of the cerebellar hemispheres, the upper
limbs, in the hind parts - the lower; in the anterior sections of the cerebellar cortex
the head and neck are represented, and in the rear sections - the trunk). Proximal divisions
limbs are projected medially, distal - lateral; hemispheres
responsible for the zones (precentral and frontal gyrus). Therefore, muscular
the cerebellar-cortical pathway can be attributed together with the conductors of the articular
muscle sense to the motor (kinesthetic) analyzer.

The main function of the cerebellum is carried out at the subconscious level.
Efferent impulses from the cerebellar nuclei regulate proprioceptive reflexes

tensile. With muscle contraction, excitement occurs
proprioceptor (muscle spindle) of both synergistic muscles and muscles
antagonists. Normally, however, the transformation of voluntary movement into a complex
the reflex does not occur due to the inhibitory effect of cerebellar impulses.
That's why
at
defeat
cerebellum
disinhibition
segmental
proprioceptive reflexes are manifested by limb movements of the type
ataxia. The cerebellum has many afferent and efferent connections.
Posterior spinal cord tract (Fleksig's tract). The first neuron is laid in
spinal ganglion, its dendrites are associated with muscle propriceptors,
tendons, ligaments and periosteum; axon as part of the posterior root through the posterior horn
fits to the cells of the Clarke column (at the base of the posterior horn). The fibers of these
second neurons are sent to the outer layers of the posterior part of the lateral cord
side, rise along the entire spinal cord and at the level
the medulla oblongata as part of the lower cerebellar pedicle are included in the worm
cerebellum. This path is designated as tractus spinocerebellaris dorsalis (posterior) or
Fleksig's path. In the cortex of the cerebellar worm there is a third neuron, which
contacts with pear-shaped neurons of the cerebellar cortex. Axons
the latter go to the dentate nucleus (nucl. dentatus). Fibers of this fifth neuron
are part of the superior cerebellar peduncle. Right and left upper cerebellar peduncles
intersect (Werneking's cross) and end at the cells of the red nucleus
the opposite side. Axons of cells of the red nucleus (nucl.ruber) immediately

go to the opposite side of the midbrain and form
ventral intersection in the tectum of the midbrain (Trout's intersection), pass in
the composition of the lateral cord of the spinal cord (in front of the pyramidal tract),
reach the cells of the anterior horns (α- and γ-motoneurons). Collection of axons
cells of the red nucleus is called tractus rubrospinalis (Monakov's bundle). In man
it is poorly developed. The main descending influences of the cerebellum are transmitted by
reticular-spinal bundle.
Anterior spinal-cerebellar path of Govers (tractus spinocerebellaris anterior).
The first neuron is located in the spinal ganglion, the second neuron is a cell
the posterior horn, but its axons go to the opposite side and
go up the spinal cord, in front of the lateral cord,
pass through the medulla oblongata, the bridge of the brain, at the level of the superior cerebral
the sails pass to the opposite side and as part of the upper leg of the cerebellum

reach the cells of the cerebellar nuclei. The further course of efferent impulses is the same as in the posterior spinal cord.
The cerebellum receives afferent proprioceptive impulses not only by paths of Fleksig and Govers, they also enter the axons of the cells of the nuclei of the thin and wedge-shaped beams, some of which do not go directly to the optic tubercle, but through the lower legs of the cerebellum follow to its worm.
In addition, axons of cells weighing tibular nuclei - mainly from the vestibular lateral nucleus (Deiters nucleus), they end in the nucleus of the clivus of the cerebellum. Fibers of cells of this nucleus in the composition the upper and, possibly - the lower legs of the cerebellum are suitable for the cells of the reticular the formation of the brainstem and to the vestibular lateral nucleus, from which conductors form the descending tracts - the vestibular-spinal cord and reticular-spinal cord, ending in the cells of the anterior horns of the dorsal brain. Along this vestibular-cerebellar-muscular pathway, regulation of body balance.
From the cerebellum through the vestibular lateral nucleus, connections are established and with nuclei of the oculomotor nerves (as part of the medial longitudinal bundle).
The function of the cerebellum is obviously corrected by various parts of the cortex. brain. This is indicated by the numerous connections of almost all lobes of the brain with cerebellum. The most massive of them are 2 bundles - frontal-bridge cerebellar and occipitotemporal-cerebellar.
Fronto-cerebellar (tractus fronto-ponto-cerebellaris) - a set axons of cells predominantly of the anterior sections of the upper and middle frontal convolutions. In the depth of the lobe, they are collected in a compact bundle and form an anterior the leg of the inner capsule. Then they pass at the base of the brain stem and on their the same side end with a synapse at the cells of the brain pons. The axons of these second neurons move to the opposite side of the bridge and as part of the middle leg the cerebellum enters its hemisphere and contacts the cells of the cerebellar cortex. The processes of these neurons of the cerebellar cortex approach the dentate nucleus. Cell fibers the dentate nucleus as part of the superior cerebellar peduncle reach the red nucleus the opposite side and along the reticular-spinal tract impulses regulating the posture of a person in an upright position, in particular standing and walking.
Occipito-temporo-cerebellar pathway (tractus occipito-temporo-ponto-cerebellaris) - its first neurons are located in the cortex of the occipital and temporal lobes

(partly and parietal); their axons are collected in the subcortical white matter, then as part of the posterior part of the thigh, the inner capsule runs at the base of the midbrain
to the nuclei of the pons of the brain on their side. Axons of the bridge cells go to the opposite side and along the middle pedicle reach the cerebellar cortex. Fiber these cells approach the dentate nucleus, which has connections with the brain stem. WITH
using these tracts, coordination of the work of the cerebellum with the organs is ensured
vision and hearing.
The existing intersections of the cerebellar afferent and efferent sy-
stem ultimately lead to a homolateral connection of one hemisphere
cerebellum and limbs. With damage to the cerebellar hemisphere, disorders of it functions arise on the half of the body of the same name. Foci in the lateral cord spinal cord also cause cerebellar disorders on their side of the body.
The hemispheres of the brain are connected to the opposite hemispheres
cerebellum. Therefore, with damage to the cerebral hemispheres or the red nucleus cerebellar disorders will be detected on the opposite side of the body.
Many symptoms of cerebellar dysfunction are associated with impaired
reciprocal innervation of antagonist muscles. The essence of this phenomenon is as follows.
When performing any movement, motor neurons of muscle agonists and antagonists (e.g. flexors and extensors) are in the opposite state
excitement. If, for example, the neurons of the flexor muscles are excited, then the neurons of the extensor muscles are inhibited. The mechanism of such a conjugate (reciprocal) inhibition of the spinal motor centers consists in
next: the axons of receptor cells (their bodies are located in the spinal
ganglia) in the spinal cord are divided into branches, some of them excite motoneurons
flexor muscles, while others come into contact with intercalary cells that
have an inhibitory effect on the cells of the extensor muscles. So this
the mechanism of reciprocal innervation is carried out by the segmental apparatus spinal cord. However, its complex integrative function also involves
cerebellar impulses.
In case of violation of the coordination of the action of muscle agonist groups (non-mediocre movement), antagonists (in some phase of
active agonists), synergists (helping the work of either agonists or
antagonists), movements lose coherence, accuracy, smoothness, consistency regularity and often do not reach the goal. Muscle strength in such a patient remains sufficient, it does not have paresis, therefore, the function of the cortical-muscular pathway
saved. This form of erratic movement is called **ataxia.** (from the Greek.
taxis - order, and - negative particle), or incoordination (from Latin coordinatio - ordering, in - not).
The pathogenetic essence of ataxia is either a violation of the repiproc

innervation, or in the termination of proprioceptive signaling (from muscle spindles, encapsulated bulbous bodies - Golgi tendon bodies) by one or the other ascending afferent pathway. Ceases to flow information about the degree of muscle tension at any given moment, about the results adaptive effects of functional systems. That side gets upset motor function, which in physiology began to be called the reverse afferentation, and in cybernetics, feedback.

There are several types of ataxias. The first one is associated with defeat muscle-cortical pathways (dorsal-thalamo-cortical pathway), i.e. with a disorder functions of the motor (kinesthetic) analyzer. The clinic describes disorders are called sensitive ataxia (with them, both suffer and coordination of movements, and muscular-articular feeling).
With severe sensitive ataxia in the upper limb, it is difficult performing even the simplest actions. The sick man cannot button the buttons without splashing, bring a glass of water to your mouth, just to hit the tip with your finger
nose. At rest, involuntary movements can sometimes be seen in the fingers of the hand,
resembling athetosis (pseudo-athetosis). Coordination of movements is also impaired in
lower limbs. When trying to touch one lower limb with the heel knee joint, the other shin describes zigzags, the heel gets higher joint, then below. The second phase of this test is also poorly performed. Heel of one bottom
the limb is held along the front surface of the lower leg, the other is not smooth, but jerky, with a deviation to the sides. Muscle tone in the affected the limbs is lowered in both the flexor and extensor muscles. V standing position, staggering is noted, especially when the feet are closed and simultaneous closing of the eyes **(Romberg's symptom).** Movement becomes unsure / feet jerkily rise and fall to the ground with a thud,
the patient walks with his head down, controlling the act of walking with the help of sight; v
in the dark, these disorders intensify.
Thus, **sensitive ataxia is** always associated with a disorder. deep sensitivity and functional separation of individual segments limbs with higher brain zones. Deep Disorder Detection sensitivity in ataxia allows us to talk about its sensitive form, about its dependence on the defeat of various parts of the kinesthetic analyzer. Another characteristic feature of this type of ataxia is its intensification with closing the eyes (when turning off the control of the visual analyzer). Itself the formation of movement coordination in early childhood is closely related to the activity of the visual analyzer.
Sensitive ataxia with involvement of the posterior cords of the lower half spinal cord (for example, with syphilitic damage to the nervous system or

in case of vitamin B12 deficiency - funicular myelosis) can resist
driven by the disappearance of deep reflexes in the lower extremities. it
is explained by the degeneration of not only the fibers of the thin bundle (axons of cells
intervertebral ganglia), but also their collaterals, which are the afferent part
arcs of deep reflexes. In other types of ataxia, the extinction of deep reflexes
usually not observed.

Another type of ataxia is associated with lesions of the cerebellar systems. I appear
the associated movement disorders are collectively called cerebellar
ataxia.

Given that the cerebellar worm is involved in the regulation of contraction
muscles of the trunk, and the cortex of the hemispheres - of the distal extremities, distinguish
two forms of cerebellar ataxia.

Static-locomotor ataxia - when the cerebellar worm is damaged,
They are mainly standing and walking. The patient stands with widely spaced
legs, swaying. When walking, the body deviates to the sides, gait
resembles the gait of a drunk. Turns are especially difficult. Deviation at

walking is observed towards the cerebellar lesion.

Stability is tested in the Romberg position: a patient in
standing position, offer to tightly move the feet; the head is slightly raised,
the upper limbs are lowered along the body (sometimes Romberg's pose is complicated,
offering to stretch the upper limbs to a horizontal level or the foot on
put one in front of the other on one line - in this position hold
balance is more difficult). Initially, the patient is in the Romberg position with open
eyes and then closed. With damage to the cerebellar systems, the patient in this
pose or sways in the appropriate direction (in both directions - with bilateral
defeat), or will not be able to stand at all with shifted feet -
positive Romberg symptom. This will be both open and closed.
eyes. When standing in the Romberg position, staggering in
anteroposterior direction (with damage to the anterior sections of the cerebellar worm).

If the imbalance in the Romberg position increases significantly when closing
eye, it is more characteristic of sensitive ataxia.

When examining walking, the patient is asked to walk forward in a straight line
lines with open eyes, and then do it with closed eyes. With good
when performing these tests, the patient is asked to walk in a straight line in such a way
so that the toe of one foot touches the heel of the other.

The flank gait is **also checked** - step movements to the side. At
this draws attention to the clarity of the step and to the possibility of a quick stop when

a sudden command. In case of damage to the cerebellar systems with these studies reveal a violation of the gait of the described nature. Such a the walk is called atactic or cerebellar. Lower limbs excessively unbend and thrown forward, the patient kind of dances, the body as if lagging behind them. When the patient tries to lean back, there is no flexion in the knee joints and in the lumbar observed in a healthy person the spine.

When the cerebellar systems are damaged, the combination of simple movements nii constituting a sequential chain of complex motor acts; this is denoted as **asynergy or dyssynergy** . Asynergy is defined in part nosti, using **Babinsky's test** : the patient is in a lying position on back on a hard bed without a pillow, upper limbs crossed over the chest; his offer to sit down from this position. When performing this movement in a patient the lower limbs rise up, and not the trunk. With one-sided lesion of the cerebellum, the corresponding lower limb rises higher another.

Sometimes the **Ozhbkhovsky symptom is investigated** : the patient is sitting or standing firmly

rests (presses) with the palms of the outstretched upper limbs on the palms of the next. With a sudden removal of the doctor's hands with a downward movement, the patient sharply

tilts the body forward; a healthy person in this case remains motionless or easily deviates posteriorly.

Asynergy of the proximal upper limbs is checked in the following way. Upper limb retracted to a horizontal level the patient flexes with force in the elbow joint (forearm and hand - in position pronation, the hand is folded into a fist). The examiner tries to straighten the forearm. the patient and with a sudden cessation of resistance, the subject's hand with force hits the patient's chest. In a healthy person, this does not happen as

rapid contraction of the antagonist muscles prevents shock. This is a symptom no kickback ”by Stuart-Holmes.

Another form of cerebellar ataxia is referred to as **dynamic ataxia** : it impairs the performance of various voluntary movements limbs. This type of ataxia mainly depends on the lesion of the hemispheres cerebellum. In the most pronounced form, these disorders are found in study of movements of the upper limbs. For this, the following are performed. tests.

Finger test: the patient first with open, and then with closed eyes with the index finger from the position of straightened and laid to the side the upper limb tries to touch the tip of the nose. On defeat of the cerebellar systems are observed overshooting, passing and emergence tremors of the hand when approaching the target - intentional tremor (Latin intendo - straining, lat. tremo - trembling). If a finger slip occurs when performing a test only with closed eyes, this is typical for a sensitive

ataxia.
Calcaneal-knee test: in the supine position, the patient bends the lower nude limb in the hip joint, and he must put the heel of one feet on the other knee. Then, lightly touching (or almost on weight), make a movement along the front surface of the lower leg down to the foot and back. This the patient makes the test with open and closed eyes. With cerebellar ataxia, the patient misses, hitting the heel in the knee area, and then the heel slides to the sides when holding it on the lower leg.
Samples for diadochokinesis (Greek diadochos - next, replacing); upper the limb is bent at the elbow to a right angle, the fingers are spread and slightly bent. In this position, pronation and supination of the hand are quickly completed. (imitation of screwing in a light bulb). With damage to the cerebellum, awkward, sweeping and asynchronous movements are observed - adiadochokinesis . Often at the same time, a slowed down rate of movements is revealed - bradykinesia.
Test for the proportionality of movements : the upper limbs are extended forward palms up, fingers spread (apart). At the command of the doctor, the patient must quickly turn the hands with the palms down (pronator test). On the side lesions of the cerebellum, there is excessive rotation of the hand - dysmetria (hypermetry). This phenomenon can also be detected during the test with a hammer: the patient holds the neurological hammer by the handle with one palm, and I and II fingers with the other - squeezes alternately the narrow part of the handle, then the elastic tray. At the same time, excessive movements are revealed - spreading fingers and their disproportionate closing.
Index test: a patient with a finger tries to hit the hammer, which which is moved in different directions.
Schilder's test : the patient stretches his arms forward, closes his eyes, then one upper limb is lifted up to a vertical level and on command lowers it to the level of the horizontally outstretched other hand. If the descending the upper limb will be below the horizontal level, this is **hypermetria** .
In addition to the violation of these tests with movements of the limbs, in case of damage cerebellar systems, other simple and complex motor ac- you. Let's note some of them.

Speech disorder - as a result of speech motor incoordination musculature, the patient's speech becomes slower (bradilalia}, loses smoothness, at the same time it becomes explosive, emphasis is placed on the unnecessary syllables - chanting speech.
Changes in handwriting - handwriting becomes uneven, oversized (megalography). The patient cannot draw a circle or other regular shape.
Nystagmus - rhythmic twitching of the eyeballs when looking to the sides or

up (a kind of intentional tremor of the oculomotor muscles). In suffering cerebellar systems, the plane of nystagmus usually coincides with the direction arbitrary movements of the eyeballs - when looking to the sides of the nystagmus of the horizontal, looking up and down - vertical. Sometimes nystagmus is congenital. Such nystagmus is usually present not only with the abduction of the ocular apples to the sides (with muscle tension), but also when looking straight ahead ("spontaneous nystagmus "). A.V. Triumfov (1974) proposed the following feature for distinguishing congenital from acquired nystagmus: if horizontal nystagmus with looking to the side is the result of an acquired disease of the nervous system, then when looking up it becomes vertical or disappears; congenital nystagmus when looking upward retains its previous character (horizontal or rotatory nystagmus).

In case of damage to the cerebellar systems, in addition to the violation of quality side of voluntary movements, muscle tone may change (muscle dystonia). Muscle hypotension is most often observed: muscles become flabby, lethargic, hypermobility of the joints is possible. Moreover, they can decline deep reflexes.

Coordination of movements is impaired with suffering from the frontal and temporal lobes and their conductors (tractus cortico-ponto-cerebellares). In such cases, they get upset walking and standing, the body deviates posteriorly and to the side opposite to the focus.

A mimic fall in the arm and leg is detected (hemiataxia). With this kind of violation coordination, other signs of damage to the corresponding lobes of the cerebral hemispheres.

Ataxia can also occur when the function of the vestibular analysator, in particular its propriopeptors in the labyrinth - labyrinth, or vestibular, ataxia. It upsets the balance of the body while walking the patient deviates towards the affected labyrinth. Systemic dizziness, nausea, and horizontal-rotatory nystagmus. On the side of the affected labyrinth, hearing may be impaired.

Thus, the disorder of coordination of voluntary movements is observed it occurs with damage to both the cerebellum itself and the conductors along which impulses from muscles, semicircular canals of the inner ear and cerebral cortex and are diverted from the cerebellum to the motor neurons of the cerebral trunk and spinal cord. Patients with lesions of the cerebellar systems at rest usually no pathological manifestations are found. Different kinds incoordination appears in them only with muscle tension.

SYMPTOMS OF DEFEATS OF THE BRAIN SHEETS. CHANGES IN SPINAL FLUID

The brain and spinal cord are covered with three membranes: hard, arachnoid and soft. The dura mater (pachymeninx) consists of two
leaflets. The outer one is tightly attached to the bones of the skull and the spine and is like
would be their periosteum. Inner layer (dura mater proper)
is a dense fibrous tissue. In the cranial cavity, both of these sheets
adjacent to each other, only in some places they diverge and form a wall
venous sinuses. An epidural is located in the spinal canal between the sheets
fiber - loose adipose tissue with a rich venous network.
The arachnoid membrane (arachnoidea) lines the inner surface of a hard and
many cords are connected to the pia mater. Arachnoid
covers the brain tissue and does not sink into the furrows. Pia mater (pia
mater or leptomeninx) covers the surface of the brain and spinal cord and, following their relief, grows together with the medulla.
The posterior and anterior roots, moving away from the spinal cord to the side and downward,
go through the meninges. Therefore, with meningitis and other meningeal
lesions can be involved in the process and roots. Between soft and spiderweb
the membranes have a space called the subarachnoid. It contains
the cerebrospinal fluid circulates in the cerebrospinal fluid - the cerebrospinal fluid. On
the base of the brain, the subarachnoid space expands and forms large
cavities filled with cerebrospinal fluid (basal cisterns). The largest of them is located
lies between the cerebellum and the medulla oblongata - cisterna cerebellomedullaris. V
the vertebral canal, the subarachnoid space surrounds the spinal cord. Below
its endings at the level of the vertebrae Li - L P it increases in volume, and in it
the roots of the cauda equina are located (terminal cistern, cisterns terminalis).
Cerebrospinal fluid is also found inside the brain and spinal cord,
filling the ventricular system: right and left lateral, III ventricle, water supply
brain (sylviev), IV ventricle, central spinal canal. From the IV ventricle
it enters the subarachnoid space through the median aperture IV
ventricle (paired opening of Magendie) and lateral aperture of the IV ventricle
(Lushka's unpaired foramen) in the posterior cerebral sail.
Cerebrospinal fluid is formed in the cells of the vascular plexuses of the head
brain. This led some authors to call pl. chorioideus and tela
chorioidea by the choroid gland of the brain.
The amount of cerebrospinal fluid in humans is relatively constant. V
on average, it is 120-150 ml. Most of the cerebrospinal fluid is located in the subarachnoid
space. The ventricles contain only 20-40 ml. It is produced
continuously in an amount of 600 ml during the day and is also continuously absorbed into
venous sinuses of the dura mater of the brain through the arachnoid villi.
The accumulation of such villi in the venous sinuses (especially a lot of them in the upper

sagittal sinus) are called arachnoid (pachyon) granulations. Per
due to the inflow and outflow of this liquid, the constancy of its volume in
ventricles and in the subarachnoid space. Partial absorption
fluids and into the lymphatic system, which is carried out at the level of the nerve sheaths,
into which the meninges continue. The movement of cerebrospinal fluid in
different directions associated with pulsation of blood vessels, breathing, head movements and
torso.
The physiological significance of cerebrospinal fluid is diverse. Before
in total, it serves as a hydraulic cushion for the brain, which provides
mechanical protection of brain tissue during shocks and concussions. However, she
turns out to be an internal environment that regulates absorption processes
nutrients by nerve cells and maintains osmotic and

oncotic balance at the tissue level. Cerebrospinal fluid has
also protective (bactericidal) properties, antibodies accumulate in it. She
takes part in the mechanisms of regulation of blood circulation in a confined space
cranial cavity and spinal canal. Cerebrospinal fluid does not circulate
only in the ventricles and in the subarachnoid space, it also penetrates into
the thickness of the medulla along the so-called perivascular cracks (space
Virchow-Robin). A small amount of it also enters the periendoneural fissures.
peripheral nerves.
With meningitis of various etiologies, hemorrhage in the subarachnoid
wandering and in some other pathological conditions, cli-
nic picture, called " **irritation syndrome of the meninges** ",
or, in short, “ **meningeal syndrome** ”. Its frequent terms are
headache, vomiting, pain with percussion of the skull and spine,
hypersensitivity (general hyperesthesia) to light, sound and skin
irritation. Typical signs of meningeal irritation are
tonic tension of some skeletal muscle groups: 1) extensor muscles
cervical spine; 2) flexor muscles of the hip and knee joints.
In severe forms of meningitis, persistent tonic tension of the listed
muscle groups leads to the formation of a peculiar posture. The patient lies on his side
the head is thrown back, the thighs are pressed to the abdomen, the shins to the
thighs. Sometimes tonic
some tension extends to the muscles that extend the spine
(opisthotonus). The described forced position of the body in such a pronounced degree
is relatively rare, however, the increased voltage of the listed
muscles are a constant occurrence in meningitis. Trying to passively tilt your head
forward with bringing the chin to the chest with irritation of the meninges
meets resistance due to repetitive reflex voltage
posterior cervical muscles. This symptom is called "rigidity of the occipital
muscles ”. It is more correct to talk about the rigidity of the posterior muscles.

The symptom described by the St. Petersburg clinician V.M.Kernig in
1882 Kernig's symptom is detected as follows: in a patient lying on his back
the examiner flexes the lower limb in the hip and knee joints under
right angle; in this starting position, they try to extend the knee
nomal joint, which with meningeal syndrome meets resistance. Flexors
the legs are tonically tense, straighten the lower limb at the knee joint
usually fails. Sometimes there is pain in the muscles (flexors of the lower leg), less often - in
the lumbar region and along the entire spine.
When examining the tone of the posterior cervical muscles (test for muscle stiffness occiput), also with Kernig's test, in addition to the above, there are also
distant reflex-motor reactions. They got the name
"Meningeal
Brudzinsky's symptoms ". Passive head tilt forward
causes slight flexion of both lower limbs in the hip and knee
joints - the upper symptom of Brudzinsky. Similar movement of the lower limbs
can be caused by pressure on the area of the pubic symphysis - anterior symptom
Brudzinsky. The same flexion movement in the contralateral inferior
limbs in the Kernig test is designated as "Brudzinsky's lower symptom".
With meningitis, Guillain's symptom is also observed: compression of the quadriceps muscle of the thigh
on the one hand, it causes involuntary flexion in the knee and hip
tavah of the opposite limb. With meningitis in children, there is a symptom
suspension by Lesage. Research methodology: the child is lifted,
supporting in the armpits; while the lower limbs of his
are arbitrarily pulled up to the stomach by bending them in the hip and knee
joints.
What is the mechanism of movement (tonic) disorders in cerebral irritation
shells? There is a widespread point of view that tonic tension of the posterior cervical

muscles and flexor muscles of the lower leg with meningitis is a reflex
a defensive reaction that reduces the tension of the posterior roots, relieves pain. V
of the movement disorders described above, there is an increased tonic
muscle stretch reflex. When testing for the stiffness of the occipital muscles (when identifying
of Kernig's symptom), additional stretching of the corresponding
muscles and the most dramatic manifestation of the tonic myotatic reflex.
The true symptoms of irritation of the pain receptors of the meninges
ankylosing spondylitis is a symptom: when tapping on the zygomatic arch, the head
pain and a painful grimace involuntarily occurs on the corresponding half of the face.
If the patient has symptoms of irritation of the meninges and
cerebrospinal fluid, inflammatory changes are detected, then
meningitis is diagnosed. Blood in the cerebrospinal fluid indicates
subarachnoid hemorrhage. However, with various diseases (pneumonia,
appendicitis, etc.), especially in children, symptoms of irritation may appear

meninges without any changes in the cerebrospinal fluid. Such
cases speak of meningism.
There are several ways to extract cerebrospinal fluid:
1) lumbar puncture of the terminal cistern; 2) suboccipital puncture of the cerebellar medullary cistern; 3) cisternal puncture through a milling hole in the skull.
Lumbar puncture is relatively safe, its technique is simple. Dot-
You can bed in the patient's position, both sitting and lying. The patient is placed on a hard
the bed in a position on its side. The lower limbs should be bent at the hip
and knee joints. The head is tilted as much as possible until the chin touches
with the sternum.
To determine the puncture site, palpate the most elevated
points of the iliac crests, mark them and connect with a straight line with a cotton
a swab moistened with a 3% alcohol solution of iodine. This is the so called line
Jacobi; it passes at the level of the spinous process of the Liv vertebra (according to some
authors, in the interval L III - Liv). Puncture is performed between the spinous processes
Lm - Liv or Liv - Lv.
After the puncture site is outlined, the skin around it on a sufficiently large
distance is treated twice with 70% alcohol, then lubricated with 3% alcohol
iodine solution. To avoid getting it on the meninges and irritating them
before the puncture, the excess is removed with a gauze ball dipped in alcohol. Then
local anesthesia is performed with a 0.5% solution of novocaine before formation
"Lemon peel". Infiltrate with a solution of 5% novocaine in
an amount of about 3-5 ml during the future puncture to a depth of 2-4 cm.After
passage of the dura mater (in adults it happens at a depth of 4-7 cm, in
children - up to 3 cm) there is a feeling of “failure” of the needle. After that, carefully
remove (usually not completely) the mandrel from the needle. Noticing the release of fluid,
immediately insert the end of the connecting tube of the pressure gauge into the needle pavilion and
measure the cerebrospinal fluid pressure. In the patient's lying position, it is
It consumes an average of 100-180 mm of water column. In the sitting position, the cerebrospinal fluid pressure is somewhat
above - 200-300 mm of water. Art. To measure the cerebrospinal fluid pressure, it is usually used
simple water pressure gauge - sterile glass tube with a lumen diameter of 1
mm (if the cerebrospinal fluid level rises by 10 cm, then this corresponds to a pressure of 100 mm
water Art.). Pathological processes localized in the cranial cavity and in the
the spinal canal, can disrupt the circulation of cerebrospinal fluid.
Under normal conditions, there is a close relationship between venous and
cerebrospinal fluid pressure. The liquorodynamic tests described below during the block
CSF pathways are based on the registration of violations of this ratio.

Kwekenstedt's test: assistant I and 11 fingers of both hands covers the lower
the nude part of the patient's neck and squeezes the cervical veins for 5 s, maximum 10 s. O
the onset of an increase in venous pressure in the cranial cavity is judged by swelling

facial and temporal veins, hyperemia and cyanosis of the skin of the face and injection of the sclera.
Venous plethora of the brain leads to an increase in intracranial
pressure and an increase in cerebrospinal fluid pressure. The level of the cerebrospinal fluid column.
the gauge tube rises significantly. After the cessation of vein compression
the cerebrospinal fluid pressure quickly decreases to the initial level. All this is happening
with a passable subarachnoid space. With complete obstruction
CSF pathways within the spinal cord with compression of the cervical veins CSF
the pressure does not rise. With a partial block, the cerebrospinal fluid pressure rises unknowingly
decreases significantly and slowly after the cessation of compression.
Pussep test: the patient's head passively leans forward, chin
presses against the chest. In this case, there is a partial compression of the cervical veins.
The cerebrospinal fluid pressure at this moment rises by 30-60 mm of water. Art. On return
head to its original position, the cerebrospinal fluid pressure drops to the previous figures. At
block of the subarachnoid space, Pussep's test of cerebrospinal fluid pressure is not enhances.
Stukey's test: the doctor's assistant presses his hand on the anterior abdominal wall
at the level of the navel for 20-25 s. As a result, the abdominal veins are compressed and
stagnation occurs in the vein system inside the spinal canal. CSF pressure at
this increases by 1-1.5 times. After the cessation of compression, it decreases to
initial level. This reaction of cerebrospinal fluid pressure persists in the presence
blocking the subarachnoid space at the level of the cervical or thoracic
department of the spinal column.
It is customary to depict the results of CSF dynamic tests graphically: along the axis
abscissas mark the phase of one or another moment of the experiment, and along the ordinate - the value
cerebrospinal fluid pressure. At the end of the pressure fluctuation measurements, 5-8 ml are extracted
cerebrospinal fluid for clinical research. The volume of extracted cerebrospinal fluid depends on
the patient's condition, the nature of the disease, the level of cerebrospinal fluid pressure. At the end
nii the listed manipulations quickly remove the puncture needle, the puncture site

grease with 5% alcohol solution of iodine and close with a sterile cotton ball (better - moistened with glue). The patient is prescribed bed rest for 2-3 days.
Suboccipital and ventricular punctures are performed by a neurosurgeon.
Suboccipital puncture. The patient's neck and nape are carefully shaved, then
treated with 5% alcohol solution of iodine and 70% alcohol. Puncture is performed in position a patient lying on his right side; a roller is placed under the head so that the posterior midline torso and head were on the same horizontal axis with the plane on which the patient lies.
The head is tilted forward. Palpate the spinous process of the Cn and puncture the tissues over the process along
the middle line. Then the needle is directed obliquely upwards at an angle of 45-60 ° to the plane passing through
the middle line of the occipital bone. At the moment of sensation when the occipital bone is touched, the needle
pull back, the outer end is gently lifted and sliding along the scales of the occipital
bones, the needle is inserted by movement into the depth of 3-3.5 cm.At the moment of puncture of the atlanto-occipital membrane
some resistance is determined, after which a “failure” is felt due to hit
needles into the cerebellar cistern. After removing the cerebrospinal fluid, the needle is removed with a smooth motion.
The puncture site is sealed. Bed rest is prescribed for 2-3 days. Suboccipital puncture contraindicated for volumetric processes in the posterior cranial fossa, craniospinal tumors localization.
Extraction of 5-8 ml of cerebrospinal fluid proceeds without complications. Occasionally, phenomena can be observed
post-puncture meningism (for several days, the patient has a headache,
sometimes vomiting occurs). However, there are diseases in which the lumbar
puncture is life-threatening and requires special care. These include brain tumors,
especially when located in the posterior cranial fossa. If a posterior cranial tumor is suspected pits, high intracranial hypertension with congestive optic discs
lumbar puncture is contraindicated.
If you suspect intracranial hypertension, you should have a syringe ready with a warm isotonic sodium chloride solution for the forced introduction of fluid when
signs of wedging of the cerebellar tonsils into the foramen magnum. Puncture in such patients must be carried out in the conditions of the neurosurgical department. Requires caution when checking
puncture in patients with spinal cord tumors (possibly increased paresis and disorders sensitivity - the so-called jamming syndrome "). After extracting the required amount

cerebrospinal fluid, it is advisable to re-measure the cerebrospinal fluid pressure, which is almost always falls below the original level.
The definition of the Ayala index (J) according to the following formula may be of practical importance:
J = V • Pi / Pi, where V is the amount of CSF taken (ml); Pr is the residual pressure of the cerebrospinal fluid (mm of water column); Pi
- initial pressure of cerebrospinal fluid (mm of water column). In healthy individuals, the value of the index varies within
5.5-6.5. With an index above 7.0, hydrocephalus or serous meningitis can be assumed, the index less than 5.0 is typical for the subarachnoid space block.
The main pathogenetic mechanisms of increasing intracranial pressure

phenomena are excessive production of cerebrospinal fluid, the difficulty of its outflow at normal
malnom production, a combination of hypersecretion with impaired
outflow. Difficulties
outflow of cerebrospinal fluid can be caused by impaired patency
cerebrospinal fluid (occlusive forms) and its insufficient absorption
(resorptive forms) or their combination. Increased spinal pressure
fluid is observed in inflammatory intracranial processes (due to
excess fluid production or increased permeability of the vessel walls
brain), with tumors, parasitic cysts that increase the volume of brain tissue,
cicatricial adhesive processes between the soft and arachnoid membranes, with
craniostenosis, trauma, etc.
An increase in cerebrospinal fluid pressure leads to an increase in venous pressure in
cranial cavity, impaired metabolic function of the cerebrospinal fluid, etc.
venous hypertension leads to the expansion of the diploic veins and fundus veins, which
can be determined by ophthalmoscopy.
The symptom complex of increased intracranial pressure consists of
cerebral symptoms associated with compression of the brain and its membranes. Sick
complain of a bursting dull headache when lying down, getting worse
at night and after sleep, dizziness, nausea, “cerebral” vomiting. They reveal
bradycardia, enlarged veins in diameter and edema of the optic nerve head,
plasmorrhage in the fundus; on radiographs of the skull - premature
pneumatization of the main sinus, osteoporosis of the back of the sella turcica, expansion
diploic veins, enhancement of the pattern of digital impressions on the bones of the
cranial vault, etc.
Hypertensive-hydrocephalic syndrome is caused by an increase in the number of
va cerebrospinal fluid in the cranial cavity and increased intracranial
pressure. By localization, hydrocephalus is internal (cerebrospinal fluid accumulates in
ventricles of the brain), external (cerebrospinal fluid accumulates in the sub-arachnoid
space) and mixed.
With blockade of the cerebrospinal fluid at the level of the median and lateral apertures IV
the ventricle or aqueduct of the brain develops an occlusive syndrome. With acute
occlusion at the level of the apertures of the IV ventricle, the IV ventricle of the brain expands and
Bruns syndrome is observed: the sudden development of nausea, a sharp headache,
dizziness, ataxia; breathing and cardiovascular activity are disturbed.
These phenomena are aggravated by turning the head and trunk. In cases of occlusion
aqueduct of the brain develops a quadruple syndrome: nausea, vomiting,
oculomotor disorders, vertical nystagmus, paresis of gaze up or down,
“Floating” gaze, coordination disorders.
For occlusion at the level of the interventricular foramen (Monroe foramen)
expansion of the lateral ventricles develops with general cerebral symptoms and

hypothalamic-pituitary disorders.
CSF hypertensive syndrome should be differentiated from dislocation
tional syndrome and the syndrome of tentorial tentorium Burdenko-Kramer.
Dislocation syndrome - displacement of the brain stem that occurs with edema and swelling of the brain, hematoma, tumor, etc. With volumetric supratentorial
brain stem formations (anisocoria, dysfunction of the cranial nerves,
reticular formation with a change in the clarity of consciousness, etc.), with brain damage
in the posterior cranial fossa, symptoms of dysfunction of the frontal lobe appear.

Burdenko-Kramer tentorial tinge syndrome occurs when irritated
zenia of the tentorium of the cerebellum, innervated by the branch of the optic nerve of the V pair: pain in
eyeballs, photophobia, blepharospasm, lacrimation, sometimes increased
separation of mucus from the nose. When wedging and pinching the brain stem and cerebellum into
tenderloin, these symptoms of irritation of the tentorium of the cerebellum are joined by
intense headache with vomiting, dizziness, involuntary rolling back
heads, upward gaze paresis, nystagmus, Hertwig-Magendie syndrome, sluggish pupil response
to light, bilateral pathological reflexes against the background of oppression of deep reflexes.
Normally, the cerebrospinal fluid has a relative density of 1.005-
1.007; her reaction is slightly alkaline; pH, like blood, is close to 7.4; amount of protein from
0.2 to 0.4 g / l. The content of inorganic substances in the cerebrospinal fluid is the same as in the blood:
chlorides - 7-7.5 g / l; K - 4.09-5.11 mmol / l; Ca - 1.25-1.62 mmol / l. Concentration glucose in the cerebrospinal fluid compared with blood is about half as much and is 0.45-
0.65 g / l. The composition of the cerebrospinal fluid is highly dependent on the functioning
blood-brain barrier. The blood-brain barrier is understood as
histohematogenous barrier, selectively regulating the metabolism between blood
and the central nervous system. It provides relative immutability
composition, physicochemical and biological properties of cerebrospinal fluid and the adequacy of micro-
environment of individual nerve elements. Morphological substrate of hemato-
the encephalic barrier are anatomical elements located between
blood and nerve cells: capillary endothelium, basement membrane of a cell, glia,
choroid plexuses, membranes of the brain. A special role in the implementation of the function
The blood-brain barrier is performed by neuroglial cells, in particular astrocytes.
Their terminal perivascular legs are adjacent to the outer surface of the capillaries,

selectively extract substances necessary for nutrition from the bloodstream
neurons, and return the products of their metabolism into the blood. In all structures
the blood-brain barrier, enzymatic reactions can occur,
contributing to chemical transformations of substances coming from the blood
(oxidation, neutralization, etc.). Blood-brain barrier permeability
is not the same in different parts of the brain and, in turn, can change.
For example, in the hypothalamus, the permeability of this barrier in relation to
biogenic
amines, electrolytes, some viruses, toxins are higher than in other departments
the brain, which ensures the timely receipt of information in a humoral way
to the higher vegetative centers. In relation to the used chemotherapeutic
drugs and antibiotics permeability of the blood-brain barrier
selective, which leads to the need for endolumbar administration of drugs.
Normally, there is some difference in the composition of the ventricular and
lumbar cerebrospinal fluid.
Normally, the cerebrospinal fluid is transparent, colorless, but in some diseases it
specifications are subject to change. With meningitis, it becomes cloudy from
the presence of a large number of shaped elements. Sometimes the cerebrospinal fluid
acquires
yellow-greenish color. This is the so-called xanthochromia of the cerebrospinal fluid,
which
observed, for example, with pneumococcal meningitis, after subarachnoid
hemorrhages, with brain tumors. With blockade of the spinal subarachnoid
space by a tumor in the cerebrospinal fluid, the protein content rises sharply and it
takes on a jelly-like appearance. In combination with xanthochromia, this
phenomenon
is referred to as congestive Frouan's syndrome or Nonne's compression syndrome.
Normal CSF contains up to 5 lymphocytes in 1 ml. In patients with various
diseases of the nervous system, their number can increase to tens, hundreds and
even thousands. An increase in the number of cells in the cerebrospinal fluid is called
pleocytosis . In these
cases, neutrophils, eosinophils, monocytes, obese
cells, plasma cells, macrophages. An important diagnostic value is
detection of tumor cells in the cerebrospinal fluid. This can be seen in sarcomatosis
and

carcinomatosis of the meninges, cerebellar medulloblastoma and other neoplasms
brain development located close to the cerebrospinal fluid pathways.
With the help of biochemical studies of cerebrospinal fluid, protein content is
determined
(Pandy test, Roberts-Stolnikov method). Normally, the cerebrospinal fluid contains
0.2-0.4 g / l
squirrel. With the help of the Nonne-Apelt reaction, the content is tentatively revealed
globulin fraction of protein in cerebrospinal fluid. The severity of the reaction is
assessed in

crosses (up to four). The statement of
colloidal reactions Lange, Takata-Ara and others.
With a number of infectious diseases of the nervous system, it turns out
it is necessary to conduct a bacteriological examination of the spinal
liquid (bacterioscopy and sowing it on nutrient media). Only this way
it is possible to establish with full certainty the etiology of the disease
and prescribe the most rational treatment. This primarily applies to diagnostics
purulent meningitis.
In some cases, it is necessary to conduct a virological study
cerebrospinal fluid.
If you suspect a syphilitic disease of the nervous system, you should
produce immunological Wasserman reactions and sedimentary reactions. Necessary
take into account that the Wasserman reaction in the cerebrospinal fluid is carried out
according to a special technique, cerebrospinal fluid
requires more than serum (up to 1 ml). Now they use more sensitive
the reaction of immobilization of pale treponema (RIBT).
In some patients, there is an isolated increase in the amount of protein.
(hyperproteinosis) in the cerebrospinal fluid, the number of cells remains
unchanged. This painting is called
protein-cell dissociation. It occurs in tumors of the head and spinal
brain, spinal arachnoiditis with a block of the subarachnoid space and
venous stasis.
More often, a simultaneous increase in the number of cells is observed in the
cerebrospinal fluid.
(pleocytosis) and an increase in the amount of protein (hyperproteinosis). Such a
picture of cerebrospinal fluid
happens with meningoencephalitis of different etiology.
Already the sight of cerebrospinal fluid (turbidity) makes one think of
meningitis. Count
cellular elements reveals pleocytosis. Globulin reactions are
positive, the total protein content is increased. Smear examination from sediment
cerebrospinal fluid can reveal predominantly neutrophilic pleocytosis, the presence
gram-negative diplococci. Their intracellular localization causes
suspected meningococcal meningitis. If gram-positive
extracellular diplococci, pneumococcal meningitis should be assumed. For
clarification of the diagnosis requires additional research, in particular
crops of cerebrospinal fluid, which must be carried out in accordance with all the rules
bacteriological technology. To avoid contamination, it is best to do so
so that the tube with the culture medium is directly placed under the drops of liquid
from the needle
during a puncture. If purulent meningitis is suspected, the most suitable for
sowing are culture media containing blood or blood serum.
Only with bacterioscopy of cerebrospinal fluid using Ziehl-Nielsen staining can
detect a rare form of torulent meningitis caused by a special type
yeast fungus.
With tuberculous meningitis, the cerebrospinal fluid may remain clear. However, in

in many cases, after 12-24 hours, a thin
cobweb film. Mycobacteria can be sown from it; protein samples
turn out to be positive. The number of shaped elements is increased, but less
degree than with purulent meningitis; usually lymphocytes predominate. In a smear from
Films when stained according to Ziehl-Nielsen may show mycobacterium tuberculosis.
Sowing the cerebrospinal fluid on a special nutrient medium can give growth only through
few weeks.
In some diseases, pleocytosis in its severity prevails over

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the multiplicity of the increase in the protein content, cellular-protein dissociation. it occurs in many neuroinfections.
Detection of erythrocytes or their decay products in the cerebrospinal fluid indicates the penetration of blood beyond the walls of the cerebral vessel and is important in differential diagnosis of the nature of cerebral stroke. With significant
impurities of blood in the cerebrospinal fluid diagnose subarachnoid hemorrhage. In both
cases, the question arises whether the admixture of blood is accidental, depending on the injury
a vessel with a puncture needle, or the patient has a hemorrhage in the cranial cavity or the spinal canal. There is a trick with which you can set
accidental ingestion of blood into the cerebrospinal fluid ("travel blood"). To do this, collect dripping
liquid from the puncture needle into several test tubes. If in each subsequent
In the test tube, the liquid is more and more cleared of blood, which means its source was close. V
in such cases, erythrocytes in test tubes quickly settle, the supernatant remains
Xia colorless. "Traveling" blood can be distinguished from blood in
subarachnoid space when applying a drop flowing from the puncture
CSF needles on white filter paper. If the blur has
homogeneous pink or red color, this indicates hemolysis due to
prolonged contact of blood with cerebrospinal fluid in the subarachnoid space. At accidental ingestion of blood, the stain has 2 zones: red (with aggregated erythrocytes in the center) and colorless (due to diffusion of normal cerebrospinal fluid) - by
edges.
If an admixture of blood in the cerebrospinal fluid is associated with a hemorrhagic stroke, the fluid
above the sediment has a yellowish color (xanthochromia). In a smear from the sediment under
a microscope can see destroyed erythrocytes, sometimes macrophages,
loaded with hemoglobin.

GENERAL BRAIN SYMPTOMS

CRANIAL NERVES AND SYMPTOMS OF THEIR LESION

The main components of the peripheral nervous system are nerves,
which connect the central nervous system to other parts of the body, and the ganglia - groups of nerve cells (nodes) located at different points in the nervous system

A nerve is a bundle of motor (motor) and sensory (sensory) fibers
together with connective tissue and blood vessels. Large nerves (there are 43) go actually from the nervous system: 12 pairs leave the lower part of the brain
(cranial nerves) and 31 pairs from the spinal cord (spinal nerves).

Cranial nerves - nerves whose roots are connected to the trunk
brain. They serve mainly the senses and muscles of the head,
although a very important cranial nerve - the vagus (X pair) - serves the organs digestion, heart and air passages in the lungs. Some cranial
nerves, such as the optic nerve to the eye (II pair), contain only sensory
fibers.

Thus, a person has **12 pairs of cranial nerves** that exit at the base
brain:

I - olfactory (nervus olfactorii),
II - visual (n. Opticus),
III - oculomotor (n. Oculomotorius),
IV - block (n. Trochlearis),
V - trigeminal (n. Trigemenus),
VI - abducting (n. Abducens),
VII - facial (n. Facialis),
VIII - auditory, or vestibular-cochlear (n.vestibulocochlearis),
IX - glossopharyngeal (n. Glossopharyneus),
X - wandering (n. Vagus),
XI - additional (n. Accesorius),
XII - sublingual (n. Hypoglossus).

Of them:

- Three pairs - sensory (sensitive) - I, II, VIII,
- Six pairs - motor (motor) - III, IV, VI, VII, XI, XII,
- Three pairs - mixed - V, IX, X.

All of these nerves innervate the muscles of the face, larynx, pharynx, tongue and part of the neck, and
vagus nerve - muscles of internal organs.

Sensory fibers of the cranial nerves are peripheral
olfactory, visual, gustatory, auditory and skin analyzers, with their
lesion, pain and other sensory disorders appear. Inflammatory
processes, trauma, tumors, strokes and some other pathological processes can cause various symptoms of cranial nerve damage - paresis, paralysis,

neuropathy and neuralgia, neuritis. cranial nerve lesion

I pair - olfactory nerve

With a lesion, hyposmia (decreased sense of smell), or anosmia is observed (lack of smell). As a rule, they are one-sided. When irritated temporal cortex (for example, by volumetric process) olfactory hallucinations - dysosmia and parosmia.

There is also a pathology called olfactory agnosia (unrecognition familiar smells).

The function of smell is investigated using various aromatic substances. (peppermint drops, perfume, ethyl or camphor alcohol), with the nasal passages are closed alternately. Do not use ammonia and other substances with pungent odors, since the trigeminal nerve (V pair) can react with irritation their branches, that is, the appearance of pain.

II pair - optic nerve

With a complete break of the optic nerve, amaurosis (blindness) occurs. As a consequence

various pathological processes are possible amblyopia or hemiambliopia (decreased visual acuity due to toxic damage to the orbital part optic nerve; in the second case, visual impairment occurs in opposite fields), hemianopsia (loss of visual fields), which is characterized by blindness external (temporal) or internal visual fields (there are several types hemianopsia, which differ depending on the degree of nerve damage), scotomas - loss of fields of view by sectors.

When the visual cortex (occipital lobe) is irritated, visual hallucinations. With damage to the outer surface of the occipital lobe (on the left - in right-handers

and vice versa) visual agnosia can also be observed - the patient loses the ability recognize objects by their appearance.

Color blindness (violation of color perception) is detected using multicolor tables.

Studies of visual acuity are carried out using Kryukov tables (letters decreasing size), the study of visual fields - using the perimeter. Fields vision can be checked by stretching a towel in front of the patient's face, which he should be divided in half (in the presence of hemianopsia, the patient divides the towel into

unequal parts) or with a slit lamp. Important in

neurology has a study of the fundus, the data of which can provide valuable information about the presence of a pathological focus in the brain (volumetric processes).

III pair - oculomotor nerve

Anatomy and physiology:

The nucleus of the oculomotor nerve is located at the bottom of the Sylvian aqueduct, on

the level of the anterior tubercles of the quadruple in the cerebral peduncle. The nerve leaves the skull along with
abducent, block and 1st branch of the trigeminal nerve through the superior orbital slit. Innervates 5 external (striated) and 2 internal (smooth) muscles.
The nucleus of the oculomotor nerve consists of 5 cell groups: two outer large cell nuclei, two small cell nuclei (Yakubovich) and one internal, unpaired, small-cell nucleus of Perlea. From the paired outer large cell nucleus fibers are emitted for the following external muscles: lifting the upper eyelid, turning the eyeball upward and somewhat inward, turning the eyeball apple upward and somewhat outward, moving the eyeball inward, moving the eyeball downward and somewhat inward. From paired small cell (parasympathetic) nucleus Yakubovich fibers go to the smooth inner muscle of the eye -
constricting the pupil. From unpaired internal small cell (accommodative) the nuclei are parasympathetic fibers for the ciliary muscle.
This nerve provides movement of the eyeball and innervates the muscle, constricting the pupil and carrying out the reaction of the pupils to light.
When this nerve is damaged, the following symptoms may appear:
- ptosis (drooping of the upper eyelid),

- diplopia (double vision) when looking up and in,
- mydriasis (pupil dilation),
- exophthalmos (standing of the eyeballs from the orbits),
- divergent strabismus and impaired convergence reaction (ability to reduce eyeballs to the bridge of the nose) and accommodation (the ability to clearly see objects,
those who are closer to the distant point of clear vision; accommodation disorders are due to
paralysis of the ciliary muscle).
Symptoms of oculomotor nerve damage can be:
- anisocoria (difference in pupil size),
- nystagmus,
- complete immobility of the pupil,
- loss of pupillary reactions to light - friendly constriction of the pupils.

IV pair - trochlear nerve

Anatomy and physiology:
The core is located in the bottom of the Sylvian aqueduct at the level of the rear hillocks
quadruple. Fibers from the nucleus make a complete crossover in the anterior cerebral sail. The block nerve leaves the cranial cavity through the superior orbital fissure. Innervates in the orbit the superior oblique muscle, which rotates the eyeball outwards and downwards.
With a lesion, the following symptoms occur: diplopia (double vision) with looking down, i.e. under his feet, and a converging squint. Is amazed in isolation extremely rare, most often due to exacerbation of chronic otitis media.

V pair - trigeminal nerve

The name of this nerve is due to the fact that it has three branches:

1.ocular,

2.maxillary and mandibular nerves, which supply nerve
the ends of the scalp, forehead, nose, upper and lower eyelids, cheeks and lips, and teeth, mucous membranes of the nose, gums, tongue and chewing muscles.

Due to the fact that the trigeminal nerve is mixed, with its defeat
there are sensitive (hypesthesia, hyperesthesia or pain) and motor
disorders (paralysis of the chewing muscles). There are strong paroxysmal
pain in the innervation of this nerve - in one or more of its branches. Such
pathology is called trigeminal neuralgia, which is common
disease. The cause may be pathological processes leading to
narrowing of the holes through which the branches of the nerve enter the cranial cavity. These are local
diseases (sinuses of the nose, ear, eyes, teeth; purulent processes of the skin and subcutaneous tissue of the face; periostitis, herpetic ganglioneuritis), general infections,
facial trauma, and in elderly patients - atherosclerosis of the vessels in the
face. Among the others
reasons - multiple sclerosis.

Pain in the area of this nerve, simulating neuralgia, can be observed with
tumors of the middle cranial fossa, trigeminal neuromas, arachnoiditis
the base of the brain. The process is localized not only in the peripheral part of the nerve, but,
often - in its central segments. The disease also occurs due to irritation
the nerve itself, and during various processes, in particular, angiospasms in the gasser's node.

Only one of the three branches of the nerve may be affected, but it is possible neuralgia of two or all three branches. In the area of innervation of the trigeminal nerve can

sometimes also radiate pain associated with a disease of the abdominal organs, uterus.

The trigeminal nerve is rich in vegetative fibers, and therefore the main
a manifestation of the disease is intense and burning painful paroxysms in the zone innervation of the affected branch (s) lasting from a few seconds to 1-2 minutes, repeating from several to tens of times a day. They are often accompanied
gustatory paresthesias, pain tics, vegetative-vascular disorders: hyperemia and edema face, increased secretion of saliva, lacrimation, impaired sweating,
nasal congestion, pain in the heart, increased blood pressure, sometimes - photophobia.

Trigger points are found on the face (in the upper orbital, infraorbital
and chin openings - the exit site of the trigeminal nerve - its I, II and III branches), irritation which causes an attack of pain. Depending on the localization of the process

(neuropathy of the III branch of the nerve) simultaneously possible spasms of the facial muscles -
paresis of the chewing muscles.
An attack of pain can be triggered by external influences
(touch, wind, loud sound), facial expressions while eating, laughing, coughing, conversation, vivid emotions. Characterized by a chronic course of the disease with all
increasing exacerbations, in connection with which there is a gradual asthenization sick.
Research methodology: the state of the chewing muscles, mandibular,
corneal and superciliary reflexes, study of skin sensitivity in areas
innervation of all three branches of the trigeminal nerve, as well as in the zones of segmental
innervation. Study of sensitivity (general and gustatory) in the anterior 2/3 of the tongue.

VI pair - abducens nerve

Anatomy and physiology:
The nucleus is located dorsally in the pons varoli, at the bottom of the rhomboid fossa. It leaves the cranial cavity through the superior orbital fissure into the orbit, where it innervates
the muscle that abducts the eyeball outward.
With a lesion, a convergent strabismus appears, diplopia (double vision) with looking towards the affected muscle (outward), the inability to rotate the eye apples outwards.
In case of damage, isolated paralysis of the rectus muscle of the eye occurs, which leads to converging strabismus, diplopia (inability to turn this eye
outwards), especially when looking towards the affected muscle; sometimes - to dizziness and a forced position of the head.
Nuclear lesions are accompanied by paralysis (or paresis) of the facial musculature and central paralysis of the limbs (Fauville symptom), Possible also gaze paralysis in the direction of the affected muscle and focus.
A neurological hammer is used to study the function of these nerves.
with the help of which the mobility of the eyeballs is checked (they offer to look up, down, outward, inward). Pay attention to the width and uniformity of the eye slits and on the shape and size of the pupils.
The reaction of the pupils to light is checked with a flashlight or with your palms, first closing his eyes tightly and then quickly lifting one of his hands.
Neuropathies of III, IV and VI pairs of cranial nerves (oculomotor, block and diverting) are considered together, because normally their functions are interrelated. Nerves are often affected simultaneously or apart by a fracture of the base of the skull,

basal arachnoiditis, encephalitis, bone pathology in the orbit, cavernous sinusitis, diabetes mellitus, mushroom poisoning, tumors. With joint paralysis this group of nerves (ophthalmoplegia totalis), the movement of the eye is completely excluded.

Methods for studying the function of the eye-motor nerves.
The width of the eye slits, the movement of the eyeballs in all directions, the state of the pupils (their size, shape), the reaction of the pupils to light, convergence and accommodation, eyeball standing (enophthalmos, exophthalmos). If there is a hidden failure (complaints of diplopia with complete preservation of the mobility of the eye apples) research with red glass (consultation of a neuro-ophthalmologist, ophthalmologist).
Neurological syndromes of damage to the oculomotor nerves.
Weber's syndrome is a pathological process in the brain stem: paralysis oculomotor nerve, accompanied by paralysis of the opposite limbs.
Benedict's syndrome - oculomotor nerve palsy and cerebellar ataxia opposite limbs (red nuclei are involved in the pathological process).
Fauville syndrome - abducens and facial nerve palsy with paralysis opposite limbs (pathological process in the pons varoli).
Complete ophthalmoplegia - no movement of the eyeball, pupillary no reactions (phenomenon of persistent mydriasis). External ophthalmoplegia - movement the eyeball is absent, pupillary reactions are preserved. Ophthalmoplegia internal - the movements of the eyeball are not disturbed, there are no pupillary reactions.

VII pair - facial nerve

Anatomy and physiology of the facial nerve:
The facial nerve is mixed. The motor nucleus of the nerve is located in bridge, the axons of the cells bend around the nuclei of the abducens nerve, forming the inner knee facial nerve. The parasympathetic nucleus is nucl. salivatorius sup., carrying out innervation of the submandibular and sublingual salivary glands, as well as lacrimal gland. The sensitive portion of the nerve is represented by cell processes the geniculate node (homologue of the spinal ganglia), the dendrites of which through the chorda tympani anastomoses with branches of the trigeminal nerve and ends with gustatory receptors in the anterior 2/3 of the tongue. The axons of the cells of the geniculate node as part of the facial nerves enter the brain stem and are sent to the nucl. tractus solitarii from glossopharyngeal nerve. At the base of the brain, the nerve exits in the cerebellar pontine angle and further goes through the internal auditory canal into the fallopian canal. Here the nerve forms outer knee. From the cranial cavity, it exits through the styloid opening and, passing through the parotid salivary gland, it innervates the facial muscles of the face, some muscles of the head and neck (parotid muscles, the posterior abdomen of the digastric muscles, platysm).

Central neurons for the facial nerve are located in the lower section
precentral gyrus. For innervation of the upper facial muscles
cortico-nuclear fibers approach the nucleus of their own and the opposite side, and lower - only with the opposite.
In the face area, it forms many branches, the so-called "crow's feet",
and innervates all facial muscles, and also gives branches for the anterior third of the tongue
and salivary glands.
The defeat of the facial nerve causes paralysis of the facial muscles, which, in depending on the cause of the occurrence, is called neuropathy (neuritis, paralysis) facial nerve.

Neuropathy of the facial nerve (neuropathea (neuritis) n. Facialis) - the most common disease among mononeuropathies. In most cases, against the background of infection and
cooling, the nerve is compressed in a narrow bone canal, followed by its
ischemia and degenerative changes. Congenital contributes to compression narrowness of the channel or its outlet. Sometimes diseases are preceded by local infections (mumps, exacerbation of chronic otitis media) or trauma
parotid region.
Spontaneously neuropathy occurs due to an infectious lesion
inner ear; with syphilitic diseases of the brain (basilar specific
meningitis, limited meningitis of the base of the brain). Of the infections, rheumatism is noted,
flu (as a complication, this disease can be included in the picture of postinfluenza encephalitis), viral infections (acute poliomyelitis). Of organic diseases
should be borne in mind of the tumor (primarily - the cerebellopontine angle), diffuse sclerosis. In children, it occurs with poliomyelitis and in cases of birth trauma.
The clinical picture is based on movement disorders in the form of weakness and atrophy of all facial muscles of the face, usually on one side.
Due to the greater strength of the muscles of the intact half of the face, it
asymmetry: on the side of the paresis, a fold on the forehead does not form, the forehead does not wrinkle, the eyebrow does not
rises; the nasolabial fold is smoothed; wider palpebral fissure, with
closing the eyes does not close (lagophthalmus), there is
lacrimation, the eyeball goes up (Bell's phenomenon). Bared mouth
is pulled over to the healthy side, the patient cannot whistle, etc., the cheek is swollen, the corner of the mouth is down, while eating solid food gets between the gum and cheek, and liquid
pours over the edge of the mouth on the affected side. Asymmetry and stiffness of the paretic
the sides intensify with mimic movements: smile, laughter, crying, conversation.
On the affected side, painful sensations, paresthesias, vasomotor
trophic disorders. In most cases with high nerve damage
hyperacusis is observed (increased perception of sound, especially low tones),

violation of taste in the front 2/3 of the tongue and dry mouth.
It is necessary to distinguish the peripheral nature of the lesion from the central one. With peripheral (extracranial) lesions, unilateral nerve palsy is noted
and the whole half of the face suffers. With central (intracranial, basal) paralysis only the lower branch is affected - from the cerebral cortex to the nerve nucleus (unilateral lesion
areas of the central section of the cortical analyzer or pathways,
accompanying, for example, hemiplegia). The nucleus or fibers of the facial nerve can to be involved in the pathological process in the brain stem (vascular, infectious, oncological), while the innervation of only the lower part of the face is disturbed and there is a paresis of the lower half of one side of the face, while the patient is in a state close your eyes and wrinkle your forehead on the affected side.
The disease begins suddenly: paresis develops within a few hours or
days. Partial restoration of nerve function occurs within the first week,
full - within 2 months in 2/3 of cases. In 1/3 of patients after 4-6 weeks,
a complicated form of the disease with contracture and an increase in the tone of some facial muscles and involuntary movements on the sore side, arising
at the moment of eating, smiling, laughing and other mimic actions.
The contracture is accompanied by unpleasant sensations of constriction and tension, aggravated by excitement, physical stress, in the cold.
With a neurological examination, already with an external examination, it turns attention to the symmetry of the face. For a more detailed check of the functions of the facial

nerve to the patient is offered to collect the folds on the forehead ("surprised"), frown ("Get angry"), close your eyes ("hard, as if soap had gotten in"), puff out your cheeks, show your teeth, smile and whistle (or "blow out a match").
Clarifying the diagnosis of neuropathy and identifying the prognosis of recovery contribute to the classical electrodiagnostics with the measurement of the excitability of the nerve branches
and facial muscles for different types of current and electromyography.
It is necessary to carefully examine the ear, the paranasal cavities; conduct epidemiological analysis, since the disease often occurs due to infections.

VIII pair - the auditory, or vestibular cochlear nerve

This pair of nerves unites two functionally different sensory
nerve, one of which - the cochlear (auditory) - provides the perception of sounds, and the other
- vestibular - regulation of balance and orientation of the head and body in space.
Thus, the nerve has two branches: the truly auditory and vestibular parts of the nerve.
Violation of the cochlear (auditory) part leads to hearing disorders: hypacusia (hearing loss), anacusia (deafness - hearing loss), or hyperacusis (increased perception of sounds).
With the pathology of the vestibular part of the nerve, vestibular ataxia occurs: upset the balance of the body (tilts and falls in the direction of the defeat) and coordination

movements, dizziness, vomiting and nystagmus (involuntary rhythmic
twitching of the eyeballs, especially when they are abducted, in a horizontal, vertical or rotational plane).
Neuropathy of the VIII nerve occurs due to intoxication with various substances (for example, a number of antibiotics), as a complication of influenza infection, skull injuries,
in the vascular process in the vertebrobasilar basin and neurinoma of the nervous trunk.
The function of the auditory nerve is investigated by otolaryngologists, but in neurology, with
necessary, check the whispered speech (on both sides alternately) at a distance of 6 meters. In this case, words are called that contain loud consonants ("forty
six ”,“ artillery ”, etc.). If the patient cannot hear from 6 meters, then the distance is needed
decrease until the patient hears the words called.
The presence of nystagmus is determined by examining the movements of the eyeballs: when they
abduction to the sides (horizontal nystagmus) and up (vertical nystagmus).
With irritation of the temporal lobe cortex, auditory hallucinations occur, up to to difficult (patients hear music, various voices).

IX pair - glossopharyngeal nerve

Anatomy and physiology:
It is a mixed nerve, mostly sensory. His motor cells
are located in the "combined" core (shared with the X pair), which is located in medulla oblongata. The axons of these cells leave the cranial cavity through the jugular
hole and fit to the stylopharyngeal muscle. Central motor neurons
located in the lower parts of the precentral gyrus, their axons are part of pyramidal pathway and end at the motor nuclei on both sides.
The first sensory neurons are located in two jugular nodes - the superior and bottom. The dendrites of these cells branch out in the posterior 1/3 of the tongue, soft palate, pharynx,
pharynx, anterior surface of the epiglottis, auditory tube and tympanic cavity.
The axons end in the gustatory nucleus in the medulla oblongata. From the above nuclei axons move to the opposite side and join the medial

loop, in the composition of which they are sent to the optic tubercle, where the cells of the 3rd
neuron. The fibers of the third neuron end in the temporal lobe cortex, while taste impulses reach both cortical zones.
The IX pair of cranial nerves contains secretory (autonomic) fibers for
parotid gland. The salivary nucleus (shared with the X pair) is located in medulla oblongata. Parotid secretory cells receive impulses as
from the taste core and from the cortical sections of the taste analyzer (temporal lobe).

It provides innervation to the posterior third of the tongue, palate, middle ear, pharynx and
vocal cords.
Dysfunction of this nerve leads to a variety of disorders:
taste disturbance in the posterior third of the tongue or on the side of the affected nerve, anesthesia
(loss of sensitivity) of the mucous membrane of the pharynx and tongue, dysphagia (impaired swallowing) -
since the vagus nerve is also involved in the innervation of the pharynx.
For unilateral dysfunction of the parotid salivary gland, dry mouth
may be absent or insignificant, as this function is compensated
the work of the rest of the salivary glands.
Glossopharyngeal neuropathy is a rare disorder that usually presents with
neuralgia syndrome with severe paroxysms of pain lasting from several
seconds to 1-3 minutes - in the pharynx, tonsils and the posterior third of the tongue irradiating into the ear.
Pain attacks can be triggered by swallowing, hot or cold food,
coughing, laughing, talking, accidentally touching the back third of the tongue, and accompanied by bradycardia, arrhythmia, drop in blood pressure, fainting.

X pair - vagus nerve

Anatomy and physiology:
The vagus nerve has a variety of functions. It not only carries out
innervation of striated muscles in the initial section of the digestive and
respiratory apparatus, but is also a parasympathetic nerve for most
internal organs. From the point of view of neurological diagnosis, they matter
disorders of the innervation of the soft palate, pharynx, larynx.
Motor fibers start from the cells of the united nucleus. The axons of these
cells in X pairs leave the cranial cavity through the jugular foramen and
innervate the muscles of the soft palate, pharynx, larynx, epiglottis, upper part esophagus, vocal cords (recurrent nerve). Central motor neurons
are located in the lower part of the precentral gyrus, their processes pass into
the composition of the corticonuclear pathway and end in both united nuclei.
X pair contains motor fibers for smooth muscles
internal organs and secretory for the glandular tissue of internal organs. They
start at n. dorsalis n. vagi (parasympathetic nucleus).
Peripheral sensory neurons are located in two nodes - the upper and
lower, which are at the level of the jugular foramen. The dendrites of the cells of these nodes
end in the occipital pia mater, external auditory
passage, on the back of the auricle, in the soft palate, pharynx and larynx.
Some of the dendrites also reach the more distal parts of the respiratory tract, gastrointestinal tract and other internal organs. The axons of the cells of the upper and of the lower node enter the medulla oblongata and end in the taste core.
The axial-cylindrical processes of the cells of this nucleus (second neurons) pass to the opposite side and together with the medial loop are directed to the visual

the hillock where the cells of the 3rd neuron are located. Axons of the third neuron are composed of

the posterior thigh of the inner capsule to the cells of the lower part of the posterior central
convolutions.
This is the largest autonomic nerve. It provides fibers for the muscles
and the vessels of all internal organs, as well as the pharynx, soft palate, larynx and epiglottis.
With a unilateral lesion, there is a drooping of the soft palate, deviation
uvula (in the healthy side), dysphonia (hoarseness), dysphagia (violation swallowing).
With bilateral lesion, aphonia occurs (the voice becomes whispering,
soundless), choking and coughing appears when eating, liquid food falls out
through the nose. Cardiovascular and respiratory activity is impaired, as a result of which
there may be death of the patient.
The research technique of IX and X pairs of cranial nerves includes examination of the condition
soft palate: normally it is located symmetrically and when pronouncing the letter "a-a-a"
rises equally on both sides; uvula location: it is normal
along the midline. The patient is asked to drink a few sips of water or swallow saliva - swallowing should be free, without choking.

XI pair - accessory nerve

Anatomy and physiology:
The accessory nerve is a purely motor nerve. Body peripheral
motor neurons are located at the base of the anterior horns I – YI of the cervical segments. The axons of these cells go out to the lateral surface of the spinal cord,
rise up and through the large opening of the occipital bone enter the cavity
skull. In the cranial cavity, the fibers of the XI pair attach to themselves sensitive fibers from the vagus nerve (the cerebral part of this nerve) and leave the cavity
skull through the jugular foramen, after which they are divided into two branches: external and
internal.
The inner one joins the vagus nerve, and the outer branch innervates
sternocleidomastoid and trapezius muscles.
Central neurons are located in the middle of the corticonuclear bundle,
make a partial crossing and go down to the cells of the XI pair nucleus.
It innervates the sternocleidomastoid muscle (head turns in
sides - opposite to the nerve) and the trapezius muscle (movements of the scapula and collarbones when they are raised and pulled back).
Damage to the nerve leads to paresis or paralysis of these muscles, which is expressed in their
atrophy: it is difficult to turn the head in a healthy direction (the formation of

torticollis), shrug of the shoulders, abduction of the shoulder blades to the spine, is limited
raising hands above the horizontal line.
In the study, the patient should normally perform all these movements without difficulties.
Occasionally there are muscle cramps innervated by the XI nerve; they are more often unilateral
or
are
the result
cortical
or
subcortical
irritations. Tonic spasm gives a picture of "torticollis"; clonic - jerking
head in the opposite direction, sometimes with simultaneous lifting of the shoulder.
Bilateral clonic spasm leads to head nodding
(Salaam's convulsion).

XII pair - hypoglossal nerve
Anatomy and physiology:
Motor nerve. The nucleus of the XII pair is located in the medulla oblongata.
The axons of the cells of this nucleus merge into a common trunk, which leaves the skull through
the canal of the hyoid nerve of the occipital bone. Innervates the muscles of the tongue. Central
motor neurons are located in the lower part of the precentral gyrus. Axons
these neurons pass as part of the corticonuclear bundle through the knee of the internal
capsules, legs of the brain, bridge and at the level of the medulla oblongata pass to
the opposite side to the nucleus of the hypoglossal nerve, i.e. exercising, full
cross.
This is the motor nerve of the tongue, which provides articulation when speaking and promoting food while eating for swallowing.
The defeat of this nerve leads to paresis or paralysis of the corresponding
half of the tongue and is accompanied by atrophy and thinning of the muscles of the tongue, deviation of it
in the direction of defeat and dysarthria (speech impairment: it becomes intertwined and
indistinct - "porridge in the mouth"). To test the function of the hypoglossal nerve to the patient
offer to stick out your tongue. Normally, it should be located in the midline.
Bulbar paralysis.
A characteristic feature of the topography of the brain stem is the congestion on a small space of nuclei IX, X, XII pairs of cranial nerves. Unilateral defeat of IX, X and XII pairs nerves (glossopharyngeal, vagus and hypoglossal) leads to the so-called bulbar paralysis, which is characterized by the presence of "three D":

1. Dysphonia (nasal, nasal tone of voice or hoarseness);

2. Dysarthria (disorder of articulation - slurred speech, characterized by difficulty in the pronunciation of some letters - "l", "s", "b", "p");

3. Dysphagia (disorder of chewing and swallowing - choking on food, ingestion of liquid food into the nose).

Atrophy of the muscles of the tongue with fibrillar twitching, paresis of the soft palate is noted. Sensitivity is not upset. Facial (VII pair) and trigeminal (V pair) usually also suffer. nerves, as a result of which the patient's face is amimic, the mouth is open, saliva flows out of it.

Pseudobulbar paralysis.

With damage to the central motor neurons IX, X, XII pairs of cranial nerves with pseudobulbar paralysis develops on both sides, which is clinically manifested as follows symptoms: ("three A"):

1. Aphonia (almost silent, whispering speech);

2. Anartria (serious disorder of articulation up to the inability to speak);

3. Afagia (serious disorder of swallowing up to the impossibility of performing the act of swallowing).

Reflexes of oral automatism appear (proboscis, Marinescu-Radovici, etc.)

As an independent disease, bulbar palsy is rare, usually it joins amyotrophic lateral sclerosis and syringomyelia when a painful process also affects the bulbar part of the brain. This must be borne in mind when recognizing, as well as tumors of the medulla oblongata, syphilis, pseudobulbar paralysis due to bilateral lesions of the cortical pathways in the brain. Infectious diseases of the brain, poliomyelitis and encephalitis, also accompanied by bulbar paralytic disorders, always develop very sharply.

HIGHER BRAIN DISORDERS

For neurological diagnostics, it is important to study the characteristics of higher nervous activity, behavior and psyche of the patient. Higher cerebral functions include gnosis, praxis, speech, memory and thinking, consciousness, etc.

A person's ability to speak and think is provided primarily by exceptional development of the cerebral cortex, the mass of which is about 78% of the total mass of the brain.

Neurophysiological studies have convincingly shown that the activity of the cerebral cortex is entirely dependent on from the activity of the structures of the brain stem and subcortical formations. The cerebral cortex can function normally only in close interaction with subcortical formations. V in recent years, ideas about the vertical hierarchical organization of functions have been developing nervous system, about the annular cortical-subcortical connections. This gives rise to the old term “Higher cortical functions” to prefer the more expedient - “higher brain functions”.

To assess modern ideas about the localization of functions in the cerebral cortex it is necessary to briefly consider the data of morphology, physiology, neuropsychology and clinic of

ganic lesions of the brain.
Kiev anatomist V.A. Betz (1874) first drew attention to the differences in the fine structure (architectonics) cortical fields. The main type of structure of the cortex (with the exception of its most ancient sections,
included in the limbic region) - six-layer. It includes the following layers: molecular layer, outer granular layer, layer of small and medium pyramidal cells, inner granular layer, layer large pyramidal cells, a layer of polymorphic cells. The severity of these layers in different departments
the cortex is not the same, the structure of myelin fibers also varies
(myeloarchitectonics). V.A.Bets described
11 fields with difference in histological structure. Later, K.
Brodmann (1909); O. Vogt, C. Vogt (1922); K. Economo, G. Koskinas (1925) and others.
Cytoarchitectonic features of the structure of various parts of the cerebral cortex
depend on the thickness of the cortex, the width of its individual layers, the size of the cells, the density of their location in
different layers, the severity of horizontal and vertical striation, separation of individual layers on sublayers and other specific features of the structure of a given field. These features are at the heart of
division of the cerebral cortex into regions, subregions, fields and subfields. The area is allocated to
based on more pronounced stable important signs. Fields are highlighted by less distinct less stable, in comparison with the areas, signs. An evolutionary approach has made it possible to create
modern classification of cerebral cortex fields.
The frontal region occupies 23.5% of the surface of the cortex, it includes fields 8-12, 44-47 and 32. This
the area is one of the most complex in its cytoarchitectonics and is characterized by significant thickness of the bark, the severity of layers II and IV, thick layer III, which is divided into
3 sub-layers, a relatively thick layer V, which can be divided into 2 sub-layers. For the frontal lobe
characterized by a gradual transition from one field to another and a large length of transition zones between
fields. Fields of the frontal area are highly individualized. This area is connected
with the highest, most complex associative and integrative functions, it plays an important role in higher nervous mental activity and the organization of the second signaling system.
The precentral area occupies 9.3% of the surface of the cortex, includes fields 4 and 6. It characterized by agranularity, i.e., the absence of layer IV and very weak development of layer II, the presence
very large pyramidal cells in layer V (giant pyramidal neurons and located in
field 4), relatively large thickness of the cortex, poverty in small cells, quite pronounced radial striation with weakly expressed horizontal striation. Precentral
the area is the core of the motor zone; its fields are directly related to the implementation of arbitrary
movements. The postcentral area occupies 5.4% of the surface of the cortex and includes fields 3/4, 3, 1, 2,
43. It is characterized by a relatively small thickness of the bark, a sharp severity of layers II and IV,
a large number of small cells in all layers, clarified by the V layer, functionally this area is associated with the reception of various types of sensitivity, and the areas of perception of stimuli with

different parts of the body are built according to the somatotopic type. Damage to this area leads to
anesthesia of the opposite half of the body.
In the parietal lobe, 2 areas are distinguished: the upper parietal and lower parietal regions, which
separated by an inter-parietal sulcus.
The inferior parietal region occupies 7.7% of the surface of the cerebral cortex. It includes in fields 39 and 40, characterized by a large thickness of the cortex, dense cells, radial striation passing through all layers. the severity of the II and IV layers, a gradual transition lower (V, VI, VII) layers into each other, a fuzzy boundary between the bark and the white matter. This area

relates to complex associative, higher integrative and analytical functions. At
its damage disrupts reading, writing, some complex forms of movement (apraxia), etc.
The superior parietal region occupies 8.4% of the surface of the cerebral cortex. It includes in itself fields 5 and 7. It is characterized by horizontal striation, average thickness of the bark, well-defined
dividing layers II and IV, large pyramidal cells of layers III and V, clarified layer V.
Radial striation is revealed only in the upper layers of the cortex. This area is also relevant to the most complex integrative and associative functions. It carries out analysis and synthesis information entering the brain through the skin-motor, visual, auditory and others analyzers. When this area is damaged, the sensations of localization of the limb are disturbed, the direction
her movement, etc.
The occipital region occupies 12% of the surface of the cerebral cortex. It includes fields 17, 18 and 19. Characterized by dense cells, impossibility of differentiation of II and III layers,
with an enlightened V layer, a columnar grouping of cells on the lower floor, a sharp border between
bark and white matter. Functionally, the occipital region is associated with vision.
The temporal region occupies 23.5% of the surface of the cortex. It includes the following subareas:
upper (fields 41, 42, 41/42, 22, 52, 22/38), which is characterized by a large number of small cells
in all layers, enlightenment of layer V, the presence in it of a small amount of large pyramidal cells;
middle (fields 21 and 21/38), which is transitional between the upper and basal subareas, but it also has some peculiar structural features: an uneven border line between layers I and II, wide V layer, small groups of large cells in layers III and IV, etc .;
basal (20-b, 20-s, 20-1, 20/38 fields), in which there is a fusion into a single complex of layers V-
VII, dense cells, narrow layer IV, horizontal striation in the upper layers, vertical striation - in the lower;
temporo-parietal-occipital (37-a, 37-b, 37, 37ab, 37aa), which is characterized by a clear discharge 7
layers, denseness and severity of layers II and IV, fuzzy border between layers III and IV, unsharp
radial striation. The temporal region is related to the auditory analyzer.
The insular area occupies 1.8% of the crustal surface. It includes fields 13 and 14 and peripaleocortical fields. This area is characterized by a relatively large width,
dense, wide IV layer, pronounced horizontal striation. Ostrovova fields

areas are associated with the function of speech. The peripaleocortical fields are associated with the synthesis of olfactory and
taste sensations.
The limbic region occupies 4% of the surface of the cortex. It includes 23, 23/24, 24, 25 and peritectal fields. Peritectal fields are located between the actual limbic fields,
tape and support in the hippocampal gyrus (taenia tecta and subiculum). Cytoarchitectonic the characterization of the limbic region as a whole seems to be very complex, and the characteristic features
for all of its fields are missing. The limbic area is associated with autonomic functions.
The ancient cortex (paleocortex) includes the olfactory tubercle, a diagonal region, transparent septum, periamygdala area and prepiriform area. Characteristic of
the ancient crust is weakly demarcated from the underlying subcortical formations.
The old cortex (archicortex) includes the hippocampus, subiculum, dentate fascia, and taenia tecta.
The old crust differs from the ancient crust in that it is clearly separated from the subcrustal formations. Old
and the ancient crust does not have a six-layer structure. They are presented as three-layer or single-layer
structures that occupy 4.4% of the cortex of the human cerebral hemispheres.
The cells of the cerebral cortex are less specialized than the cells
nuclei of subcortical formations. This increases the compensatory capabilities of the cortex, since the functions
the affected cells can be taken over by other neurons. Lack of narrow specialization of cortical neurons creates conditions for the emergence of a wide variety of interneuronal connections, formation of complex ensembles of neurons to perform various functions. However,
despite the known nonspecificity of cortical neurons, certain groups of them anatomically and functionally more closely related to certain specialized departments of the nervous
systems. The existing morphological and functional ambiguity of areas of the cortex allows talk about the cortical centers of vision, hearing, smell, etc., which have a certain localization.
In the history of the study of the localization of functions in the cerebral cortex for many years there were 2 tendencies: the desire to emphasize the equivalence (equipotentiality) of cortical fields due to its high plasticity and the concept of narrow localization psychomorphologism, i.e. That is, an attempt to localize even the most complex mental functions in limited cortical centers.

Outstanding research by I.P. Pavlov and his school on the dynamic localization of functions and the further development of these ideas by P.K.Alekhin (1971), N.A. Bernstein (1966) and others led to
the emergence of new ideas in this area.
The cerebral center or cortical section of the analyzer, according to I.P. Pavlov, consists of a "nucleus" and
"Scattered elements" (1936). The nucleus is relatively homogeneous in morphological plan a group of cells with an accurate projection of the receptor fields. "Scattered elements" are near
nucleus or at a distance from it and carry out elementary and undifferentiated analysis and synthesis
incoming information.
In the cortical representations of the analyzers, even vertically, 2 groups of cellular zones. Of the 6 layers of cortical cells, the lower layers have connections with peripheral receptors (layer IV) and with
muscles (V layer). They are called primary or projection cortical zones due to their

direct communication with the peripheral parts of the analyzer. The upper layers of the cerebral hemispheres
the human cerebral cortex is the most developed expression; they are dominated by associative connections with others
sections of the cortex, and they are called secondary zones (II and III layers), or projection-associative,
zones.
Such a structure is found in the occipital cortex, where the visual pathways are projected, in temporal - where the auditory paths end, in the postcentral gyrus - the cortical region sensitive analyzer, etc. Morphological heterogeneity of primary and secondary zones accompanied by physiological differences. Experiments with stimulation of the cerebral cortex have shown
that the excitation of the primary zones of the sensory divisions leads to the emergence of elementary
sensations. For example, irritation of the primary zone of the occipital lobe causes the appearance of photopsies, and
the same irritation of the secondary zones is accompanied by more complex visual phenomena - the subject sees people, animals, various objects. Therefore, it is assumed that it is in
in the secondary zones, operations of recognition (gnosis) and, in part, action (praxis) are carried out.
On the horizontal plane in the cortex, tertiary zones are also distinguished, or zones of overlap of cortical
representative offices of individual analyzers. In the human brain, they occupy a very significant place and are located primarily in the temporoparieto-occipital and frontal zones. Tertiary zones enter into extensive connections with cortical analyzers and provide the production of complex
integrative reactions, among which the first place in a person is occupied by meaningful actions (planning and control operations), requiring the complex participation of various parts of the brain.
Functionally, several integrative levels of the cortical
activities.
The first signaling system is associated with the activity of individual analyzers and carries out the primary stages of gnosis and praxis (integration of signals coming from the outside world along the
cameras of individual analyzers, the formation of response actions taking into account the state of the external and
internal environment as well as past experience).
The second signaling system is a more complex functional level of cortical activity; she integrates systems of various analyzers, making it possible to meaningfully perceive the environment
the world and a conscious attitude towards it. This level of integration is closely related to speech activities - the understanding of speech (speech gnosis) and the use of speech (speech praxis).
The highest level of integration is formed in a person during his social development and as a result
learning process - mastering skills and knowledge. This stage of cortical activity provides purposefulness of certain acts, creating conditions for their best implementation.
Complex brain activity could not be carried out without the participation of the storage system information. Therefore, the memory mechanism is one of the most important components of this activity. In fur
memory systems are essential as a function of fixing information (memorization) and
the function of obtaining the necessary information (memory), and the function of moving flows

information from blocks of random access memory to blocks of long-term memory and vice versa. This
dynamism allows you to learn new things.
In recent years, the very concept of "function" has undergone a change as applied to processes, happening in the brain. Currently, a function is understood as a complex adaptive de-
the activity of the body, aimed at the implementation of any physiological or psychological task. This adaptive activity can be carried out by different
ways; it is important that the result corresponds to the task set for the body. Complexity, multi-storey structure of such functional systems, their interchangeability
individual links indicate that they can only be provided by a complex of joint
working zones, each of which contributes to their implementation. Local defeat

a certain part of such a system is accompanied by the appearance of certain clinical symptoms volumes that reflect the violation of some aspect of the activity of a complex functional system.
It should be emphasized that the localization of a symptom of a lesion and the localization of function are far from the same
and also. Functions such as, for example, speech, are associated with the work not only of the cortex, but also of many departments.
brain (subcortical, stem), so they cannot be localized in narrow cortical “centers”.
So, according to modern concepts, the higher brain (mental) functions are
functional system with a complex hierarchical structure, they are conditioned reflex in their mechanism, have a socio-historical origin and develop in each individual already
after birth and only in a social environment, under the influence of the level of civilization of a given
society, including linguistic culture.
French anatomist R. Vgosa in 1861 in two patients who suffered from a disorder during their lifetime
predominantly own (internal) speech, found focal lesions of the left hemisphere
the brain, which included the lower part of the third frontal gyrus. This section of the crust (Broca's zone) of steel
considered as the center of articulated speech, and speech disorders with focal lesions of this zone
began to be called, at the suggestion of A. Trousseau, aphasia (from the Greek. a - negation, phasis - speech).
A decade later, the work of the German neuropathologist K. Wemike appeared. Based
ideas about the predominantly sensory function of the posterior parts of the brain and the motor function of its
anterior divisions, he supported the concept of the center of Brock's articulated speech and contrasted
to him in the first temporal gyrus (Wernicke's zone) the auditory center of speech, where, in his opinion, are stored
auditory sound patterns. Accordingly, two centers of speech (motor and sensory) are distinguished by 2
the main forms of aphasia are motor and sensory.
It has now been proven that disorders of human speech associated with the breakdown of language
generalizations cannot be qualified only as motor and sensory.
80-90s of the XIX century. were marked by the flowering of narrow localizationism, when they began to describe
more and more new areas of the cerebral cortex, supposedly responsible for one or another mental function:

naming center, counting center, dictation center and spontaneous writing center, reading center, etc.
With focal lesions of these centers, syndromes of amnestic aphasia, acalculia, various agnosias, alexia, etc. One of the most famous classifications of aphasias of that time was classification W. Wernicke-Lichtheime. The latter absorbed the well-known by that time empirical knowledge and embodied, albeit in a very schematic form, the idea of a hierarchical organization of the functioning of the brain.

SPEECH AND ITS DISORDERS

Speech is a specific human form of activity that serves communication between people. She characterized by the processes of receiving, processing, storing and transmitting information using language, which is a differentiated system of codes denoting objects and their relations sheniya.

Human speech is realized with the help of breathing apparatus, chewing, swallowing, voice formation and articulation. The central link of the speech apparatus is the cerebral cortex - predominantly dominant hemisphere.

The entire set of physiological mechanisms involved in the formation of speech can be divided into **2 groups** - mechanisms of perception and mechanisms of speech reproduction.

There are 2 main types of speech - impressive and expressive.

Impressive speech - comprehension of speaking and writing (reading). Into the psychological the structure of impressive speech includes the stage of primary perception of a speech message, the stage of decoding message analysis (analysis of the sound or letter composition of speech) and the stage of correlating the message with certain semantic categories of the past or one's own understanding of the oral (written) communication.

Expressive speech - the process of speaking in the form of active oral speech or independent letters. Expressive speech begins with the motive and intention of the statement, then the stage follows internal speech (the idea of an utterance is encoded into speech schemes) and ends with an expanded speech utterance.

Thus, there are such aspects of speech activity, interconnected with each other, as perception, recognition of verbal signals, central semantic processing of the perceived messages and processes that induce speech utterance. In the implementation of speech activity both hemispheres of the brain take part, however, different parts of the cortex play in this process has a different role.

The process of perception and recognition of sound verbal signals is carried out with the presenter

the participation of the secondary cortical fields of the auditory analyzer, mainly of the left (dominant) hemispheres of the brain. Sound analysis and synthesis of speech signals are carried out here, recognition of the phonemic composition of speech is provided. Non-phonemic parameters of sounds, such as duration, volume, timbre, melody, etc., are analyzed mainly in the right hemispheres of the brain. Thus, the speech-auditory analyzer is located in the temporal lobes and left and right hemispheres of the brain. Visual verbal signals are perceived and recognized in the cortical fields of the visual analyzer of the occipital lobe; are carried out here spatial visual analysis and synthesis of letters (graphemes). In recognizing the tactile images of words (in

deaf-blind-mute) the central role is played by the secondary zones of the cortex of the skin-kinesthetic analyzer
in the parietal lobes of the brain.
The process of semantic processing of the perceived message (understanding the meaning of words,
semantic processing of information, various speech intelligent operations)
are provided by the complex integrative activity of various parts of the cerebral cortex.
Posterior tertiary associative complex of the fields of the cerebral cortex (mainly of the left)
- temporo-parietal-occipital region - associated with the analysis and synthesis of information obtained during
verbal communication in the form of counting, spatial, logical-grammatical and visual-figurative intellectual operations requiring simultaneous mental operation with one or
several characters or images. Anterior prefrontal associative complex tertiary
cortical fields is associated mainly with the programming of verbal intellectual operations and monitoring their implementation.
The processes of generating a speech utterance at the design stage are due to the integration excitations of various fields of the cerebral hemispheres, but above all - prefrontal tertiary fields of the left hemisphere. The implementation of speech utterance (oral active speech) is carried out
predominantly with the participation of the premotor and postcentral parts of the left hemisphere cortex,
responsible for the efferent and afferent coordination of the motor speech act. Organization independent written speech, in addition to the cortical zones listed above, includes secondary cortical fields of the auditory analyzer, necessary for the analysis of the sound composition of the word; secondary
cortical fields of the visual analyzer, necessary for the analysis of written letters; motor, premotor and postcentral zones of the left hemisphere cortex, in which motor and
sensitive projections of the right upper limb performing the act of writing. At different stages mastery of writing and in various forms of written speech (independent writing, writing under dictation, copying of text, etc.), the neuronal organization of writing is different.
In clinical practice, various forms of aphasia, dysarthria, alalia, mutism and general underdevelopment of speech.
Under **aphasia** understand the central disturbance already formed speech, t. E. Breakdown speech, in which the ability to use words for
expressing thoughts and communicating with others while maintaining the function of the articulatory apparatus and
hearing sufficient for the perception of elementary speech sounds. Based on the main types of speech,
2 types of aphasias are distinguished **:** sensory (receptive, impressive) - lack of understanding of the speech of others -
and motor (expressive) - violation of the expression of active oral speech.
Neurological examination of speech function reveals various variants of these
the main types of aphasias, depending on the predominant localization of the focus of brain damage
sections of the functional speech system. These options are: **motor aphasia (aphasia Broca)** - characterized by a violation of all components of expressive speech; spontaneous speech is not
possible. The patient pronounces only single words or syllables that have been preserved in his memory, repeating them
(speech embolus). Comprehension of individual words, short phrases and tasks given in writing, saved. The patient may notice mistakes in incorrectly constructed phrases. Pronunciation

preserved in the patient's word-embolus, it is sounded with intonation and melody adequate to that
what he wants to express. This is accompanied by expressive facial expressions and gestures.
Motor aphasia is observed when the posterior parts of the inferior frontal gyrus are affected (zone
Broca) of the left hemisphere. With incomplete destruction of this zone, speech is possible, but it is poorly understood,
slow, with the search for the right words, devoid of expressiveness, the spoken words are distorted,
paraphasias are noted literal (permutation of syllables) and verbal (replacement, permutation of words),
the correct grammatical structure of phrases is broken, there are no declensions and conjugations (agrammatism).
According to A.R. Luria, motor aphasia occurs in two variants: motor afferent aphasia, with which all types of oral speech are lost - spontaneous, automated, repetition of the
my words, the name of the displayed items. The articulation of sounds is especially grossly disturbed,
similar in place of education (for example, front-lingual: d. t, l, and) or by the way of education (for example, slotted: w, e, sch, x). Reading and writing also suffer. This variant of aphasia occurs
usually with damage to the parietal lobe cortex adjacent to the x postcentral gyrus,
the kinesthetic basis of the movements of the articulatory apparatus (strength, volume and direction
movements of the muscles involved in articulation). Often this aphasia is combined with oral apraxia.

(disorder of complex movements of the lips and tongue). The kinesthetic program of speech movements is disrupted
zheniy; motor efferent aphasia is characterized by a disorder of switching from one speech units (sound, word) to another. The articulation of individual sounds is preserved, pronunciation is difficult
a series of sounds or phrases. Productive speech is replaced by the constant repetition of individual sounds
(literal perseveration) or words (verbal perseveration), and in severe cases presented
speech embolus - the only sound or word that the patient utters when trying to
either say.
Another distinctive feature of speech in efferent motor aphasia is “telegraph
style ”: the patient makes phrases mainly from nouns, verbs are almost absent in them.
Automated speech, poetry reading, singing are saved. Disrupted reading, writing and titles items. This variant of aphasia occurs when the lower parts of the premotorium of the left cortex are affected.
hemispheres of the brain.
Sensory aphasia (Wernicke) is characterized by impaired understanding of speech as others persons, and his own, that is, the auditory gnosis is disturbed. Elementary perception of hearing in a patient with
stored. and the phonemic is broken. A phoneme means a semantic and distinctive feature language. In Russian, these features include voicedness and voicelessness of consonants (b, p, d, s, s),
the stress and unstressed syllables (flour, flour), the hardness and softness of the endings (chalk, stranded). In others

languages, semantic and distinctive features may be different (for example, the longitude of sounds in
English, etc.). The patient perceives speech as noise or conversation unknown to him language. Due to the lack of auditory control, expressive speech is secondarily disturbed. Sick speaks a lot and quickly (logorea - speech incontinence), his speech is incomprehensible to others,
there is a lot of paraphasia (distortion, inaccurate use of words). Sometimes speech is a stream of meaningless, inarticulate sound combinations ("verbal okroshka") and abundant literature
ral and verbal paraphasias, distortions of words that are similar in sound or meaning. Frequent repetitions of the same words or syllables are observed (perseveration). Your speech defect
patients are usually unaware. Repetition of the proposed syllables (such as "ba-pa", "ta-da", "sa-za"), words,
reading and writing are also impaired. Even with partial sensory aphasia, the patient does not perceive
differences in the spelling of the words "fence", "cathedral", "constipation", etc., confuses the letters "s" and "z", "p" and
“B”. Sensory aphasia occurs when the cortex of the left temporal lobe (middle and posterior superior temporal gyrus - Wernicke's zone or field 22 according to Brodmann).
With the defeat of the lower and posterior parts of the parietal and temporal regions, development of
amnestic aphasia. It is characterized by forgetting the name of objects, names. The sick person cannot
name the subject, although it defines its purpose well. For example, if you show a pen to a patient, then
he will say - "this is what they write." The patient immediately remembers the right word when prompted for the initial syllable.
(calling him only "ru", the patient will immediately say "pen"). Comprehension of speech is not impaired. Reading aloud
Maybe. Spontaneous writing is upset with a major defect, dictation is possible. Speech the patient with amnestic aphasia is saturated with verbs, but there are few nouns in it.
Amnestic aphasia should be distinguished from the broader concept of **amnesia** (disorder memory for previously developed ideas and concepts). Various types of amnesia are more likely to occur with
lesions of the mediobasal parts of the temporal and frontal lobes.
With the defeat of the left parietal-temporal region in right-handers, semantic aphasia occurs, with
which the understanding of the meaning of sentences, expressed with the help of complex logical grammatical structures.
Such a patient does not understand the relationship expressed with the help of prepositions (circle under a square,
triangle over the circle). The meaning of comparative constructions is not available to him (for example, hair in
Lena is darker than Olya's, but lighter than Katya's. Who has the lightest hair?), Returnable designs
(the fox ate the chicken, the chicken ate the fox), the so-called attributive constructions (“father’s brother” and “father
brother ").
Finally, there is often **total aphasia** , in which receptive and
expressive speech in all its forms. This is observed with extensive damage to the left hemisphere from Broca's zone to Wernicke's zone.

Alexia (reading and reading comprehension disorder) and agraphia (loss of ability
write correctly while maintaining the motor function of the upper limb) are usually included in
sensory and motor aphasia syndrome, and sometimes they come to the fore and are found as
would be isolated. Such "isolated" agraphia can occur with a limited focal
defeat of the posterior part of the second frontal gyrus (next to the projection of the pyramidal
pathways for the right
upper limb, and “isolated” alexia - with foci in the angular gyrus (gyrus angulans)
dominant hemisphere, at the junction of the occipital and parietal lobes.
Hemispheric lesions involving a complex speech system usually cause
violations of various aspects of this function - a complex of speech disorders, which are often
mixed
shany. Nevertheless, in most cases, it is possible to establish a predominantly motor (frontal,

partly parietal), sensory (temporal, occipital) or other form of speech disorders.
When conducting a linguistic and psychological analysis of speech functions,
variants of aphasia with the disintegration of phonemic and morphological generalizations (with
the defeat of the tertiary
zones of the cortex of the left temporal lobe), in violation of lexical and phraseological
generalizations (in case of damage
tertiary zone of the cortex of the left temporo-parietal-occipital region) and with a violation of
syntactic
generalizations (with damage to the cortex of the posterior parts of the left frontal lobe). These
subtle speech disorders
functions can be detected in patients with various variants of partial speech disorders.
Mutism is the absence of verbal communication in a patient while the speech apparatus is
intact. it
usually manifestation of reactive neurosis, hysteria, or mental illness (schizophrenia).
Alalia - systemic speech underdevelopment resulting from the defeat of cortical speech
zones under the age of 3 years. Alalia, like aphasia, is subdivided into motor and sensory. Motor
Alalia
characterized by underdevelopment of expressive speech. Sound production is disturbed, it is
difficult to build
phrases, the structure of words decreases (sounds and syllables are rearranged and
skipped); active dictionary
poor. The understanding of the addressed speech is preserved, but with special research it is
possible
determine the failure and impressive (sensory) speech. With sensory alalia, impaired
understanding of addressed speech while maintaining the perception of elementary sounds,
auditory
agnosia. In this case, there is always an underdevelopment of motor speech (mixed, total alalia),
since
impressive speech develops in children earlier than expressive speech.
For neurological diagnostics, the ability to evaluate this type is also important.
speech disorders like dysarthria. This term refers to an articulation disorder that can
be caused by central (bilateral) or peripheral muscle paralysis
speech motor apparatus, damage to the cerebellum, striopallidal system. With dysarthria, phrases
patients are correctly constructed, the vocabulary does not suffer. They don't pronounce words
clearly; especially
sounds “r”, “l”, hissing letters are difficult for articulation. Often such patients feel
that they have a kind of "porridge in the mouth." Phonetically incorrect pronunciation of certain
sounds due to

functional disorders are referred to as dyslolia. It is successfully eliminated with speech therapy classes.
In the study of speech function, speech, writing and reading are analyzed separately.
Study of the oral expressive function of speech. The patient is asked to tell his story.
diseases, the content of the pictures shown, retell the story you just listened to, etc.
The possibility of repeating the proposed words and phrases is checked (for example, "shipwreck", "earth
an earthquake "; "Firewood on the grass", "pink grapes ripen on Mount Ararat", etc.). Attention is drawn
on speech activity, a set of words (rich, limited, the presence of speech emboli, telegraph style), on the correctness of the construction of phrases, the presence of paraphasia (literal, verbal), the ability
repeat words exactly. The patient's reaction to his mistakes is taken into account (does he notice them, are there
attempts to correct), the presence of agrammatisms, perseveration, the way of pronouncing words, intonation and their
adequacy.
Automated (ordinary) speech is examined, offering to count from 1 to 10 and in the opposite direction
order, list the letters of the alphabet, days of the week, months, finish the proverb started by the doctor,
familiar song.
Study of the receptive function of speech: suggest the patient to show called by the doctor objects (they are in the patient's field of vision), the doctor asks questions: show me what the door is locked with?
what do they write? what do they sew? what is the firewood used for? etc., body parts; follow simple and complex instructions
(show your tongue, nose, close your eyes, etc.); correct incorrectly written grammar and
the meaning of the sentence; explain the meaning of metaphors ("golden hands", "iron health", "wolf
appetite ","" one is not a warrior in the field ","" a bee flies from a wax cell for tribute in the field, "etc.).
Marie's experience: the patient is given 3 sheets of paper and asked to throw one on the floor, and put the other
on the bed, return the third to the doctor.
Experience of the Year: the patient is asked to put a large coin in a small glass, and a small - in big. The experience can be complicated by placing 4 cups of different sizes and offering the patient
put a coin in a certain order in one or another glass.
Understanding complex multi-link instructions: go to the table, pick up a glass and place him to the window; when I raise my right hand, stand, when I raise my left, take a book.
Understanding of attributive constructions: brother's father; father's brother; father's father. Show on
image "daughter's mother", "mother's daughter". Show the clock with a pencil. Draw a circle under
cross. The cutlet was eaten by a boy. The fox was eaten by a chicken.
Understanding the designation of time: five minutes past seven, five minutes to eight. Show time on
dial with movable hands.
Study of the ability to reproduce oral speech. Names of displayed items.
If the patient does not name the subject, it is necessary to find out if the hint of the first syllable helps, and

also the sound of tapping on an object or feeling an object by a sick person.
Is there a tendency to pronounce old names when showing new items?
(perseveration). Is there (especially in a state of emotional excitement) pronouncing
individual phrases, exclamations, interjections. Ability to pronounce words while singing.
Reading research. Comprehension of written language and some symbolic images.
Identification of items with their names written on cards. Understanding the meaning of the written
words, numbers, phrases of varying complexity.
Reaction to misspelled words, phrases, missing letters. Execution of written
instructions (close your eyes, raise your hand, etc.). Recognizing the time by the hands on the watch dial,
recognition of drawings.
Reading aloud printed and written text, individual letters, syllables, words, phrases (short and long). Comparison of the understanding of speech and writing (with identical texts).
Study writing. Dictation letter. Copying from printed and handwritten text.
Automated letter (invite the patient to write his last name, first name and patronymic, a series of numbers,
days of the week, months, years).
Writing the names of the displayed items. Written answers to oral questions.
Written story about your illness.
An offer to draw an object, to copy a drawing.
Comparison of research results on writing and speaking.
Account research: check written account and oral, performing various
arithmetic operations, solving written and oral problems of varying complexity.
It is necessary to find out the dominant hemisphere. To identify hidden left-handedness, it is proposed
several tests: crossing the forearms on the chest, while the right forearm (in the upper one) is above; fold the hands into a lock - the thumb of the right hand will also be on top.

GNOSIS AND ITS DISORDERS

Gnosis (Greek gnosis - cognition, knowledge) is the ability to recognize objects by sensory perceptions. For example, a person not only sees, but also recognizes previously seen objects. Recognition
is a complex function of individual analyzers, it is developed in the process of individual experience (by the type of conditioned reflexes); the information received is fixed (memory function).

Agnosias (recognition disorders) develop when secondary zones are affected within some or one analyzer. However, recognition usually occurs from the complex impact of external stimuli, from the sum of sensory perceptions. A person is able to recognize objects and phenomena not
only by simple sensory influences, but also by their verbal designations (the function of the second
signal system according to I.P. Pavlov).
With agnosia, the elementary forms of sensitivity remain intact and are violated.
complex forms of analytic-synthetic activity within a given analyzer.
Visual (optical) agnosia, or the so-called mental blindness, occurs with a defeat
the outer cortex of the occipital lobes (fields 18, 19 and 39). The patient cannot recognize objects and
their realistic depiction (subject agnosia of Lissauer), perceives only their individual
signs and guesses about the general meaning of the object or its image. For example, looking at glasses,

the patient says: "a ring, and another ring, and a crossbar - probably a bicycle." Often the sick themselves
they say “I don’t know”, “I don’t see”. At the same time, they see objects, go around them and do not bump into them. Among
visual agnosia a special place is occupied by the syndrome of simultaneous agnosia. It manifests itself
inability to synthetically perceive parts of the image that form a whole.
It is customary to distinguish between 2 main forms of visual agnosia: apperceptive and associative.
With apperceptive agnosia, the patient perceives only individual signs of an object or its iso-
but cannot define it as a whole. With associative visual agnosia, the patient
clearly perceives objects as a whole and whole images, but does not recognize and cannot name them.
With less severe disorders, signs of visual agnosia are detected only in complicated
conditions, in particular when the perception of crossed out or retouched images. For example, the window cover in the drawing depicting the window is recognized and correctly named by the patient (“frame”). If
cross out this drawing with several dashed lines, then it ceases to recognize the image
window coverings.
Visual agnosia on the perception of images of letters or numbers is especially pronounced with a lesion.
secondary parts of the occipital lobe of the dominant hemisphere (left - in right-handers). On defeat
the secondary zone of the occipital-parietal region of the subdominant (right) hemisphere of the brain
visual agnosia is manifested by not recognizing persons (prozapagnosia) or ignoring perceptions in
left half of the visual field (unilateral spatial agnosia). The last ha-
characterized by the fact that the visual perception of individual objects or their images remains

intact, but the ability to assess the spatial relationship is impaired. The sick person cannot distinguish between the right and left sides, makes mistakes when determining the time by the location of the arrows
on the clock, when reading and displaying the contours of a geographic map. This type of agnosia occurs when
defeat of the tertiary zones of the parieto-occipital parts of the cerebral cortex.
A set of objects and drawings is used to study visual agnosia. By presenting them
the examinee is asked to determine, describe their appearance, compare which objects are larger, which
smaller. Using a set of pictures (color, monochromatic, outline), recognition is assessed not only subjects, but also plots. Along the way, visual memory is also checked: they present several pictures,
then mixed with previously not shown and asked to select already seen pictures.
Auditory (acoustic) agnosia (“mental deafness”) is characterized by impaired ability
recognize objects by their characteristic sounds, without seeing them, for example, falling on a tiled floor
a coin by sound, a dog by a bark, a clock by its ticking, a bunch of keys by a sound when shaken, water
- by its murmur, etc. With such a relatively rare disorder, the impression of
hearing loss of the patient, however, in fact, it is not the perception of sounds that suffers, but the understanding of their signal

values. Perhaps a violation of recognition of famous musical melodies - amusia. Auditory agnosia
appears when the secondary zones of the temporal lobe of the dominant hemisphere of the brain are affected
(transverse temporal gyri).
Sensitive agnosia is expressed in the lack of recognition of objects when they act on receptors superficial and deep sensitivity. The most common variant of it is in the form of a tactile agnosia: in a patient with a sufficiently intact fine tactile sensitivity, it is lost
the ability to recognize objects when groping with closed eyes. This phenomenon is called astereognosis. True astereognosis appears when the parietal lobe is affected (secondary zone, predominantly in the dominant hemisphere of the brain - field 40), when they remain intact elementary skin and kinesthetic sensations. Unrecognition of objects to the touch by patients with
loss of superficial and deep sensitivity in the examined hand is designated as pseudoastereognosis and occurs when sensitive conductors are damaged in any of the areas from spinal cord to the thalamus and cerebral cortex (postcentral gyrus).
The phenomenon of auto-diagnosis is closely related to astereognosis, which consists in the difficulty
determine the location of individual parts of the body. in violation of recognition of parts of his body; and
metamorphopsia, when the patient begins to perceive parts of his body or foreign objects unusual, altered in shape or size. With macropsia, objects appear to the patient.
excessively large, with micropsia - unusually small. Sometimes the phenomenon of polymelia occurs -
sensation of false limbs (third upper or lower limb), which may appear
motionless ("the extra hand lies and presses on the chest") or moving. Such options sensitive agnosia with impaired recognition of parts of one's own body occur when
parietal lobe of the right hemisphere. It is very characteristic that with a pathological focus in the subdominant
(right) hemisphere of the brain, the phenomenon of sensitive agnosia may be accompanied by a violation
perception of their own defect - anosognosis (Anton-Babinsky syndrome): the patient does not notice
self disturbances of sensitivity and paralysis (most often in the left side of the body).
Pain agnosia, spreading to the whole body, is rare. Injections at the same time
perceived as touching; the patient does not feel pain.
Olfactory and gustatory agnosia - the loss of the ability to identify smells and gustatory sensations (with damage to the mediobasal areas of the temporal lobe cortex). These types of agnosia are encountered
are rare and it is practically difficult to distinguish them from anosmia and ageusia, which are found in lesions
receptor neurons and conducting systems of the olfactory and gustatory analyzer.

PRAXIS AND ITS DISORDERS

Praxis (from the Greek praxis - action) - the ability to perform sequential complexes movements and perform targeted actions according to the developed plan. When implementing complex motor acts, the work of skeletal muscles must occur in the correct
sequence with simultaneously coordinated contractions of many muscle groups. Such actions arise in the course of vocational training.
Complex actions are formed on the basis of kinesthesia - continuously incoming information from proprioceptors when performing any movements. The visual
analyzer. In teaching and performing complex motor acts of a person, it is especially important has a speech signaling (oral and written). Therefore, praxis disorders previously associated

in total with the pathology of the kinesthetic analyzer, they also depend on the defeat of speech functions. With another
On the other hand, for the implementation of the latter, an impeccable praxis of the speech organs is necessary.
Apraxia is characterized by a loss of skills developed through individual experience, complex purposeful actions (everyday, industrial, symbolic gestures, etc.) without pronounced signs of central paresis or impaired coordination of movements.
In classical neurology, there are several main types of apraxia.

Ideatorial apraxia is caused by the loss of a plan or design for complex actions, while the sequence of individual movements is disrupted (for example, when asked to show how a cigarette, the patient strikes this cigarette on the box, then takes out a match and holds it out to his mouth).
Patients cannot perform a number of verbal tasks, especially symbolic gestures (shaking a finger, give a military salute, etc.), but they are able to repeat, imitate the actions of the investigator.
Ideatorial apraxia occurs when the supramarginal gyrus (gyrus supramarginalis) is affected by the parietal
lobes of the dominant hemisphere (in right-handers - left) and always bilateral.
With constructive apraxia, the correct direction of action suffers first of all; sick
it is difficult to construct a whole from parts, for example, to fold a given geometric figure from matches
(rhombus, square, triangle). With this type of apraxia, lesions are more often found in the angular gyrus
(gyrus angularis) of the parietal lobe of the dominant hemisphere. Apraxic disorders also bilateral.
Motor apraxia, or apraxia of Dejerine's performance, is characterized by a violation not only spontaneous actions and actions on assignment, but also on imitation. It is often one-sided (for example,
with damage to the corpus callosum, it can occur only in the left upper limb).
The defeat of the parietal lobe near the postcentral gyrus causes apraxia due to violation of kinesthesia (afferent, or kinesthetic, apraxia). Arbitrary movement of the patient can only be produced with constant visual control.
With foci at the junction of the parietal, temporal and occipital lobes (zone of statokinesthetic analyzer), violations of spatial relationships occur when performing complex
motor acts (spatial apraxia). The patient cannot give a straightened hand
horizontal, frontal or sagittal position, draw an image oriented in
space, get to the desired point. Makes spatial errors when writing, being not in
able to correctly correlate parts of complex letters and showing signs of a mirror
letters.
With the defeat of the lower sections of the postcentral gyrus of the dominant hemisphere (right-handed
- left), oral apraxia develops, usually combined with motor afferent aphasia. Sick
cannot find the positions of the vocal apparatus necessary for pronouncing the corresponding sounds,
sounds close in articulation are mixed, writing is broken.
With damage to the frontal lobe (zone of efferent systems), the skills of complex
movements and action programs with impaired spontaneity and purposefulness (frontal apraxia).
The patient is prone to echopraxia (repeats the movements of the examiner) or inert stereotyped movements that he does not correct and does not notice. Such patients find it difficult to perform non-
typical programmed actions: for example, when the doctor asks to raise its upper

the patient's limb must raise the II finger, or in response to one knock, raise the right, and in response to two
knock to raise the left upper or lower limb, etc.
For the occurrence of apraxic, as well as aphatic, disorders of great importance is violation of connections between the cortical parts of the analyzers (especially kinesthetic) and executive motor systems. Therefore, apraxic and aphatic disorders
occur with lesions not only of the cortical, but also of the deep, subcortical parts of the brain, where more
the fibers of these paths are compactly located.
The study of gnosis and praxis is carried out with the help of special tasks. Offered objects surrounding the patient, and their recognition by sensory perceptions is checked (visual, auditory, tactile, gustatory, olfactory).
Checking the gnosia of one's own body (identifying astereognosis, auto-diagnosis, digital agnosia,
pseudomelia and other disorders of the body scheme).
The ability to perform simple actions: close your eyes, stick out your tongue, give your tongue the position of the "tube", put it between the teeth and the lower lip, whistle; spread your fingers form a ring with your fingers.
Actions with real objects: light a match, salt bread, pour water into a glass,
comb your hair with a comb.
Actions with imaginary objects: show how to catch flies, hammer a nail, drink from glasses, eat soup, etc.
Gestures: how to shake a finger, how to give a military salute, how to send an air kiss and other Imitation of the actions of the doctor.
Transitive actions: point the index finger of one hand or the other to the right or left eye, etc.
The study of the ability to perform simple arithmetic operations in the mind and in writing.
Performing tasks for the construction of figures (folding matches, cubes).
The study of speech, gnostic and practical functions requires patience from the patient and doctor. When the patient is tired, it is necessary to take breaks for rest.

MEMORY AND ITS DISORDERS

Memory mechanisms as one of the most important components of higher nervous and mental activity not yet fully disclosed. Data on the important role of ribonucleic acid (RNA) and oligopeptides have been obtained
in the implementation of the memory function, the formation of circular neuron-glial structures (in which pulses can circulate for a long time, thereby providing storage of information), etc. In the system of each the analyzer is fixing information, so we can talk about visual memory, auditory,
tactile, etc. In case of irritation of the occipital-parietal-temporal region during neurosurgical operations under local anesthesia, the patients had sensations characterized by the recollection of any events, persons, objects, sounds. At the same time, memory as a mental process is associated with the work of a holistic
brain, therefore, we can only talk about memory centers conditionally. Experimental and clinical studies have shown that brain structures such as
hippocampus, cingulate gyrus, anterior nuclei of the thalamus, mammillary bodies, septa, vault, amygdala lex, hypothalamus, which make up the large and small circle of Papets.
Memory function is influenced by emotions, attention, degree of interest,
purposefulness. Distinguish between mechanical memory (more elementary, visual-figurative) and semantic (more complex, abstract).
Memory disorders are very diverse and occur not only with organic brain damage.
They are observed with overwork, neurosis, intoxication, etc. Isolated violations are possible memorization with a fairly confident possession of previously accumulated information or, conversely, a decrease
memory efficiency, that is, the inability to remember quickly with good memorization.

Memory impairment with the loss of the ability to preserve and reproduce the acquired knowledge is indicated amnesia (Greek amnesia - forgetfulness, memory loss). There are several types of amnesia.
Fixation amnesia - weakening or lack of memorization of current, recent events
events while retaining the knowledge acquired in the past. Such memory disorders especially
expressed in alcoholic encephalopathy (Korsakov's amnestic syndrome), as well as in atherosclerosis
cerebral vessels, some intoxications (for example, with carbon monoxide poisoning). When expressing
female violation of memorization of new facts and circumstances develops amnestic disorientation in
environment and people around, in time and sequence of events.
Progressive amnesia is the gradual depletion of the stocks of acquired information and knowledge.
A decrease in memory develops in a certain sequence: from more specific elements to more general ones, from later acquired information to earlier; it is more firmly anchored, more organized and
automation; from less emotionally saturated to more emotionally significant (Ribot's law).
In case of traumatic brain injury with switching off of consciousness in the subsequent, upon restoration of consciousness,
loss of memory (full or partial) for a certain period of time is often noted. Complete
loss of memories can be limited only by a period of impaired consciousness (congrade amnesia)
or extends to events preceding the state of altered consciousness - from several hours,
days to months or even years (retrograde amnesia). If you lose memories of events, experiences,
which were at the end of the disorder of consciousness, this indicates anterograde amnesia. Combination
the last two options for amnesia, i.e. loss of memory for previous trauma (illness) and
subsequent events are called anteroretrograde amnesia.
There is also post-hypnotic amnesia (gaps in memory for events that occurred during
hypnosis), katatimny amnesia (loss of memory of psychogenic unpleasant, affectively saturated
impressions and events).
Much more common is not loss, but memory loss - hypomnesia. Rarely observed
hypermnesia - unusually good (phenomenal) memory. Hypermnesia usually refers to mechanical
memory.
Patients with memory loss often have a symptom of false memories -
pseudo-reminiscences, in which real events of the past are transferred by patients to the present. If
false memories of patients are fantastic, unrealistic, patients talk about events,
which in reality could not occur, then this is evidence of confabulations.
The sensations of “already seen” (deja vu), “already heard” are peculiar memory impairments.
(deja entendu), when a person suddenly begins to seem that the whole situational situation was once exactly him
worried. Sometimes the opposite phenomenon is observed - “never seen” (jamais vu), “never
heard ”(jamais entendu), when a familiar phenomenon is felt as if for the first time in life. These
phenomena occur when the temporal lobe is irritated (tumor, cicatricial adhesive process, etc.).
A number of psychological techniques are used to study memory. Find out the state of the mechanical memory is possible with a test with memorization of 10 words: the subject is slowly called 10 words and asked for them
repeat; the order in which the words are repeated does not matter. Then the same words are repeated up to 5 times. After each
repetitions indicate how many words out of 10 the patient reproduced. Finally, an hour later, they ask to name the same
words, but without re-naming them. Using the data obtained, a memorization curve is made.
Usually, after the 3rd repetition, 9-10 words are memorized; and this figure is held in the 4th and 5th samples. In one hour
remains in memory 8-10 words. With poor memorization, the number of words reproduced is much less.

To assess visual memory, the subject is offered a table with 10 words, which is shown in
for 1 min, and ask to write down the memorized words.
Short-term memory can be examined by offering to repeat a certain text with a line
addition. For example, “one special original”, “two kind wild porcupines”, “three fat quiet
tarantula "," four devils scratched the skull of an eccentric "," five quails sang, having a hearty dinner ", etc.
with hypomnesia, the patient finds it difficult to accurately reproduce the text already with the addition of the 2nd or 3rd phrase. With amnesia
the patient finds it difficult to repeat even one suggested phrase, for example, “black earth, green grass, blue sky".

For the study of logical memory, associative memorization, the “words +
Pictures". The patient is called 10 words and asked to pick up cards with a picture for each word
various objects: for example, to the word "light" - an image of a light bulb, "lunch" - loaves of bread, "carrots" -
hare, "mushroom" - forests, etc. After the subject selects the cards in response to the named words, their
put it aside and after an hour they are asked to remember from the pictures which words they correspond to. In this
the technique is important not only the number of words that the patient remembered, but also the features of his associations
- primitiveness, complexity, pretentiousness.
The pictogram method is also used. The patient is asked to draw pictures that make it easier to remember
presented words. Along with assignments, depict specific concepts ("sunny day", "spring", "cheerful
holiday ”), offer more abstract ones (“ friendship ”,“ development ”,“ intensification ”). Character is assessed
drawings - their excessive detailing or, conversely, symbolism.

THINKING AND ITS DISORDERS

Thinking is the highest form of cognitive activity, in the process of which internal
connections between objects and phenomena of the surrounding world. Thinking is closely related to speech - the most important
a means of communication between people, making it possible to transfer information, knowledge, experience. Process
thinking includes the stages of analysis, synthesis and generalization. It is carried out mainly in representations,
judgments, inferences and concepts.
A person's thinking is judged by his statements and activities. Level of thinking, ability to
penetration into the essence of emerging problems and the development of the most adequate solutions are included in the concept
intelligence.
Allocate congenital dementia (oligophrenia) - the ability to learn suffers, the child can
advance in mental development, but he is not able to catch up with his age level; delay
mental development (the child is capable of learning; when eliminating the causes of this delay and the corresponding
pedagogical activities, the child can catch up with his age level); dementia
(dementia) - characterized by an increasing loss of acquired skills and higher brain functions.
There are 3 degrees of dementia: idiocy (complete absence of phrasal speech and the possibility of social adaptation), imbecility (there are primitive speech, basic service skills and the ability to
the simplest labor operations; the intellect of patients is delayed in development at the level of a 3-5-year-old child) and
debility (higher levels of the psyche suffer - abstract thinking, logical analysis; orientation in
practical everyday questions is quite adequate).
The various types of thought disorders are detailed in the course of psychiatry. Here we note
occurring disorders of the pace of thinking - painfully accelerated thinking (the flow of thoughts is so accelerates, that the patient does not have time to pronounce the words, prepositions, endings and even whole words are omitted) and
painfully slow thinking (characterized by poor associations, lethargy,
stuck).
Thoughtful thinking consists in extreme viscosity, stiffness of mental
processes, it is difficult for the patient to switch from one topic to another, he cannot single out the main, main, gets stuck on insignificant details, trifles. This symptom is most common in epilepsy.
Disruption of thinking is characterized by the absence of logical connections between words in the patient's speech;
speech becomes devoid of any meaning; grammatical connections in sentences can be preserved.
If both the logical and grammatical connection is broken, they speak of incoherent thinking.
Reasoning - fruitless reasoning, philosophizing, idle talk, lack of concreteness
content.
Pathological ideas (delusional) are a false, erroneous judgment, inference, inaccessible to criticism
and correction. In terms of content, delusional ideas are divided into 3 main groups:
relationship, poisoning, exposure), greatness (delirium of invention, wealth) and self-deprecation (delirium self-accusation, sinfulness, guilt, hypochondriacal, etc.). When patients have delusional ideas
consultation of a psychiatrist is required. Topical diagnostics according to the peculiarities of thinking disorders has not yet been

developed, however, it is known that pronounced disorders of thinking and intelligence develop with defeat frontal lobes of the brain.
For the study of thinking, one of the simplest methods is to sequence
events in a series of drawings. The patient should arrange the cards with pictures in a logical sequence. and compose a story based on them. You can also read a short story to the patient and ask him to retell it.

content; at the same time, memory, perception, the ability to highlight the main storyline are assessed.
The analysis of the patient's interpretation of proverbs and metaphors can be very indicative. For example, the proverb, “seven times
measure, cut once ”, metaphors“ golden hands ”,“ dead night ”,“ stone heart ”,“ knife in the bosom ”, etc. patients with dementia understand literally and cannot interpret their figurative meaning.
The method of comparing concepts is informative: the subject is offered in pairs and asked
explain what their similarities and differences are. Along with comparable concepts (tram - bus, lake - river, hunger - thirst), offer and incomparable (glass - rooster, rain - sugar, wind - salt, etc.). Evaluated
the ability of patients to think logically, to generalize, to compare.

CONSCIOUSNESS AND ITS DISORDERS

Consciousness is the highest form of reflection of reality, which is a combination of
mental processes that allow a person to navigate in the world around them, time, their own personality, ensuring the continuity of experience, unity and diversity of behavior. It is associated with material processes, embodied in cerebral neurodynamics, speech, object-related human activity. V clinical practice, the state of consciousness is judged by orientation in the environment (in time, place stay, people around) and in your own person.
Disturbances of consciousness are conventionally subdivided into states of shutdown and dimness of consciousness.
Consciousness shutdown syndromes. Stunnedness - characterized by an increase in the threshold of perception. Responses in such patients are caused by stimuli of only greater than usual strength. Questions,
asked to the patient, you have to repeat several times, pronounce them louder than usual. Voice contact it is difficult to establish with the patient. Patients are lethargic, inhibited, indifferent (the events of the surrounding
do not really attract their attention), are disoriented. Their answers are monosyllabic or confusing contradictory, they poorly fix attention on the topic of conversation, facial expressions are poor. This condition is usually
observed with a brain tumor and is referred to as “congestion”.
Stupor is a condition in which patients do not respond to verbal appeals, are immobile. Unconditional their reflexes are preserved - pupillary reflexes to light, corneal reactions to painful irritations. Also retained and deep reflexes on the upper and lower extremities. Sometimes with such a patient it is possible to establish
contact by patting on the cheek or with repeated loud calls. The patient opens his eyes, tries say the words, but after a few seconds it stops responding again to any stimuli.
Coma is the most profound shutdown of consciousness, in which both conditioned and unconditioned reflexes (with the exception of breathing and cardiac activity). Pathological foot
reflexes (Babinsky reflex).
The state of switching off consciousness occurs when the function of the reticular formation of the brain stem is impaired
(turning off activating systems), with brain injury, cerebral strokes, severe
intoxications (endogenous, exogenous) and infectious diseases (typhoid fever, influenza, pneumonia, meningoenpephalitis), brain tumors, epilepsy, etc.
Clouding syndromes. Delirious syndrome is characterized by impaired orientation in
self. One of the leading symptoms of delirium is visual, as well as auditory and tactile hallucinations. Patients experience scenes in which memories of actual events are bizarre.
mixed with hallucinatory images, often frightening content. The emotional sphere is usually characterized by the affects of fear, anxiety, which correspond to the delusional ideas of relationship, persecution.
The behavior of patients corresponds to the experienced pathological experiences, they are excited. restless do not stay in bed. In the evening and at night there are exacerbations of hallucinatory experiences; sick can become dangerous to themselves and others. During the day, periods of clear consciousness with critical attitude to their experiences. At the end of delirium, memories are only partially preserved.

Clouding of consciousness of the delirious type is observed with alcohol intoxication ("delirium tremens"), with
traumatic and infectious brain lesions.
Amentive syndrome is a more severe state of confusion with impaired
orientation both in the environment and in one's own personality. With amentia, patients lose the ability
establish connections between surrounding phenomena and objects. They are confused, they look surprised
scared. Fragmentary hallucinations, more often auditory, may be observed. Their speech is
incoherent. Amentia may
last up to several weeks and months. When the condition worsens, complete
amnesia. The amentive condition occurs in severe infections (especially in encephalitis with lesions
brain stem), intoxications against the background of changes in general reactivity.
Oneiric syndrome is characterized by the presence of fantastic delirium, bright dream-like
hallucinations. All the experiences of patients are unusually colorful, bizarre. They see themselves
traveling in space, on other planets, in other historical eras. With oneiroid, patients usually
immobilized, lie motionless for a long time in bed or freeze in ridiculous poses with a blissful smile on
face. Only occasionally does psychomotor agitation occur. Orientation in the environment and in oneself
broken. The oneyroid state lasts for several days or weeks and is more often observed with
encephalitis, intoxication, schizophrenia. Upon leaving the oneyroid, patients can remember their experiences
and tell about them.

Twilight clouding of consciousness is expressed by a sudden sharp narrowing of the field of consciousness.
Distinguish between the hallucinatory-paranoid form and outpatient automatisms. With hallucinatory
The paranoid form of the behavior of patients is determined by the content of hallucinatory and delusional
experiences.
Outwardly, it may look orderly, purposeful. Often anxiety, fear, tense
the affect of anger, causeless rage. It is impossible to contact the sick, their answers are incoherent,
incomprehensible. Under the influence of hallucinatory and delusional experiences, patients can commit
meaningless,
sometimes aggressive, actions and therefore dangerous for themselves and others. The twilight state lasts from
several minutes to several days, ends abruptly, sometimes turning into deep sleep. No
memories of the experience usually do not remain.
Ambulatory automatisms include somnambulism and trans-twilight states without productive
symptoms (no delirium, hallucinations, affective disorders). Sleepwalking (sleepwalking) occurs during
sleep. At night, the patient gets out of bed and performs a number of sometimes rather complex automatic
actions:
dresses, walks around the room, goes up to the attic of the house or goes outside, after which he usually returns
to bed and continues to sleep. After awakening, the patients do not remember their actions.
Trance is a short-term state of ambulatory automatism, in which the patient commits
impulsive purposeful actions, which he does not remember in the future (leaves for another area of the city and
etc.).
Twilight states are most typical for epilepsy, traumatic brain injury, pathological
intoxication. Twilight consciousness is possible with hysteria, but in this case, the possibility remains partially
orientation in the environment, and later on there is partial amnesia.
Impaired consciousness usually manifests itself in a state of wakefulness with dysfunction of the reticular
formation of the brain stem, as well as in pathology of the cerebral cortex.
In recent years, the “locked-in” syndrome observed in defeats has been identified.
brain stem, - complete immobility of the patient, with the exception of blinking and movement of the eyeballs
along
vertical plane. If it is possible to come into contact with such a patient, then it is found that a difficult
his mental activity is preserved.
The syndrome of akinetic mutism is observed in predominantly subcortical lesions with
relative preservation of the function of the cerebral cortex.

NEUROTIC SYNDROMES

In practical work, a neuropathologist has to constantly identify and differentiate
signs of organic damage to brain structures and functional disorders of the nervous system. Patient
it is with functional disorders of the nervous system that there is much more. That's why
it is advisable to give at least a brief summary of the main neurotic syndromes.
Asthenic syndrome is characterized by increased fatigue and exhaustion. Sick

complain of general weakness, irritability, decreased appetite, sleep disturbance (dyssomnia). Decreases working capacity. The mood is unstable, there is tearfulness, lack of confidence in their abilities. Frequent headaches, dizziness of an orthostatic nature (with a sudden rise from bed, when straightening from the tilt position, etc.). Asthenic syndrome is characteristic of neurasthenia and many somatic and infectious diseases.

Obsessive-compulsive disorder (obsessive-compulsive disorder) is manifested by various painful doubts, fears, drives, actions, etc., that arise in a person against his will. With obsession the patient is almost always critical of this condition. With all the understanding of the absurdity of their patients cannot get rid of fears and actions. Obsessive fears (phobias). In terms of content, they can be different: fear of closed rooms (claustrophobia), acute objects, narrow streets or wide squares (agoraphobia), fears of getting sick with a serious illness (cardiophobia, carcinophobia). The patient realizes that he is completely healthy, but obsessive fears do not give him rest. Obsessive drives are expressed in the appearance of unusual unpleasant desires - to jump out on the tramway, trains, grab a passerby by the nose, spit in the back of the head in front of a sitting person, etc. Such obsessions usually they do not turn into actions, but the struggle with them is painful and painful for the patient. Obsessive actions extremely varied: shutting eyes, sniffing, biting nails, coughing.

Hysterical syndrome is characterized by emotional instability. Patient behavior determined more by feelings, mood than reason, and has demonstrative features. Demanding to myself special attitude, patients constantly strive to be in the spotlight. There is a tendency to posturing, eccentricity, fantasy, theatricality. These patients often have neurological disorders in the form of paralysis (with preserved deep reflexes, the absence of pathological carpal and foot signs), hemianesthesia (with a border strictly along the midline), amaurosis (with the preservation of a direct reaction pupil to light in the "blind" eye), etc. Hysterical syndrome is the basis of neurosis.

VASCULAR DISEASES OF THE BRAIN AND SPINAL CORD

Acute disorders of cerebral circulation

Transient disorders of cerebral circulation

It is customary to refer to transient disorders of cerebral circulation such disorders of cerebral hemodynamics, which are characterized by suddenness and short duration of dyscirculatory disorders in the brain, manifested focal and / or cerebral symptoms. According to the WHO recommendation to transient disorders of cerebral circulation include those cases when all focal symptoms disappear in 24 hours. If they last more than a day, then such observations should be regarded as a cerebral stroke.

To transient disorders of cerebral circulation, past transient ischemic attacks, include hypertensive cerebral crisis, manifested as focal and cerebral symptoms.

Transient ischemic attacks

Etiology. Transient ischemic attacks complicate for many diseases, but most often atherosclerosis and hypertension. Much less often they are found in vasculitis of different etiology (infectious-allergic, syphilitic), with vascular systemic diseases (periarteritis nodosa, SLE, temporal arteritis), heart disease (heart defects, mitral prolapse - left atrioventricular - valve, myocardial infarction, etc.). Osteochondrosis the cervical spine also affects the blood flow in the vertebral arteries and is a common cause of transient neurological symptoms vascular genesis. Thus, a transient ischemic attack is

complication of one of many diseases, which requires clarification in each specific observation.

Pathogenesis. One of the most common mechanisms for the development of transit ischemic attacks are considered to be an embolism of cerebral vessels, and emboli are the smallest particles separated from blood clots located in the cavity of the heart or in the great vessels, and can also consist of crystals cholesterol expelled from disintegrating atheromatous plaques.

An important role in the development of attacks is played by arterio-arterial embolism, in which emboli are formed in large arteries, more often in the main vessels of the head, from where, moving along with the blood flow, they enter the terminal branches of the arterial system, causing them to occlude. Arterio-arterial microemboli consist of an accumulation of blood cells - erythrocytes and platelets, which form cellular aggregates that can disintegrate, undergo disaggregation, and therefore, can cause temporary vessel occlusion. Increased aggregation of erythrocytes and platelets and the formation of microemboli contribute to the appearance of ulcerated atheromatous plaque in the wall of a large vessel, changes in physical and chemical characteristics of the blood.

Transient ischemic attacks can also be caused by thrombosis or obliteration of a large vessel, often the main one on the neck, when preserved and a normally formed arterial circle of the large brain is able to restore blood flow distal to the occlusion site. Thus, a well-developed network collateral circulation in thrombosis of the vessel is able to prevent persistent

ischemia of the medulla, causing only a transient violation of the cerebral hemodynamics.

In some cases, transient ischemic attacks occur by the mechanism "Stealing" - the diversion of blood from the main cerebral vessels into peripheral circulatory network. So, with occlusion of the subclavian artery blood supply to the hand is carried out from the vertebrobasilar basin, from where, to the detriment of the brain carries out a retrograde blood flow ("subclavian syndrome robbery "). Transient ischemic attacks can develop with stenosis cerebral or great vessels of the head, when the fall joins blood pressure due to various physiological or pathological conditions (deep sleep, myocardial infarction, cardiac rhythm, bleeding, etc.).

Clinic. Transient ischemic attacks develop in most cases acute, sudden and much less frequent prolongation with slow development of focal and cerebral symptoms. Clinical manifestations are varied and depend on localization and duration of dyscirculatory disorders.

Distinguish between cerebral symptoms and focal, caused by disorders

blood flow in a specific vascular basin. General cerebral symptoms in those cases when they occur, they are characterized by headache, vomiting, feeling of weakness, veils before the eyes, short-term disturbances of consciousness are possible.
Focal,
or
regional,
symptoms
are determined
localization
dyscnrulatory disorders in the internal carotid artery system or
vertebrobasilar basin.
Transient ischemic attacks have a different duration - from
several minutes to a day, usually 10-15 minutes. A characteristic feature of attacks is their
repeatability. The frequency of relapses is different and ranges from one to 3 times or more per year.
The greatest recurrence is observed with the development of attacks in the vertebrobasilar
seine. The prognosis when they appear in the carotid system and the brain stem is the most
serious. With the indicated localization, transient ischemic attacks are often preceded by
the development of cerebral stroke in the period from one to 2-3 years, but more often during
the first year after the appearance of the first ischemic attack. Greatest risk of developing
a stroke exists for a month after the attack. The most favorable prognosis
have transient ischemic attacks developing in the bed of the labyrinth artery and proceeding with a less-like symptom complex. The prognosis is unfavorable for the development of attacks against the background of cardiac pathology, especially accompanied by a violation
heart rate. Keep in mind that attacks can be more than just precursors
cerebral stroke, but also myocardial infarction.
According to the course and prognosis of cerebral circulation disorders to severe forms
transient ischemic attacks approaching stroke with reversible neurological deficiency, or the so-called minor stroke. This term denotes a stroke,
in which the restoration of the affected brain functions occurs within
the first 3 weeks from the moment of an acute episode of cerebrovascular accident.
Highlighting small strokes is justified by the fact that focusing on them
attention will help to find out what features of vascular lesion and mechanisms recovery and compensation contribute to the fastest elimination of the deficit
brain functions. Among the pathogenetic mechanisms, all the same mechanisms are noted
which cause transient ischemic attacks.

Diagnostics. With the sudden appearance of focal and cerebral symptoms disorders of cerebral circulation, if they continue for several hours, one cannot be sure whether this violation will be transient or develop cerebral infarction. In these cases, the diagnosis of transient ischemic attack is made retrospectively after the disappearance of symptoms of cerebral circulation. In the lungs cases where the symptoms of circulatory disorders last from several minutes to hours, the diagnosis of a transient ischemic attack is not very difficult.
In some cases, it becomes necessary to differentiate the attack from partial epileptic seizures, fainting.
A transient ischemic attack can develop against a background of complete well-being and be the first manifestation of the pathology of the cardiovascular system. Therefore, it is clear that in such cases it is very important to find out the underlying disease, which is complicated attack.

Treatment. They carry out treatment taking into account the pathogenetic mechanisms of the development of the attack and underlying disease, complicated by transient ischemia. Treatment should be aimed at eliminating or compensating for the underlying disease, prevention of recurrence of transient ischemic attack and prevention cerebral stroke. In mild cases with the disappearance of symptoms of the disorder blood circulation within the next few minutes, it is possible to examine and outpatient treatment. In cases with sufficiently persistent and pronounced neurological disorders and especially with repeated attacks is indicated hospitalization. It is attacks that are repeated at short intervals that often precede the rapid development of a stroke.
Therapeutic measures should be aimed at improving the brain blood flow, activation of collateral circulation, improvement of microcirculation. In most cases, due to the short duration of transient ischemic attacks, therapeutic measures during the actual episode of transient ischemia are impossible. Therefore, the main task is to clarify the possible cause of the attack. and prevention of repeated transient ischemia and ischemic stroke. Sick, who have undergone a transient ischemic attack, it is necessary to carry out a complete examination of the cardiovascular system and the hemostasis system (ECG, with the need for long-term monitoring of the ECG to exclude transient disturbances heart rate changes, echocardiography, Doppler ultrasonography of the main and if possible, intracranial vessels, if indicated - significant stenosis of the magastral vessels of the head - angiography, general and biochemical blood test to exclude vasculitis of various etiologies, blood diseases, hemostasiological study). This will allow for more targeted treatment and prevention. In cases of a prolonged episode of ischemia during its

manifestations, when it is not yet clear whether it will be an attack or a real stroke, examination
and the treatment is carried out as in a cerebral stroke. Further therapy includes normalization of the state of the cardiovascular system - elimination of violations heart rate, normalization of blood pressure (remembering the danger its excessive decrease, which can lead to repeated transient ischemic attacks and stroke), therapy for other background diseases that caused the development of the attack.
The main thing in the further management of patients is the use of antiplatelet agents. and if indicated - anticoagulants. The most effective antiplatelet agents are acetylsalicylic acid (aspirin) at a dose of 250-300 mg per day at one time or

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ticlopidine (tiklid) at a dose of 500 mg per day. Duration of taking these drugs ranges from one to several years, depending on the condition of the patients, tolerance of drugs, indicators of hemostasis. It is necessary in this case, of course, take into account contraindications, possible side effects of these drugs - nausea, vomiting, constipation, stomach bleeding while taking acetylsalicylic acid, neutropenia, diarrhea and other gastrointestinal disorders while taking tiklide. Therefore, clinical and laboratory monitoring of the patient's condition is necessary. At
the presence of a stomach or duodenal ulcer, erosive gastritis,
the use of acetylsalicylic acid is contraindicated and ticlide is prescribed.
Less effective, but useful in these cases, is pentoxifylline
(trental, agapurin). Shown a positive effect in terms of reducing aggregation and enhancing platelet disaggregation of bromcamphor (0.5 g 3 times a day).
Controlled studies of the action of dipyridamole (courantil) have not shown it effectiveness in the prevention of cerebral stroke.
Therapy of transient ischemic attack with prolonged and severe
course, embolic genesis should include anticoagulants (after exclusion with by the power of CT of the hemorrhagic nature of the vascular cerebral disorder, since small in volume hemorrhagic foci can give an attack clinic). At first
prescribe heparin with intravenous, preferably continuous administration at a dose of 20,000-30,000
DB for several days with the subsequent transition to indirect anticoagulants in adequate doses, determined by clinical and laboratory data, during
several months.
Intravenous administration of rheopolyglucin (400 ml), sulfocamphocaine 2 ml is shown
2-3 times a day, 1 ml of a 24% solution of aminophylline intramuscularly or 10 ml of a 2.4% solution in
10-20 ml of 20-40% glucose solution intravenously. These drugs have antiplatelet action. Significant volumes of fluid and administration of aminophylline contraindicated in the presence of signs of heart failure, violations
heart rate.
It is possible to use drugs of metabolic action - piracetam 3-4 g / day,

Cerebrolysin 10-20 ml intravenously drip in isotonic sodium chloride solution.
When stenosis of the common or internal carotid artery is detected in a volume of more than 70%
surgical treatment (endarterectomy) is indicated.

Hypertensive cerebral crises

Hypertensive cerebral crises are, according to some sources, 13-15%
from all acute disorders of cerebral circulation. Against the background of the rise blood pressure exceeding 180-200 mm Hg, cerebral autoregulation
blood circulation is disrupted and cerebral hyperemia may develop,
accompanied by cerebral and focal symptoms that disappear during
days. A crisis is an exacerbation of hypertension or symptomatic
hypertension.

Clinic and diagnostics. According to the characteristics of hemodynamic disorders, it is possible
distinguish three types of crises: hyperkinetic, in which the heart rate increases eukinetic ejection with normal cardiac output and increased
general peripheral resistance responsible for the development of acute hypertension, hypokinetic with reduced cardiac output and, accordingly, with a sharp
an increase in total peripheral resistance. Identifying the underlying mechanism

hypertension in crisis is important for the choice of treatment.

Treatment. Determining the type of hypertensive crisis makes it possible to optimally choose an antihypertensive drug for the rapid relief of a hypertensive crisis with taking into account the peculiarities of hemodynamic disorders.

Acute hypertensive encephalopathy

Against the background of high arterial hypertension, in some cases, acute conditions characterized by severe headache, nausea, vomiting, non-systemic dizziness followed by impaired consciousness that may last
several days and often ends in death. At the heart of acute hypertensive encephalopathy is cerebral edema, multiple small foci of hemorrhage and ischemia.

Acute hypertensive encephalopathy is rare and accompanies
renal hypertension, eclampsia, essential hypertension. It develops in persons suffering from arterial hypertension, and develops with rises in systolic
blood pressure above 200-270 mm Hg. and diastolic - above 120 mm Hg.

Pathogenesis. The development of acute hypertensive encephalopathy is associated with
filtration edema and brain swelling in response to increased cerebral blood flow with high blood pressure and with intravasal plasmorrhages and
hemorrhages developing in the pia mater of the cerebral hemispheres
and the brain stem. In addition, the development of cerebral edema, as a rule, leads to a decrease in
cerebral blood flow, which determines the developing small foci
softening of the brain parenchyma.

Causes and mechanisms of development of brain tissue hypoxia arising from

hypertensive encephalopathy, until recently remain the subject of discussion.
The theory of spasm has become widespread. According to this theory, in response to an acute and significant increase in blood pressure, a sharp narrowing occurs cerebral vessels - their spasm, causing a decrease in blood flow and ischemia of the brain tissue. By
the cessation of spasm develops a paralytic state of the arteries, which accompanied by a sharp increase in the permeability of the vascular walls in relation to
the liquid part of the blood, plasma and even erythrocytes, since it was assumed that the spasm
severely damages the walls of the arteries. As a result, cerebral edema may occur, plasmorrhages and more or less large hemorrhages.
Another hypothesis was based on the fact that hypertensive encephalopathy is the result of excessive stretching of the arterioles. In recent years, the theory of stretching
again attracts attention, since it was found that the permeability of the vessel rises in the widened, and not in its narrowed area. It was found that the narrowing cerebral vessels is only an expression of their normal autoregulatory response, allowing cerebral blood flow in conditions of arterial hypertension to remain on normal, not elevated levels.
Study of cerebral blood flow with changes in blood pressure,
carried out by a number of researchers and summarized in the works of NALassen, showed that
with arterial hypertension above 200 mm Hg. an increase may be noted cerebral blood flow due to the disruption of the mechanism of its autoregulation, kosche the arteries of the brain already
cannot resist increased blood pressure and passively
stretched by blood, as a result of which blood rushes into the capillaries of the brain under
high pressure. The expansion of the cerebral vessels is not diffuse, but

localized mainly in small arteries and arterio-arterial anastomoses
zones of adjacent blood circulation.
As a result, the release of the liquid part of the plasma from the dilated vessels is noted and
the filtration edema of the brain develops, which also has a spotty character. In ka-pillars of the brain, squeezed from the outside by enlarged processes of astrocytes, increases
resistance of the microvasculature and a sharp decrease in blood flow, which leads to the development of hypoxic foci in the brain parenchyma.
Clinic. The clinical picture of hypertensive encephalopathy is
primarily from the grossly expressed cerebral symptoms. In the foreground are diffuse headaches, less often localized in the occipital region, which are worn pressing or bursting in nature and often accompanied by nausea and vomiting, a feeling of noise in the head, dizziness; predominantly of a non-systemic nature,

"Flashing flies" or a veil in front of the eyes, often a sharp decrease in vision. Vegetative-vascular phenomena are rather roughly expressed: hyperemia or pallor face, hyperhydrolysis, pain in the heart, palpitations, dry mouth. In more severe cases are marked by impaired consciousness, deafness, drowsiness, psychomotor agitation, disorientation in place and time, and generalized epileptic seizures. Meningeal symptoms may be expressed. ptoms. In the fundus, edema of the papilla of the optic nerve is detected. From focal symptoms in acute hypertensive encephalopathy, visual disturbances are often noted, as well as numbness, tingling, decreased pain sensitivity in the area of the hand, language, sometimes according to the hemitype. Movement disorders, pre-property in hand. However, it should be emphasized that focal microsymptoms in acute hypertensive encephalopathy is often absent, and the main core of clinical symptoms represent cerebral symptoms. When acute hypertensive conditions in patients may develop persistent focal symptoms, most often scattered, but mainly hemispheric localization and in zones of different vascular channels. The first episode of acute hypertensive encephalopathy is usually has a favorable outcome, however, in some observations it may end lethal.

Treatment. In acute hypertensive encephalopathy, hypotensive drugs are used drugs, diuretics. At the same time, one should not reduce too quickly and strongly blood pressure, as this can lead to aggravation and development of ischemia. It is advisable to maintain the pressure at about 150/100 mm Hg. The best the drug for this is sodium nitroprusside, which is administered intravenously with a rate of about 8 μg / kg per 1 min under constant monitoring of blood pressure. To reduce the severity of cerebral edema and reduce ICP, dexamethasone 4-6 mg 4 times a day (or an appropriate dose of prednisolone). With eclampsia continue to apply magnesium sulfate intravenously slowly 10-20 ml of 20-25% solution.

In cases of development of epileptic seizures, anticonvulsant therapy is started. After normalization of blood pressure, they switch to conventional oral antihypertensive drugs.

Strokes

According to the WHO definition, stroke is characterized by rapidly developing signs of local and sometimes diffuse dysfunction of the brain, lasting more 24 hours or leading to death and caused by causes of a vascular nature.

Cerebral stroke is observed among the population quite often. Based on materials WHO, the incidence of strokes during the year ranges from 1.5 to 7.4 per 1000 population, which in to a certain extent depends on the age of the surveyed. Stroke every year in Europe affects more than 1,000,000 people.

The incidence of stroke increases with age. Most the incidence of stroke is high between the ages of 50 and 70. So, at the age of 50-59 years, the incidence of strokes is 7.4, and at the age of 60-69 years - 20 per 1000

population.
Increase in cerebrovascular disease over the past three decades
depends on many reasons, but to a certain extent is associated with the lengthening of people's lives
and an increase in the proportion of elderly and senile people in the population.
Over the past three decades, in a number of countries, the structure of vascular diseases of the brain due to the clear predominance of ischemic strokes over hemorrhagic. If before 1945 the ratio of cerebral hemorrhages and cerebral infarctions equaled 2: 1, 4: 1, and during the Second World War 7: 1, now it is co-
the ratio became 1: 4.
Stroke is the most serious complication of heart disease.
vascular system. Mortality from cerebral strokes in highly developed countries
is among the first three reasons in the structure of general mortality, and in some years mortality from stroke exceeds mortality from myocardial infarction.
The degree of disability in stroke patients is also high. Only 20% of patients
with cerebral stroke return to work, 60% - remain disabled and 20% -
need outside care.

Hemorrhagic stroke

Hemorrhagic stroke refers to hemorrhage in the brain substance.
(cerebral hemorrhage, or parenchymal hemorrhage) and intrathecal
spaces (non-traumatic subarachnoid, subdural and epidural
hemorrhage). There are also combined forms of hemorrhage - subarachnoid
parenchymal,
parenchymal-subarachnoid
and
parenchymal
ventricular.

Cerebral hemorrhage

Etiology. Cerebral hemorrhage most often develops in hypertensive
disease, as well as with arterial hypertension caused by kidney disease,
lesion of internal secretion (pheochromocytoma, pituitary adenoma) and with systemic
vascular diseases of an allergic and infectious-allergic nature,
high blood pressure (periarteritis nodosa, red
lupus). Cerebral hemorrhage can occur with congenital angioma, arterio-
venous malformation with microaneurysms formed after cranial
brain injury or septic conditions, as well as diseases,
accompanied by hemorrhagic diathesis - with Werlhof disease, leukemia and uremia, with vascular lesions by tuberculosis, etc.

Pathogenesis. Currently, all authors recognize that in the pathogenesis

hemorrhages of greatest importance is arterial hypertension. Hypertension, especially with hypertension, leads to changes in blood vessels, fibrinoid degeneration and hyalinosis of the arteries of the kidneys, heart, brain. Vascular changes go through several stages:

subendothelial serous infiltration with increased endothelial permeability for
blood plasma is accompanied by perivascular transudation and contributes to further
we concentrate the walls of blood vessels due to the fibrinoid substance.
The rapid development of fibrinoid degeneration leads to the formation of enlarged
arterioles and aneurysms. In this case, it can be observed that blood elements
penetrate into
torn structures of the arterial walls, and in these places can form
thrombosis.
As a result of fibrinoid-hyaline necrosis of the arterial walls,
develop dissecting aneurysms, which are considered the cause of bleeding in
in case of rupture of the vessel.
The intensity and size of cerebral bleeding are determined by the size
aneurysm, the pressure of blood flowing from it and the speed of its thrombosis. Most
often hemorrhage develops in the area of the basal nuclei from the arteries supplying
striped body.
In the overwhelming majority of cases, hemorrhage develops in patients with
hypertension and all other diseases that are accompanied by
arterial hypertension. With atherosclerosis without arterial hypertension
hemorrhages are very rare.
In diseases not accompanied by arterial hypertension (diseases
blood, somatic diseases accompanied by hemorrhagic diathesis, uremia
and others), the main mechanism for the development of hemorrhage is diapedesis
due to
increasing the permeability of the walls of blood vessels.
Clinic and diagnostics. Hemorrhage usually develops suddenly,
usually during the day, during the period of vigorous activity of the patient, although
in isolated cases
hemorrhages are observed both during the patient's rest period and during sleep.
For hemorrhages in the brain, a combination of cerebral and focal
symptoms. Sudden headache, vomiting, impaired consciousness, rapid noisy air
hanie, tachycardia with the simultaneous development of hemiplegia or hemiparesis -
common
initial symptoms of hemorrhage. The degree of impairment of consciousness is
different, from
light stunning to a deep atonic coma. When determining depth
disorders of consciousness pay attention to the possibility of contact with the patient,
implementation of simple and complex instructions for patients, the ability to
communicate
anamnestic information, the speed and completeness of the patient's responses, take in
attention safety of criticism, attitude to one's condition, patient orientation
in the environment.
With a relatively deep loss of consciousness (stupor) of speech contact with the
patient
no, only the patient's reaction to loud sounds, to an injection or a series of injections is
recorded.

With a mild degree of impairment of consciousness (stunning), both in answers to questions and
following instructions, even if the patient does not have aphasia, slow reactions are visible,
increase in the latency period. Complicated instructions fail, patient
quickly “depletes” and “turns off”. Can provide information about himself, although he confuses them,
answers questions slowly and out of place. Motor activity is often noted.
the patient's anxiety, anxiety, underestimation of his condition, reaction to pain is preserved.
The stunning or stupor observed in the initial period may after a few

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hours go into a coma. Coma is characterized by a deeper impairment of consciousness and
vital functions: respiration, cardiac activity. The patient does not respond to
a single injection, weak and medium sounds, to the touch, does not withdraw the "healthy"
hand in response to a series of injections. With an atonic coma - an extreme degree of violation
consciousness - all reflexes are lost: pupillary, corneal, pharyngeal, skin, sub-
hobby reflexes are not triggered, blood pressure drops, breathing is impaired
until it stops. The general appearance of the patient with massive hemorrhage in hemispheres: eyes are often closed, the skin is hyperemic, often sick
covered in sweat. The pulse is tense, the blood pressure is increased. Eyes are often turned
towards the affected hemisphere (paralysis of the cortical center of gaze), the pupils may be
of different sizes, anisocoria occurs in 60-70% of hemorrhage hemorrhages
localization, a wider pupil is usually on the side of the focus. Often noted
divergent strabismus, which, like anisocoria, is due to compression
oculomotor nerve (III) on the side of the hematoma. These signs are symptoms
indicating the developing compression and displacement of the brainstem by hematoma
and perifocal cerebral edema, initially occurring in the hemisphere, where
there was a hemorrhage.
The most common focal hemorrhage symptom is hemiplegia, usually
combined with central paresis of the facial muscles and tongue, as well as with hemihypesthesia in the contralateral limbs and hemianopsia.
With parenchymal hemorrhages after a few hours, sometimes towards the end
1 day meningeal symptoms appear. In this case, the stiffness of the cervical muscles
may not be at all, the upper Brudzinsky symptom is rarely called. Meanwhile with
the Kernig symptom on the non-paralyzed side and
positive lower Brudzinsky symptom. Absence of Kernig's symptom on the side
paralysis is one of the criteria for determining the side of the lesion.
An increase in body temperature is observed in patients with parenchymal

hemorrhage a few hours after the onset of the disease and keeps
several days in the range of 37-38 ° C. With the breakthrough of blood into the ventricles and with the proximity of the focus
hemorrhages to the hypothalamic region, the temperature reaches 40-41 ° C. Usually, leukocytosis appears in the peripheral blood, a slight shift in the leukocyte
formulas to the left, on the 1st day of the disease there is an increase in the glucose content in
blood, usually not higher than 11 mmol / l, an increase in the residual nitrogen content is possible
in blood. Increased fibrinolytic activity of the blood is often noted and in pain
In the majority of patients, platelet aggregation is reduced.
Course and forecast. With cerebral hemorrhages, there is a high
mortality, which, according to various authors, ranges between 75-95%. Up to 42-45%
patients with massive cerebral hemorrhage die within 24 hours from the onset
stroke, the rest - on the 5-8th day of the disease, and in rare cases (usually in the elderly)
life expectancy from the onset of hemorrhagic stroke can be 15-20 days.
The most common cause of death in patients with hemorrhagic stroke is
infringement of the brain stem with hemispheric hemorrhage due to cerebral edema. Second
the place in terms of the frequency of causes of death in this group of patients is occupied by the effect of
focus, massive breakthrough of blood into the ventricular system with the destruction of vital
important vegetative formations.
Treatment. A patient with cerebral hemorrhage must be properly placed in
bed, giving the head an elevated position, raising the head end of the bed.

In case of cerebral hemorrhage, it is necessary to carry out therapy aimed at
normalization of vital functions - respiration, blood circulation, as well as to stop bleeding and to combat cerebral edema, and then discuss the possibility of
prompt removal of spilled blood.
First of all, it is necessary to ensure free passage of the respiratory
ways, for which the removal of liquid secretion from the upper respiratory tract with using suction, the use of oral and nasal ducts. With accompanying
pulmonary edema, cardiotonic drugs are recommended: 1 ml of 0.06% is injected intravenously
korglikon solution or 0.5 ml of 0.05% strophanthin solution, and also use
inhalation of oxygen with alcohol vapors in order to reduce foaming in the alveoli.
Intramuscularly appoint 1-0.5 ml of 0.1% solution of atropine, 60-120 mg of lasix intravenously and 1 ml of 1% diphenhydramine solution. To reduce peripheral resistance and unloading of the small circle of blood circulation, 2 ml of a 0.25% solution are injected

droperidol. Due to the frequent development of pneumonia, an appointment is necessary.
antibiotics.
It is necessary to apply measures aimed at preventing and eliminating hyperthermia. At a body temperature of about 39 ° C and above, 10 ml of a 4% solution is prescribed
amidopyrine or 2-3 ml of a 50% solution of analgin intramuscularly. Recommended also
regional hypothermia of large vessels (ice bubbles on the area of the carotid arteries on the neck, axillary and groin areas).
In order to stop and prevent the resumption of bleeding, it is necessary
normalize blood pressure and blood clotting. Should not be reduced
the pressure is below the usual figures for the patient.
To lower blood pressure, dibazol is used (2-4 ml of a 1% solution),
clonidine (1 ml of a 0.01% solution). In the absence of effect, aminazine is prescribed (2 ml 2.5%
solution and 5 ml of 0.5% solution of novocaine) intramuscularly or as part of a mixture: 2.5%
chlorpromazine solution 2 ml, 1% diphenhydramine solution 2 ml, 2% promedol solution 2 ml. Mixture
injected intramuscularly. Ganglion blockers are also administered - pentamin (1 ml of 5% solution
intramuscularly or 0.5 ml in 20 ml of 40% glucose solution intravenously slowly under
blood pressure control), benzohexonium (1 ml of a 2.5% solution intramuscularly).
Antihypertensive drugs should be used with caution so as not to cause hypotension.
Ganglion blockers can dramatically lower blood pressure, so prescribe
they should be followed in exceptional cases, with arterial pressure exceeding 200 mm Hg, and insufficient effectiveness of other means. Inject ganglion blockers should be done carefully with constant monitoring of blood pressure.
Shown are drugs that increase blood clotting and reduce
vascular permeability: 2 ml of 1% solution of vicasol, dicinone intravenously or intramuscularly 1-2 ml of a 12.5% solution or inside 0.5-0.75 g, calcium preparations (intravenously 10 ml of 10% calcium chloride solution or intramuscularly 10 ml of 10%
calcium gluconate solution). Apply 5% solution of ascorbic acid, 5-10 ml intramuscularly. Vicasol solution and calcium supplements should not be used anymore.
one day, since the subsequently developing hypercoagulation will require its correction with heparin 5000 units 2 times a day to prevent the development thrombosis, more often localized in the inferior vena cava system and complicated life-threatening pulmonary embolism.
In patients with hemorrhagic stroke with pathologically increased

fibrinolytic activity should be assigned aminocaproic acid. Introduce her
in the form of a 5% solution intravenously drip under the control of the content of fibrinogen and
fibrinolytic activity of blood during the first 2 days.
To reduce intracranial hypertension and cerebral edema, glycerin is used, increasing the osmotic pressure of the blood without disturbing the electrolyte balance.
Glycerin is administered at the rate of 1 g per 1 kg of body weight. Glycerin is injected intravenously twice
in the form of a 10% solution (glycerin 30.0 g, sodium ascorbate 20.0, isotonic solution of chloro-
sodium read 250.0). Osmodiuretics are used under the control of blood osmolarity, which is desirable to keep at the level of 305-315 mosmol / l. The advantage osmodiuretics is to achieve a dehydration effect without disturbing
electrolyte balance.
Infusion therapy should be carried out under the control of indicators of CBS blood and
the electrolyte composition of plasma, in a volume not exceeding 1200-1500 ml / day.
Surgical intervention for intracerebral hematoma is reduced to removal
poured blood and creating decompression.
Currently, many years of experience have been accumulated in neurosurgical hospitals.
for the surgical treatment of hemorrhagic strokes. It could be considered
the generally accepted point of view of neurosurgeons that surgical treatment is indicated for
lateral hematomas with a volume of more than 50 ml and is inappropriate for medial and
extensive hemorrhages and with a coma of the patient. Surgical
treatment for lateral hematomas is advisable to carry out on the 1st day of stroke before
development of the dislocation of the brain stem. In the surgical treatment of hematoma
mortality compared with that with conservative therapy decreases from 80 to 30 40%.

Subarachnoid hemorrhage

Etiology. In most cases, the cause of spontaneous subarachnoid
hemorrhage is a rupture of an intracranial aneurysm. Intracranial
arterial aneurysms, like aneurysms of other localization, are
limited or diffuse expansion of the lumen of the artery or protrusion of its wall.
Most cerebral artery aneurysms have the characteristic appearance of a small
a thin-walled bag, in which you can usually distinguish the bottom, middle part, and so
called a neck.
Due to these anatomical features, such aneurysms are often
called saccular. Less commonly, the aneurysm looks like a large spherical formation or diffuse expansion of the artery over a considerable extent (the so-called S-

shaped aneurysms).
The wall of the aneurysm has a characteristic structure. It usually lacks
muscular layer, there is also no internal elastic membrane. Having lost
the three-layer structure inherent in the artery of the brain, the wall of the aneurysm is a plate of connective tissue of various thickness.
In the area of the bottom, the wall of the aneurysm is usually sharply thinned. It often contains
defects - from small, barely distinguishable, to large gaping holes. Breaks aneurysms, as a rule, are localized in the area of the bottom or lateral parts of it and almost never occur in the cervix. In the cavity of the aneurysm there may be
blood clots of various ages. Large
aneurysms.

Most aneurysms are located on the arteries at the base of the brain. Their
Favorite localization is the place of division and anastomosis of the arteries of the brain.
Especially often, aneurysms are localized on the anterior communicating artery, in the place
discharge of the posterior communicating artery from the internal carotid artery or in the area
branches of the middle cerebral artery. A relatively small part of aneurysms is localized
in the system of vertebral and basilar arteries. Women have more aneurysms than men.
The question of the origin of saccular aneurysms, which constitute the overwhelming most aneurysms remain largely
open. According to a number of authors, the formation of aneurysms is based on defects
development of the vascular system of the brain, another, less numerous, group researchers emphasize the role of atherosclerosis and hypertension as the main causes of saccular aneurysms.
The concept of traumatic genesis of cerebral aneurysms is proposed
M.V. Kopylov, who believes that at the time of injury, the pressure in
arteries of the brain. When exposed to such a hemohydraulic shock,
damage to the arterial wall with the subsequent development of an aneurysm.
A small part of the aneurysms develops in connection with the entry into the artery of the brain
infected emboli. These are the so-called mycotic aneurysms,
are characterized by a predominant location on the convexital surface
brain. They most often develop in young people with prolonged
septic endocarditis. In the origin of large spherical and S-shaped
aneurysms undoubtedly the leading role is played by atherosclerosis.
Not all aneurysms cause clinical symptoms. Most of
aneurysms are an accidental finding during postmortem examination.
Aneurysms are found in people of different ages - from infancy to

senile.
Other causes of subarachnoid hemorrhage include
atherosclerotic and hypertensive changes in blood vessels, primary and
metastatic, inflammatory diseases, uremia, blood diseases.
Clinic. Usually, subarachnoid hemorrhage develops suddenly, without
any precursors. Only in a small part of patients before hemorrhage
there are symptoms due to the presence of an aneurysm: limited pain in
the frontal-orbital region, paresis of the cranial nerves (more often the oculomotor nerve).
A ruptured aneurysm can occur at the time of physical or emotional
voltage.
The first symptom of subarachnoid hemorrhage is sudden acute headache.
pain, which the patients themselves define as a "blow", as a feeling of "spreading into head of hot liquid ". At the first moment, the pain may be local in nature (in forehead, back of the head), then becomes spilled. In the future, the patient may appear pain in the neck, back and legs.
Almost simultaneously with the headache, nausea and repeated vomiting occur.
Following an attack of headache, loss of consciousness may occur. In mild cases
it lasts 10-20 minutes, in severe cases - unconsciousness continues for
many hours and even days. At the time of the rupture of the aneurysm or soon after there are epileptic seizures.
For hemorrhages from arterial aneurysms, rapid

development of the meningeal symptom complex. Examination of the patient reveals stiff neck muscles, Kernig and Brudzinski symptoms, photophobia, general hyperesthesia. Only in the most seriously ill patients with inhibition of reflex activity meningeal symptoms may be absent.
A common symptom accompanying subarachnoid hemorrhage is
mental disorder. The degree of mental disorder can vary from
slight confusion, disorientation to severe psychosis. Often after
hemorrhages, psychomotor agitation is observed or disorders of the
crumples characteristic of Korsakov's syndrome.
As a reaction to the outflow of blood into the intrathecal space, as well as in
as a result of irritation of the hypothalamic region in the acute period,
an increase in body temperature to 38-39 ° C, changes in the blood in the form of moderate
leukocytosis and shift of the leukocyte formula to the left. Along with this, in many patients, not
suffering from hypertension, there is an increase in blood pressure. V
severe cases with massive hemorrhages, severe disorders are observed
vital functions - cardiovascular activity and respiration.
In the acute stage of subarachnoid hemorrhage, a number of symptoms are due to a rapid increase in intracranial pressure (headache, vomiting). Enhancement intracranial pressure and the resulting obstruction of venous outflow lead to

the development of congestion in the fundus. In addition to varicose veins and swelling of the nipples
optic nerves, retinal hemorrhages may be detected.
In a large percentage of cases with subarachnoid hemorrhage,
also paresis of cranial nerves and symptoms of focal brain damage. Defeat skull-
nerves in patients with spontaneous subarachnoid hemorrhage can be
recognize pathognomonic for rupture of basal arterial aneurysms. Often
there is an isolated paresis of the oculomotor nerve that occurs at the time
ruptured aneurysm or soon after. In the vast majority of cases, isolating
bathroom unilateral lesion of the oculomotor nerve is observed when
hemorrhage from an aneurysm located at the origin of the posterior
connecting artery from the internal carotid.
Hemorrhage from the aneurysm of the internal carotid and anterior connective
arteries near the optic nerves and optic chiasm are relatively common
accompanied by visual impairment. Dysfunction of other cranial nerves
observed less often.
There are two main causes of cranial nerve damage in patients with
arterial aneurysms. First, direct compression of the nerve by an aneurysm
and, secondly, hemorrhage into the nerve and its sheaths at the time of rupture of the aneurysm with
the subsequent formation of connective tissue perineural adhesions.
In the acute stage of hemorrhage from a ruptured arterial aneurysm in
many patients develop symptoms of focal brain damage: paresis of the limbs,
sensitivity disorders, speech disorders, etc. The occurrence of these symptoms
is most often due to concomitant cerebral hemorrhage or local ischemia
brain caused by arterial spasm.
To study the clinical manifestations of arterial spasm at rupture
arterial aneurysms, pathological changes in the brain caused by
spasm, is currently a lot of work. Based on the angiographic data,
the most pronounced spasm of the arteries is localized near the aneurysm, but in many

cases, you can find a spasm of arteries located at a distance from it.
The duration of spastic contraction of the arteries most often does not exceed 2-4 weeks.
Disruption of blood circulation in the brain caused by a spasm can lead to both
limited ischemic brain damage and extensive cerebral infarction, which
observed in the most severe cases. It is suggested that
the resulting spasm of acute ischemia of the brain stem is the most
the likely cause of a number of severe symptoms accompanying a ruptured aneurysm,
such as loss of consciousness, impaired breathing and cardiac activity.
Of interest is the fact that arterial spasm can be
cause not only cerebral ischemia near the ruptured aneurysm, but also a distant
hemispheric lesion. So, with aneurysms of the anterior communicating artery, often
it is possible to detect local symptoms caused by impaired blood circulation in the

seine of the anterior cerebral arteries - paresis of the legs, mental changes in the "frontal"
character, praxis defects. Spasm of the middle cerebral artery leads to paresis of the opposite limbs, impaired sensitivity in them and aphatic
phenomena.
The causes of arterial spasm in ruptured arterial aneurysms have been studied not enough. It is suggested that such
factors such as damage to the artery wall and its segmental nervous apparatus toxic decomposition products of blood corpuscles.
Prognosis of intracranial hemorrhage caused by arterial rupture
aneurysms, very unfavorable. In most cases, the matter is not limited to single hemorrhage. Repeated hemorrhages from aneurysms arising
more often during the first 2-4 weeks, the course is especially difficult. They are more often observed
paresis, paralysis, and the mortality rate is approximately double (40-50%) than with primary hemorrhage.
Diagnostics. The clinic of subarachnoid hemorrhage is well studied, and in
in typical cases, the diagnosis is straightforward. However, in a number of cases in the onset of the disease, when the meningeal symptom complex has not yet fully developed
and symptoms such as vomiting, headache, increased
body temperature, the diagnosis of acute food
toxicoinfection, another infectious disease. With a relatively soft
the gradual development of the syndrome of subarachnoid hemorrhage occurs suspicion of cerebrospinal meningitis. Diagnostic difficulties in most
cases can be easily treated with CT, MRI, or lumbar puncture.
The diagnosis of subarachnoid hemorrhage is considered proven if
blood in the cerebrospinal fluid. If you suspect a subarachnoid
hemorrhage and the absence of positive CT and MRI data is urgently needed examine the cerebrospinal fluid to avoid therapeutically
wrong actions.
In the first days after subarachnoid hemorrhage, cerebrospinal
the liquid is more or less intensely colored with blood. However, the macroscopic analysis is not sufficient to confirm the diagnosis. To exclude
artifact pathway blood admixture, the taken fluid is recommended to be centrifuged. In the liquid obtained after centrifugation with subarachnoid blood
the effusion is determined by xanthochromia. Diagnosis of subarachnoid hemorrhage in
the first hours of the disease can be confirmed by the presence of leached erythrocytes with

microscopic examination of cerebrospinal fluid. A day or more after subarachnoid hemorrhages in the cerebrospinal fluid appear macrophages and lymphocytic cytosis. With a sufficiently profuse hemorrhage, CT is diagnostically significant.
Definitive diagnosis of cerebral artery aneurysm, determination of its exact

localization, shape and size are possible only with the help of angiography.
Treatment. Includes conservative and surgical methods depending on the reasons that caused intrathecal bleeding.
It is generally accepted adherence to strict bed rest for 6-8 weeks.
The duration of this period is due to the fact that the vast majority of repeated hemorrhages from aneurysms occur within 1.5-2 months after the first. Besides, a significant period is required for the formation of strong connective tissue adhesions near the ruptured aneurysm.
Due to a sharp increase in fibrinolysis of cerebrospinal fluid, it is shown aminocaproic acid 20 to 30 g daily for the first 3-6 weeks.
Because even the slightest stress or excitement can cause increase in blood pressure and provoke repeated hemorrhage, the use of sedatives or antipsychotics is necessary. The purpose of these drugs in the acute period of hemorrhage is all the more shown that many patients, those who have had hemorrhage from aneurysms are agitated. It is important to control bowel function.
Since aneurysm rupture is often accompanied by an increase in arterial pressure, it is advisable to prescribe means that normalize its level. Excess decrease in the dangerous development of cerebral infarction due to arterial spasm. Insofar as subarachnoid hemorrhage is often accompanied by spasm of the arteries of the brain, there is a need to use drugs that prevent spasm and improving collateral circulation.
For the prevention or treatment of spasms of intracranial arteries, which can complicate subarachnoid hemorrhage, the drug of choice is nimodipine. Start with intravenous drip infusion: within 1 hour at 15 mcg / kg, then in case good tolerance - 30 mcg / kg in 1 hour. Infusion, if necessary, can should continue around the clock, the volume of perfusion is not less than 1000 ml. After 5-14 days pass for taking nimodipine orally: for 7 days, 60 mg every 4 hours, regardless of food (daily dose - 360 mg).
It is undesirable to combine nimodipine with other calcium ion blockers (nifedipine, verapamil) and nephrotoxic drugs (furosemide, antibiotics aminoglycoside and cephalosporin series).
Nimodipine helps to reduce neurological deficits caused by ischemia.
Also indicated is therapy aimed at combating cerebral edema and intracranial hypertension. For this purpose, glycerol and dexamethasone parenterally. The attitude towards repeated lumbar punctures is ambiguous. Many authors do not recommend them due to the potential risk of provoking repeated blood lesions or dislocation of the brain stem.
A radical method of treating aneurysms is surgical. Term and indications to surgery are determined depending on the results of angiography and the patient's condition. In the presence of a large, accessible aneurysm and

a satisfactory condition of the patient is indicated for the operation on the 1st day. Another variant

provides for surgical intervention to avoid the acute period subarachnoid hemorrhage.

Ischemic stroke (cerebral infarction)

Etiology. Among the diseases leading to the development of cerebral infarction, the first places belong to arterial hypertension and atherosclerosis, they are often combined with diabetes mellitus. Another reason is rheumatism, which can there is a cardiogenic embolism of cerebral vessels. Among other diseases that can lead to ischemic stroke, arteritis should be mentioned infectious and infectious-allergic nature, blood diseases (erythremia, leukemia, angiopathy, fibromuscular dysplasia, etc.). The most important risk factors ischemic stroke are cardiac diseases - ischemic disease heart, myocardial infarction, arrhythmias, etc.

Pathogenesis. Cerebral infarction is formed mainly due to reasons causing a local deficit of cerebral blood flow. These include the first turn of stenosis and occlusion of extra- and intracranial vessels, embolism. In the presence of complete blockage of the vessel (extracranial, intracranial or intracerebral) cerebral infarction may not develop if well developed collateral circulation and, which is especially important if the collateral network is fast turned on after the development of significant stenosis or vessel occlusion. With the development of stenosis of extracranial or intracerebral vessels, conditions are created for local ischemia of the brain substance, if the arterial pressure. Its fall can be caused by myocardial infarction, bleeding and other reasons. In addition, with vascular stenosis, conditions are created for turbulent movement of blood, which contributes to the adhesion of the shaped elements blood - erythrocytes and platelets, and the formation of cell aggregates - microemboli, able to close the lumen of small vessels and cause the cessation of blood flow to the corresponding part of the brain. In addition, high blood pressure 200/100 mm Hg and above is regarded as an unfavorable factor contributing to constant microtraumatization of the inner lining of the artery and separation of embolic fragments from stenotic sites. In addition to thrombosis, cardiogenic and arterio-arterial embolism, hemodynamic factors, in the development of cerebral infarction play a certain role

the reaction of the vascular system of the brain and blood cells to a deficiency of the cerebral
blood circulation, as well as the energy requirements of the brain tissue. Vascular reaction
brain systems to reduce regional cerebral blood flow are different. So, in some observations, ischemia is replaced by excess blood flow, leading to
filtration perifocal edema, in others - the ischemic zone is surrounded
dilated vessels, but not filled with blood (the phenomenon of unrecovered
blood flow).
The mechanism of such a different reaction of cerebral vessels in response to ischemia to the end
unclear. Perhaps it depends on the varying degrees of hypoxia and changing in connection with this
hydrodynamic properties of blood. If in the case of maximum vasodilation with developing regional edema that occurs after ischemia, one can think of
disruption of the normal autoregulatory mechanisms of the cerebral vessels themselves in the zone
local ischemia, the phenomenon of unrecovered blood flow cannot be explained only the reaction of the vessels of the brain. It is known that capillary blood flow depends on aggregation

properties of erythrocytes and platelets to change their shape when walking through narrow
capillaries, as well as from the viscosity of the blood. Red blood cells of the size exceeding the diameter of narrow capillaries (the diameter of erythrocytes is 7 μm, and the diameter of narrow
capillaries - 5 microns), in conditions of normal blood circulation, they easily change their shape,
“Deformed” and, like an amoeba, move along the capillary bed. In patients with vascular diseases, the deformability of erythrocytes decreases, they become
more “tough”. An even greater decrease in their deformability develops in
hypoxic foci of any localization. Significant reduction in deformability
erythrocytes does not allow them to pass through a capillary whose diameter is less than the diameter
erythrocyte. Consequently, an increase in the "hardness" of erythrocytes, as well as an increase in
aggregation of platelets and erythrocytes in the area of local cerebral ischemia can be one
of the main factors that impede blood flow through the dilated vessels when
the phenomenon of unrecovered blood flow. Thus, even if the cause is eliminated, which caused local cerebral ischemia, regional edema that develops after ischemia or non-filling of blood vessels can lead to disruption of normal
the vital activity of neurons and the development of cerebral infarction.
In the pathogenesis of cerebral ischemia, the dominant role in the quality

occlusive factors are played by thrombosis and embolism of cerebral vessels, differentiation
which causes significant difficulties not only in the clinic, but also at autopsy. Thrombus
is often a substrate that embolizes the arteries of the brain, which is reflected in the widespread use of the term "thromboembolism". The formation of a blood clot in the affected
the vessel is promoted, according to currently existing views, additional or “thrombotic realizing” factors. The main ones follow
consider the change in the functional properties of platelets and the activity of biologically
active monoamines, imbalance of coagulant and anticoagulant blood factors, as well as changes in hemodynamic parameters.
Changes in the functional state of platelets - increasing their aggregation and adhesive ability, as well as inhibition of disaggregation clearly are observed already in the initial stage of atherosclerosis. Aggregation increases even more,
as atherosclerosis progresses, with the appearance of transient cerebral disorders circulation and stenosing processes in the main arteries of the head.
Local tendency to aggregation, gluing, and then disintegration (viscose metamorphosis) of platelets in the area where the inner membrane is damaged, due to the fact that it is in this place that a kind of chain reaction develops, depending on a number
humoral and hemodynamic factors.
In the simultaneous processes of thrombus formation and thrombolysis involved a complex multistage complex of coagulant and anticoagulant factors. Depending on the final prevalence of one of them in the affected
a segment of the vessel, different degrees and outcomes of thrombus formation can occur. Sometimes
the process is limited to stenosis, partial deposition of platelets and fibrin, and sometimes dense conglomerates are formed, completely obstructing the lumen of the vessel and
gradually increasing along its length.
Increase, "thrombus growth", in addition to blood hypercoagulability, contribute slowing down of blood flow and turbulent, vortex movements of red blood cells proximal
blockages.
Relative hypocoagulation contributes to a looser structure of blood clots, which

may be a prerequisite for the formation of cell embolism and, apparently, will is a factor that plays an essential role in the spontaneous recanalization of thrombi.
Thrombotic lesions of the aorta, extracranial and large intracranial arteries are one of the sources of arterio-arterial cerebral vascular embolism.
Heart damage can also serve as a source of cerebral embolism.

Cardiogenic embolism develops as a result of the separation of parietal thrombi and warts.
clear layers with valvular heart disease, with recurrent endocarditis, with congenital heart defects and during operations for congenital or acquired heart defects. Cardiogenic embolism of cerebral vessels can develop with myocardial infarction, with acute postinfarction heart aneurysms with the formation parietal thrombi, with prolapse of mitral valves and with artificial valves.
The immediate cause of cardiogenic embolism is often various lesions causing atrial fibrillation and decreased contractile the ability of the heart.
Of great importance in the development of ischemic strokes are negative psychoemotional factors leading to an increased secretion of catecholamines, which are powerful activators of platelet aggregation.
If in healthy individuals catecholamines are only stimulators of aggregation platelets, then in patients with atherosclerosis with a rapid release of catecholamines into vascular bed, they cause a sharply increased aggregation of platelets and their destruction, which leads to a significant release of serotonin and intravascular thrombus formation.
Hyperproduction catecholamines many researchers considered as a link between chronic or acute emotional stress and atherosclerotic changes in the vascular wall.
A lively discussion is underway when discussing the role of cerebral vasospasm in genesis ischemic stroke and transient disorders of cerebral circulation.
The possibility of spasm (decrease in the lumen) of the cerebral arteries and arterioles is not is in doubt. Under normal conditions, angiospasm is a common compensatory reaction in response to increased cerebral blood flow, increased oxygen content and reduced concentration of carbonic acid in the blood. From humoral factors spasmodic effect possess catecholamines, adrenocorticotropic hormone, platelet breakdown products, including prostaglandins.
Thus, spasm of cerebral vessels is an important link in the autoregulation system. cerebral circulation. However, the role of cerebrovascular spasms in the origin clinically significant cerebral ischemia is highly questionable. Until recently

no direct evidence has been obtained for the role of neurogenic spasm in the development of cerebral infarction.
An exception, as already noted, may be a spasm that complicates the course subarachnoid hemorrhage. The mechanism of the spasm may cause a small heart attack in rare cases with migraine (migraine-stroke).
Thus, local ischemia in atherosclerosis of the main or intracranial vessels, leading to the formation of cerebral infarction, occurs due to several factors: thrombosis, embolism, hemorheological and hemodynamic factors, when a drop in blood pressure due to different causes ischemia in the areas of adjacent blood vessels of the brain. The reason development of cerebral infarction, the phenomenon of extracerebral or intracerebral

robbery.
Pathophysiology and biochemistry of ischemic stroke. The brain receives the energy needed is almost exclusively from the oxidation of glucose. One a glucose molecule upon oxidation gives 38 molecules of energetically rich phosphate compounds in the form of ATP - adenosine triphosphate. The oxygen content in the arterial blood only 2 times higher oxygen consumption by the brain. Therefore, with a decrease blood flow below 50% in the brain, hypoxia occurs, which leads to a decrease energy balance of the brain. The brain compensates for energy deficits by activation of anaerobic glycolysis. The content of glucose in arterial blood exceeds many times the need for it of the brain substance. Therefore, with a decrease there is no blood flow deficiency in glucose, and the brain tries to compensate energy deficit by anaerobic breakdown of glucose. However, completely to compensate for the energy deficit due to anaerobic glycolysis is not seems possible, since anaerobic breakdown of one glucose molecule gives only 2 ATP molecules. In addition, with anaerobic glycolysis, a large the amount of lactic acid, as a result of which acidosis develops in the tissue, which aggravates further metabolic disturbances.
The next link in metabolic disorders during hypoxia is disorders lipid metabolism with activation of peroxidation processes. With ischemia, processes lipid peroxidation have their own characteristics, consisting in the absence incoming antioxidants from outside (normally inhibiting the process of peroxide lening) against the background of endogenous growth in the level of prooxidants, a decrease in the activity of enzymes, destroying peroxides, and disrupting the utilization of endogenous antioxidants. Activation of phospholipid peroxidation of cell membranes and subcellular structures of the neuron leads to the formation of free radicals, which provide cytotoxic effect and causing impaired protein metabolism and synthesis transmitters.
With a decrease in blood flow and a decrease in perfusion pressure below 50-70 mm Hg - autoregulatory barrier - autoregulation of cerebral blood flow disappears and

ischemia develops. This increases the concentration of CO_2 in the blood, which causes
vasodilation. Acidosis, which develops in
hypoxic brain tissue. With a further decrease in blood flow to 30 ml per 100 g brain substance per minute, the EEG slowly begins to change, with a decrease in blood flow to
15 ml / 100 g per minute, a change in potential develops, and with a blood flow of 10-12 ml / 100 g in
a minute the normal ion pump activity disappears. Potassium rushes from cells, and sodium penetrates into the cell. Terminal depolarization sets in and the excitability of the membrane disappears. Calcium enters the cells, and develops cytotoxic cerebral edema.
Arising from these violations
microcirculatory disorders aggravate the decrease in blood flow. Cell structure collapses, cerebral edema increases, and after cytotoxic edema, after b h, vasogenic edema develops. Reactivity of cerebral vessels in ischemic
the area disappears, vasoparalysis develops, due to acidosis.
Cytotoxic edema is a condition in which primary damage and
accumulation of fluid occurs in the parenchymal cells of the brain - neurons and glial elements. With vasogenic edema, the primary defect is localized in the blood-brain barrier and the accumulation of fluid occurs mainly in the extracellular
exact space.

Edema that develops as a result of hypoxic ischemic brain damage,
is cytotoxic, since neuronal and
glial elements, and the violation of the blood-brain barrier occurs later.
With both forms of edema in the brain tissue, the fluid content increases,
only in the cytotoxic form, edema develops due to impaired metabolism substances in the cell, and with vasogenic edema, sweating from the bloodstream increases
whey proteins. With a decrease in blood flow to 10 ml / 100 g per minute, cellular the membrane is depolarized, sodium ions from the extracellular space rush into the cell and at the same time the sodium content in the extracellular fluid decreases. Because
the sodium content in the blood remains constant, a gradient occurs, whereby sodium and water from the bloodstream rushes into the intercellular fluid and into the brain cells.
In addition, for the development of cytotoxic cerebral edema, osmotic
brain tissue pressure. Since anaerobic glycolysis, which develops during ischemia brain, leads to the formation of osmotically active compounds, then as a result an osmotic gradient is created with circulating blood. The created osmotic
the gradient leads to the release of water from the blood into the brain tissue.
Cytotoxic edema reaches its maximum within 1 hour of ischemia. At
this, if blood flow is restored, the ionic membrane is reactivated. If

cytotoxic edema lasts more than 6 hours, reactivation does not occur and develops vasogenic edema. Vasogenic edema is caused by increased vascular permeability the walls of cerebral vessels for large molecules and is characterized by an increase the content of serum proteins in brain tissue as a result of
blood-brain barrier. The development of vasogenic edema is accompanied by an increase in brain volume and, as a result, an increase in ICP. Compression possible upper parts of the brain stem crocheted gyrus of the hippocampus, pinching the trunk in
tentorial foramen, as well as entrapment of the cerebellum in the greater occipital hole, which is accompanied by compression of the medulla oblongata, which is the cause
death of a number of patients with ischemic stroke.
The reverse development of vasogenic edema is much slower than cytotoxic, so how whey proteins must first undergo resorption, which when
edematous tissue should go to the ventricular system and then to the cerebrospinal liquid. And this depends on the pressure gradient in the brain tissue, which independently
can provide the yield of whey proteins only to a small extent.
Difficulty resorption of edema is also associated with the fact that a significant part of serum
proteins are inside the cell.
All of the above indicates that the restoration of the cell to activity after vasogenic edema is extremely difficult and slow.
Clinic. The development of cerebral infarction is often preceded by transient disorders
cerebral circulation, developing in the same vascular basin as a heart attack brain.
Ischemic stroke can develop at any time of the day. Often he
occurs during sleep or immediately after sleep. In some observations, it is noted the development of ischemic stroke after exercise, taking a hot bath,
drinking alcohol, after a heavy meal. Development is often observed ischemic stroke after psychoemotional overstrain.
Ischemic stroke is characterized by the gradual development of focal neurological symptoms. An increase in focal symptoms of cerebral infarction is often

occurs in the time interval from several tens of minutes to several hours and rarely within 2-3 days. At the same time, a flickering type of development can sometimes be observed.
stroke, when the severity of neurological disorders either increases or weakens.
In addition to the slow, gradual development of focal symptoms of cerebral infarction, in
1/3 of observations are acute, lightning-fast development of symptoms, and immediately
the most pronounced. Apoplectiform development of ischemic stroke

observed with acute blockage of a large artery, it is characteristic of embolic stroke. In this case, as a rule, focal symptoms are combined with a pronounced in different
degree of cerebral symptoms.
Much less often there is pseudotumorous development, when focal
heart attack symptoms worsen over several weeks, due to
an increase in the occlusive process in the vessels of the brain.
A characteristic feature of ischemic stroke is the predominance of
focal symptoms over cerebral. General cerebral symptoms - headache, vomiting, confusion - are most often observed with apoplectiform development and may increase with increasing cerebral edema accompanying a massive heart attack brain. Focal symptoms depend on the location of the cerebral infarction. Based clinical symptom complex can be judged on the size, localization of the heart attack and
the vascular basin, in the course of which cerebral infarction develops.
Most often, cerebral infarctions develop in the pool of internal carotid
arteries. The frequency of heart attacks in this area exceeds the frequency of heart attacks in
vertebrobasilar basin 5-6 times.
Most often, heart attacks develop in the basin of the middle cerebral artery, which feeds a significant part of the brain, the basal nuclei, the inner capsule (knee, anterior parts of the hind leg and partly the anterior leg), most of the temporal lobes, the precentral area and the area of the middle and lower parts of the pre- and post-
central gyri, opercular region, semi-oval center, a significant part
parietal region and angular gyrus.
In case of impaired blood circulation in the vessels of the vertebrobasilar basin systemic dizziness, nystagmus, coordination disorders,
impaired hearing and vision, autonomic disorders, sometimes coma develops, tetraplegia, impaired breathing and cardiac activity, diffuse hypotension, or hormetonia appears.
A heart attack with occlusion of the vertebral artery leads to the development of symptoms with
sides of the medulla oblongata, cerebellum and partly the cervical spinal cord.
Infarction foci with blockage of the vertebral artery can develop in the middle brain, in the area of adjacent blood circulation of two vascular systems - the vertebral and
carotid basins. Heart attacks in the adjacent circulation area are more common for occlusion of the extracranial segment of the vertebral artery.
In the early days of ischemic stroke, the temperature reaction and significant changes in peripheral blood, as a rule, are not observed. However, with extensive heart attacks with severe cerebral edema and the effect on the brain stem may develop hyperthermia and leukocytosis, as well as an increase in the content of glucose and urea in
peripheral blood.
From the coagulation and anticoagulant system of the blood in the majority

in patients with ischemic stroke, there is a shift in the direction of blood hypercoagulation.

Increased fibrinogen, prothrombin, plasma heparin tolerance, appearance fibrinogen B with reduced or normal fibrinolytic activity usually expressed in the first 2 weeks of the disease.
In some cases, it is possible to change the hypercoagulability of the blood by its hypocoagulation.
At the same time, there is a sudden drop in the content of fibrinogen in the blood, a decrease in prothrombin index and a decrease in the number of platelets. Listed plasma (fibrinogen, prothrombin) and cellular coagulation factors are consumed for intravascular coagulation, and blood devoid of clotting factors penetrates through the vascular wall, causing hemorrhagic complications. Are developing spread non-essential hemorrhagic complications resulting from intravascular coagulation (consumption syndrome, thrombohemorrhagic syndrome, disseminated intravascular coagulation - disseminated intravascular coagulation).
In patients with ischemic stroke in the acute period, there is a high platelet aggregation and adhesion. At the highest numbers, it keeps in within 10-14 days, returning to subnormal values on the 30th day of stroke.
Cerebrospinal fluid is usually clear with normal protein content and cellular elements. Possibly a slight increase in fluid protein and lymphocytic cytosis in infarction foci bordering the cerebrospinal fluid space and causing reactive changes in the ependyma of the ventricles and meninges.
Diagnostics. In most cases, it is not possible to diagnose a stroke great difficulties. Acute development of focal and cerebral symptoms in patients mature and old age, suffering from atherosclerosis or hypertensive disease, as well as in young people against the background of systemic vascular disease or blood diseases, as a rule, indicate an acute violation of cerebral circulation - stroke or transient cerebrovascular accident.
Echoencephalography for ischemic stroke usually shows no displacement the middle M-echo. However, with extensive heart attacks due to the development of edema and displacement the brain stem can show displacements of the M-echo already by the end of the 1st day from the moment of the development of a stroke. Doppler ultrasonography makes it possible to detect occlusion and stenosis of the main arteries of the head. Important information is given by angiography, which allows to identify in patients with cerebral infarction the presence or absence of occlusive and stenosing processes in extra- and intracranial vessels of the brain, as well as the functioning pathways of collateral blood circulation.

EEG reveals hemispheric asymmetry and can be detected, the focus
pathological activity.
Characteristic changes in cerebral infarction are found on CT, while
a focus of reduced density of the brain parenchyma in the infarction zone is revealed and
peri-infarction area, in contrast to the changes found in hemorrhage in
the brain, when there are opposite changes, is a focus of increased density.
MRI is equally important for the diagnosis of stroke, allowing
visualize focal brain lesions in the first hours of the disease, the foci are very
small size and, which is very important, well reveals changes in the brain stem.
Distinguish cerebral infarction from hemorrhagic stroke in a number of observations
presents great difficulties. However, determining the nature of the stroke
necessary for differentiated treatment.
It should be recognized that there are no individual symptoms that are strictly
pathognomonic for

hemorrhage or cerebral infarction. The sudden onset of a stroke, characteristic of
hemorrhage, is often observed with occlusion of a large vessel, embolic
the nature of the stroke. And at the same time, with hemorrhages, especially diapedesic
character, the symptoms of damage to the brain substance can increase gradually over
several hours.
It is well known that a stroke develops during sleep, usually ischemic,
however, although much less frequently, cerebral hemorrhages can occur at night.
Severe cerebral symptoms, so characteristic of hemorrhage in
brain, often observed with extensive cerebral infarctions, accompanied by edema.
Arterial hypertension is often complicated by hemorrhage, but the accompanying
hypertension atherosclerosis is often the cause of the development of ischemic stroke,
often observed in patients with atherosclerosis with arterial
hypertension. High blood pressure figures at the time of a stroke are not always
should be considered as the cause of a stroke, it can also be a reaction of the stem
the vasomotor center for its ischemia.
From the foregoing, it can be seen that individual symptoms have a relative
diagnostic value for determining the nature of the stroke. However, certain
combinations of symptoms with additional research data allow correct
recognize the nature of a stroke in the vast majority of cases. So, development
stroke during sleep or immediately after sleep, against the background of cardiac
pathology, especially resistance
driven by a disturbance in the rhythm of cardiac activity, a history of myocardial infarction,
moderate arterial hypertension is characteristic of ischemic stroke. Development
stroke during the day, especially at the time of emotional stress in a patient suffering
hypertension, its onset with acute headache, repeated vomiting,
impairment of consciousness is most characteristic of cerebral hemorrhage.
From additional research methods, it must be remembered that leukocytosis with

a shift to the left, which appeared on the 1st day of stroke, an increase in body temperature and
the presence of blood in the cerebrospinal fluid or its xanthochromia, displacement of the M-echo and
the presence of a focus of increased density on CT scan indicates a hemorrhagic nature
stroke. In approximately 20% of cases, macroscopic cerebrospinal
the fluid with hemorrhage is transparent and colorless. However, microscopic
a study in this category of patients makes it possible to detect erythrocytes, and with the help of
the spectrophotometer detects blood pigments (bilirubin, oxy- and methemoglobin). At
ischemic stroke cerebrospinal fluid colorless, transparent, possibly
increased protein content. Coagulogram data, as well as radiographic
studies do not provide reliable information in favor of hemorrhagic or
ischemic stroke. Angiography should be recognized as an informative method,
however, arteriographic studies, due to the risk of complications, recommend
to carry out in cases where there is a feasibility of surgical treatment.
Currently, the most informative in determining the nature
stroke have CT and MRI data, allowing to differentiate cerebral infarction from hemorrhage in the brain.
Small hematomas located in the area of subcortical formations and in white
substance of the cerebral hemispheres, without CT, as a rule, are not diagnosed. If at cerebral infarction CT scan shows clearer data in the later stages of the heart attack, then
with cerebral hemorrhage, the most informative picture is observed in the acute stage hemorrhages.

Hemorrhagic infarction is one of the most difficult to diagnose
states. With hemorrhagic infarction, ischemic
defeat, and then, or simultaneously with it, hemorrhage appears in the infarction zone.
Most often, hemorrhagic infarctions are localized in the gray matter - the cortex
large brain, basal nuclei and thalamus. Development of hemorrhage in the focus of ischemia
most researchers associate with a sudden increase in blood flow in
ischemic zone due to the rapid flow of blood into this area through collaterals.
Hemorrhagic changes often occur with extensive, rapidly forming
cerebral infarction.
According to the development of the disease and clinical manifestations of hemorrhagic infarction
resembles a cerebral hemorrhage of the type of hematoma or diapedetic impregnation.
Therefore, hemorrhagic infarction is diagnosed during life much less often than on autopsy.
Treatment. Any acute violation of cerebral circulation requires
immediate medical attention, since the fate of the patient and the size

neurological defect depend on reasonable and appropriate therapeutic
interventions in the early stages of the disease. Emergency therapy provided by the team
specialized care, early hospitalization and intensive complex therapy
in the hospital are the main factors determining the effectiveness
treatment.
The treatment system is built on the basis of those ideas about the pathogenesis of the cerebral
heart attacks that have developed in recent years. It includes a complex of medical
emergency care measures for patients with cerebral stroke, regardless of its
nature (undifferentiated care) and differential treatment
ischemic stroke.
Undifferentiated therapy is aimed at normalizing vital
functions - respiration and cardiac activity, providing general and cerebral
hemodynamics, as well as affecting tissue metabolism.
Undifferentiated therapy includes the fight against cerebral edema, hyperthermia,
as well as prevention of stroke complications. First of all, it is necessary to ensure that
free airway patency with the help of special suction, application
oral and nasal ducts, rubbing the patient's mouth, holding
lower jaw. In the event that measures aimed at eliminating the blockage
airways are ineffective, intubate and
tracheostomy. The same interventions are used for sudden respiratory arrest,
progressive breathing disorder, with bulbar and pseudobulbar
symptomatology when there is a danger of aspiration. When breathing suddenly stops and
the absence of a ventilator at this time will certainly
use mouth-to-mouth, mouth-to-nose artificial respiration.
With the development of pulmonary edema, cardiotonic drugs are recommended: 1 ml is injected
0.06% solution of korglikon or 0.5 ml of 0.05% solution of strophanthin
intravenously.
Strofantin acts immediately, but is eliminated from the body within 2 hours.
Therefore, it is better
recommend digoxin injections. In addition to the above remedies, it is recommended
inhalation of oxygen with alcohol vapors through an oxygen inhaler or Bobrov apparatus
in order to reduce foaming in the alveoli. Oxygen is supplied through
30% alcohol solution. Inhalations of alcohol vapors continue for 20-30 minutes, then
repeated after a 30-minute break. Raise the head end of the bed with

the purpose of giving an exalted position to the patient. Intramuscularly appoint 80-120
mg of lasix, 2 ml of a 1% solution of diphenhydramine, 0.5-1 ml of a 0.1% solution of atropine. When falling
blood pressure requires the introduction of cardiotonic and vasotonic

funds.
It is necessary to control the water-electrolyte balance and CBS, especially in patients with impaired consciousness. The volume of parenterally administered fluid is usually not
exceeds 2000-2500 ml per day. Introduce isotonic sodium chloride solution, solution Ringer-Locke, 5% glucose solution. Due to the fact that violations of CBS often resist are driven by a deficiency of potassium, it is necessary to use potassium chloride inside in an amount
up to 3-5 g per day under the control of its concentration in the blood. To eliminate acidosis along
with an increase in pulmonary ventilation and oxygen therapy, as well as activities, increase cardiac output, intravenously inject 4-5% solution of bicarbonate sodium.
Decongestant therapy for ischemic stroke is aimed at removing
perifocal and cytotoxic edema. The pathogenetic method of treatment should influence various mechanisms of formation of cerebral edema, such as metabolic shifts in cells that cause swelling of brain tissue - cytotoxic edema, on
violation of the blood-brain barrier and the spread of edema in the brain parenchyma. In this regard, corticosteroids are very encouraging.
Steroids are widely used in the treatment of both diffuse and localized edema brain. The most commonly used drug is dexamethasone. This drug is 6 times more active
prednisolone, does not cause sodium and water retention and potassium loss. Dexamethasone
reduces the permeability of the blood-brain barrier and thus prevents the spread of edema into the brain parenchyma, and its membrane-stabilizing and metabolic action helps to eliminate cytotoxic edema.
Dexamethasone is usually prescribed at a dose of 16-20 mg intravenously per day (4 mg every 6
h) the first 2-3 days, followed by a gradual decrease in dosage - 12-8-4 mg.
The effect of the use of dexamethasone clinically in cases with severe edema the brain is detected in ischemic stroke after 18-24 hours. Therefore, parenteral the introduction of dexamethasone is carried out for the first 2-3 days, and then, if necessary
Continuation of the drug administration switch to oral administration. Duration the use of dexamethasone depends on the severity of the cerebral infarction and the concomitant
localized or diffuse cerebral edema that is well controlled by CT head or MRI.
Osmotic diuretics have a therapeutic effect in cerebral edema.
The introduction of osmotic diuretics induces plasma hyperosmolarity and creates osmotic gradient in relation to the extracellular space of the brain.
Removal of water along the osmotic gradient from the brain tissue is possible only when
intact blood-brain barrier when semipermeable membranes
limit the diffusion of the hyperosmolar solution into the hypoosmolar space.

In case of violation of the blood-brain barrier, a substance with an osmotic
by action, penetrating into the brain tissue, it can cause a reverse gradient. Shown, that a decrease in volume during osmotherapy occurs in intact brain tissue, while
while the affected area does not change. However, by lowering ICP, osmodiuretics increase the volume of cerebral blood flow. In addition, osmodiuretics increase the volume
plasma, reduce blood viscosity and peripheral arterial resistance, which
manifested by an increase in blood pressure and stroke volume. As a result

improves blood circulation in areas adjacent to the area of edema.
One of the most common osmotic diuretics used to treat
treatment of cerebral edema, is mannitol, a hexahydric alcohol, widespread
in the plant kingdom. The therapeutic dose of the drug is 0.5-1 g / kg of the patient's weight.
However, after the introduction of mannitol, there is a "recoil syndrome", that is, an increase in
intracranial pressure above the initial level by an average of 30-40% after 45 minutes - 2
h after administration of the drug, which is accompanied by a reduction in cerebral blood flow and
increased blood viscosity.
The recoil phenomenon can serve as an additional mechanism of ischemia and contribute to the worsening of neurological deficits. Therefore, it is more appropriate the use of glycerin. Glycerin has low toxicity, and the degree of its toxicity depends from the route of administration. When administered subcutaneously, even relatively small doses
glycerol hemolysis and hemoglobinuria occur. If glycerin is injected intravenously into
a solution containing sodium chloride or ascorbate, hemolytic and other
toxic effects of the drug are not observed, provided that the concentration
solution does not exceed 10%.
Most authors who use glycerin for the treatment of cerebral edema,
noted the absence of a “kickback effect”. An important advantage of glycerin over with mannitol is its moderate effect on systemic blood pressure - with
internal use, this indicator does not change. Glycerin increases urine output, but this is not the defining condition for its anti-edematous action. Relatively
the weak diuretic effect of the drug is due to the fact that, on the one hand, part of it is included in the metabolism of renal cells, and on the other hand, reabsorption increases
osmotically free water. Consequently, glycerin, providing a strong
anti-edema effect on the central nervous system, does not cause significant loss of water and electrolytes
with urine. In addition, it has been shown that glycerol is capable of inhibiting hydroxyl radicals

and inhibits platelet aggregation. Glycerin is injected intravenously in the form of 10%
solution of 400-800 ml per day for the first 2-3 days. Possible glycerin injection orally and rectally.
The expediency of using furosemide (lasix) in the treatment of edema is questionable brain with strokes. Furosemide causes profuse natriuresis and chloruresis. but there is no noticeable decrease in intracranial pressure. A drug
causes a negative water balance and increases hemoconcentration, this leads to a decrease in microcirculation and a violation of the rheological properties of blood, which in its
turn contributes to the development of cerebral edema and an increase in neurological deciphitis.
It is important to control the hematocrit values and maintain them at the level of 30-35,
and avoid hyperglycemia, which worsens the outcome of ischemic stroke.
In order to prevent pneumonia, it is necessary from the 1st day of stroke turn the patient in bed every 2 hours, perform percussion massage
chest. If you suspect the development of pneumonia, antibiotics are prescribed.
Control over the action of the bladder and intestines is necessary. With urinary retention
shows catheterization 2 times a day with bladder flushing with antiseptic means.
To avoid bedsores, you need to monitor the condition of the skin, wipe body with camphor alcohol. It is advisable to use anti-decubitus mattresses.
In the treatment of ischemic stroke, it is necessary to ensure improvement cerebral circulation. It is advisable to use cardiotonic drugs,

increasing the stroke and minute volume of the heart, as well as drugs that increase venous outflow of blood from the cranial cavity (strophanthin, korglikon, digoxin).
The question of vasodilating drugs for
improving cerebral circulation and increasing local cerebral blood flow in stroke. Most researchers express a point of view about the inexpediency and even the dangers of using vasodilators in ischemic stroke, since they can cause an intracerebral “steal” phenomenon. These assumptions are based on the fact that when studying the state of the vascular system of the brain in an experiment
and the clinic received data on the absence of vascular reaction in the ischemic area or their paradoxical reaction. Therefore, conventional cerebral vasodilators (papaverine, carbonic acid) lead to the expansion of only unaffected vessels, "Pulling" the blood from the heart attack area. This phenomenon is called the phenomenon of intracerebral steal.
With the recommendations of some clinicians - use vasodilators remedies in cases where it is assumed as the main cause of a heart attack angiospasm, it is difficult to agree, since angiospasm as a cause of cerebral infarction

unlikely, but for spasm of cerebral vessels after rupture of aneurysm papaverine and others
vasoactive drugs do not work, except maybe nimodipine.
The effectiveness of nimodipia in cerebral infarction is being studied, although given its
mechanism of action, you can recommend it in a dose of 60 mg 3 - 4 times orally.
To improve collateral circulation and microcirculatory link in
the zone of cerebral infarction, it is advisable to prescribe drugs that reduce
blood viscosity and blood corpuscles that reduce the aggregation properties. WITH
for this purpose, intravenous administration of low molecular weight dextran is used -
reopolyglucin at a dose of 400 ml. The drug is administered drip, with a frequency of
30 drops per minute,
daily for 3-7 days. The introduction of rheopolyglucin improves the local cerebral
blood flow leads to anti-thrombogenic action. Rheopolyglucin effect
maximum in arterioles, precapillaries, capillaries. As a result of a sharp decrease in ar-
regression of erythrocytes and platelets decreases the intensity of microcirculatory
sedimentation syndrome, expressed by low perfusion pressure, slowing down
blood flow, increased blood viscosity, aggregation and stasis of blood elements,
the formation of blood clots. Due to the well-known hypervolemic and hypertensive
actions with the introduction of rheopolyglucin need to control blood pressure,
which must be kept at least as usual for the patient. In case of systemic hypotension
it is necessary to eliminate it. The antiaggregatory effect of rheopolyglucin is
observed in
for 6-8 hours, therefore it is advisable in the intervals between the introduction of
rheopolyglucin and
after stopping dextran injections, recommend oral administration
antiplatelet agents: acetylsalicylic acid, bromcamphor, trental, etc.
The antiaggregatory effect can be achieved by intravenous administration of 2.4%
aminophylline solution, as well as 2% papaverine solution at a dose of 2
ml. Derivatives
aminophylline, as well as papaverine, have an inhibitory effect on
phosphodiesterase, whereby cyclic adenosine monophosphate (cAMP) accumulates
in the blood cells, which is one of the powerful inhibitors
aggregation.
Regular oral intake of blood cell aggregation inhibitors
after five days or a week of using them in the form of injections, it allows for
to achieve reliable prevention of thrombus formation during the entire acute period of
stroke

in the entire vascular system as a whole. Taking aggregation inhibitors (acetylsalicylic
acid, tiklid, pentoxifylline) it is advisable to continue for the next 2 years, as
the most dangerous period for the development of repeated strokes.
The use of antiplatelet agents has made it possible in recent years to a large extent
reduce the use of anticoagulants requiring for their use
regular

control
indicators
systems
hemostasis.
V
cases
the same
thromboembolic syndrome for the prevention of re-embolism and
progressive thrombosis (stroke in progress), the use of anticoagulants is indicated.
In this case, it is necessary to make sure of the ischemic nature of the stroke by
performing CT or MRI
heads.
Anticoagulant therapy begins with the administration of a direct anticoagulant
actions - heparin. Heparin is prescribed in a dose of 10,000-5,000 IU 4-6 times a day
for 3-
6 days, injected intravenously, intramuscularly or under the skin of the
abdomen. Better to introduce
heparin continuously through an internal catheter. Heparin treatment should be carried
out under
control of blood clotting time. Elongation is considered optimal.
coagulation 2.5 times. Continue anticoagulant therapy if necessary
Indirect anticoagulants are prescribed 3 days before the abolition of heparin - phenylin
according to
0.03 g 2-3 times a day or syncumar in a daily dose of 8-16 mg, while reducing
daily dose of heparin per 5000 IU.
Treatment with indirect anticoagulants is carried out under control
prothrombin index, which should not be reduced by more than 50%. Appointment
heparin, at least in small doses (10,000 IU per day), is also indicated for
prevention of disseminated intravascular coagulation and the formation of
phlebothrombosis, dangerous development
pulmonary embolism.
For thrombolytic effect, streptase, streptodecase, urokinase are used,
tissue plasminogen activator. Their appointment is justified in the first hours from the
beginning.
stroke. They must be administered at the same time as heparin. Given the high
frequency
hemorrhagic complications with systemic administration of thrombolytics, the most
appropriate
cotton method of their use is local thrombolysis with
catheter under X-ray control to the site of thrombosis. Efficiency and
the safety of this method of urgent therapy for cerebral infarction is still being studied.
In the complex treatment of ischemic stroke, in recent years, they have used
agents that increase the resistance of brain structures to hypoxia. Expediency
the use of antihypoxants is determined by the fact that metabolic disorders in
cells of the brain parenchyma usually precede in time a gross lesion
the brain in the form of edema and, moreover, are one of the leading causes of edema.

The feasibility of prescribing antihypoxic therapy is also determined by the fact that in conditions of acute cerebral circulation deficiency and disorganization
metabolism is more beneficial to temporarily reduce the energy requirements of the brain and thereby
to some extent increase its resistance to hypoxia.
Accordingly, it is considered justified to prescribe drugs that have
inhibitory effect on energy balance. For this purpose, apply
antipyretic drugs, regional hypothermia, and new
synthetic drugs that have an inhibitory effect on enzymatic
processes and metabolism in the brain, as well as substances that increase energy production in
condition of hypoxia. Such substances include methylphenazine derivatives and piracetam (nootropil). This group of antihypoxants has a positive effect

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on the processes of tissue respiration, phosphorylation and glycolysis.
It should not be forgotten that a number of patients with ischemic stroke with intact with consciousness and criticism, they are seriously worried about their
disease. Therefore, this category
patients are shown psychotropic drugs. It is necessary to carry out prevention phlebothrombosis. For this purpose, shin bandaging, passive and active gymnastics are shown.
nastika, as well as venotonic drugs.
Course and forecast. The greatest severity of the condition in patients with ischemic a stroke is observed in the first 2-3 days. Then there is a period of stabilization or improvement when patients begin to decrease the severity of symptoms.
At the same time, the rate of restoration of impaired functions can be different. At good rapid development of collateral circulation is possible
restoration of functions on the very first day of stroke, but more often recovery begins in a few days. In some patients, the lost functions begin to recover.
stop in a few weeks. Known and severe course of stroke with stance
stabilization of symptoms.
Mortality in ischemic stroke is 20-25%. In patients
have had an ischemic stroke, there is a risk of recurrent disorders
cerebral circulation. Recurrent strokes develop more often in the first 3 years after stroke. The first year is considered the most dangerous, and very rarely repeated strokes
develop 5-10 years after the first stroke.
Prevention.
Includes a set of activities aimed
on
systematic monitoring of the health status of patients with cardiovascular
diseases, the organization of the patient's work and rest regime, nutrition, health improvement
working and living conditions, timely treatment of cardiovascular diseases, arterial hypertension.

To risk factors for cerebral infarction, except for atherosclerosis, arterial hypertension and heart disease include emotional stress, diabetic disorders
exchange and hypercholesterolemia, obesity, physical inactivity,
smoking. Application
oral contraception is also a developmental threat
thrombotic cerebral infarction and is considered a risk factor.
A great risk of developing a stroke is represented by transient ischemic
attacks representing a specific prodromal syndrome of cerebral infarction.
For this category of patients, in order to prevent stroke, either long-term
antiplatelet therapy, or surgery on the great vessels
heads.

Lacunar stroke

The first description of lacunar infarctions in the inner capsule belongs to
French neurologist P. Marie. The syndrome he described was characterized by post-foamy growth of neurological disorders in the form of several episodes
hemiparesis with the subsequent development of dysarthria, ataxia, pseudobulbar paralysis,
dysfunction of the pelvic organs and gait with light steps - marche a petit pas
(lacunar state of the brain).
A characteristic feature of lacunar heart attacks is a favorable outcome with
partial or complete recovery. Small gaps are meant
deeply located heart attacks due to primary lesion
penetrating branches of the cerebral arteries. With the greatest frequency, gaps arise in

basal nuclei, inner capsule, pons of the brain, sometimes in the white matter of the convolutions
brain. Their location is due to the highest frequency of arterial lesions.
lenticular nucleus and striatum, thalamoperforating branches of the posterior cerebral artery and paramedian branches of the basilar artery. The defeat of small arterial branches is associated with the development of microateromas in them, lipohyalinosis,
fibrinoid necrosis. The possibility of microembolism as a cause is also not excluded.
development of lacunar infarction.

Chronic cerebrovascular accident

Initial manifestations of cerebrovascular insufficiency

In recent years, in clinical practice, the term initial
manifestations of cerebral circulatory insufficiency (NPNMK). The basis for
of this diagnosis ”are the following complaints: headache, non-systemic
dizziness, noise in the head, memory impairment, decreased mental performance.
Typically, these symptoms occur during a period of significant emotional and
mental stress, requiring a significant increase in cerebral blood circulation
niya. If two or more of these symptoms recur frequently or persist for a long time (at least 3 last months) and there are no signs of organic
lesions of the nervous system, a presumptive diagnosis of NPNMK is made.

The etiological factors of these conditions in most cases are atherosclerosis, atherosclerosis with arterial hypertension - the so-called hypertensive atherosclerosis, hypertension. It must be borne in mind that symptoms similar to those that occur with PNLUI may be due to not only vascular, but also other factors - chronic infection, neurosis, an allergic condition, malignant tumor and other reasons for which a differential diagnosis should be made. With suspected vascular genesis the described violations require instrumental and laboratory confirmation defeat cardiovascular systems (ECG, doppler ultrasonography, rheological characteristics, lipid spectrum, etc.).

Treatment and prevention of progression of chronic cerebrovascular failure or stroke include the elimination or compensation of the underlying vascular disease, normalization of work and rest, sanatorium treatment, symptomatic therapy. Dispensary observation is necessary for patients.

Encephalopathy

Under dyscirculatory encephalopathy implied slowly progressive lack of blood supply, leading to the development multiple small focal necrosis of the brain tissue, manifested gradually growing defects in brain function. Consideration should be given to the possibility of subclinical ongoing acute cerebral dyscirculatory disorders, including small lacunar infarctions that form characteristic of discirculatory encephalopathy symptomatology.

Etiology.

By the main etiological reasons allocate atherosclerotic, hypertensive, mixed, venous discirculatory encephalopathy, although, by definition, other causes are possible leading to

chronic vascular cerebral insufficiency (rheumatism, vasculitis, other etiology, blood diseases, etc.). In practice, the greatest etiological significance in development of discirculatory encephalopathy have atherosclerosis, arterial hypertension and their combination.

Pathogenesis. The pathogenesis is due to insufficient cerebral circulation in its relatively stable form or in the form of repeated short-term episodes discirculation. As a result of pathological changes in the vascular wall, developing due to arterial hypertension, atherosclerosis, vasculitis, etc., there is a violation of the autoregulation of cerebral circulation, there is an increasing dependence on the state of systemic hemodynamics, which also turns out to be unstable due to the same diseases of the cardiovascular system. To this are added disorders of neurogenic regulation of systemic and cerebral hemodynamics. The aging process of the nervous, respiratory, cardiovascular systems, which also leads to the development or intensification of hypoxia brain. By itself, brain hypoxia leads to further damage to the mechanisms autoregulation of cerebral circulation.

Important in the development and course of dyscirculatory encephalopathy have rheological and biochemical characteristics of blood, which are in large as a reflection of the underlying disease. Disorders of microcirculation are found, due to an increase in the functional activity of platelets, blood viscosity, latent signs of disseminated intravascular coagulation. Most significant rheological changes are observed in patients with types III and IV hyperlipoproteinemia.

Clinic and diagnostics. The clinical picture of discirculatory encephalopathy, as it follows from the definition, has a progressive development, and on the basis of the severity of symptoms is divided into three stages. Stage I is dominated by subjective disorders in the form of headaches and a feeling of heaviness in the head, common weakness, increased fatigue, emotional lability, memory loss and attention, dizziness, often of a non-systemic nature, instability when walking, sleep disorders. In contrast to the initial manifestations of cerebral insufficiency circulation, these phenomena are accompanied, although mild, but rather persistent objective disorders in the form of anisoreflexia, discoordinating phenomena, oculomotor failure, symptoms of oral automatism, decrease memory and asthenia. At this stage, as a rule, formation does not occur yet. distinct neurological syndromes (except asthenic) and with adequate therapy it is possible to reduce the severity or eliminate individual symptoms and diseases in general. This brings stage I discirculatory encephalopathy closer to initial manifestations of cerebral circulation insufficiency, which gives the basis for some authors to combine them into the group of “initial forms of vascular cerebral failure ”. There is a certain sense in this, since therapeutic measures are similar.

The set of complaints of patients with stage II discirculatory encephalopathy is similar to such in stage I, although the frequency of memory impairments, work capacity, and headache is increasing.

surroundings, instability when walking, a little less often there is a complaint about

headache and other manifestations of the asthenic symptom complex. At the same time, one
however, focal symptoms become more pronounced in the form of revitalization of reflexes
oral automatism, central failure of the facial (VII) and sublingual

(CP) nerves, coordination and oculomotor disorders, pyramidal
insufficiency, amiostatic syndrome, increased mnestic-intellectual
and emotional (faintheartedness) disturbances. At this stage, it is already possible
isolate
certain
dominant
neurological
syndromes
-
discoordinator, pyramidal, amiostatic, dysmnestic, etc., which
can significantly reduce the professional and social adaptation of patients.
In stage III of discirculatory encephalopathy, the volume of complaints decreases, which
combined with a decrease in patients' criticism of their condition, although complaints persist
memory loss, instability when walking, noise and heaviness in the head, sleep disturbance.
Objective neurological ratios are much more pronounced.
structures in the form of fairly clear and significant disco-ordinatorial, pyramidal,
pseudobulbar, amiostatic, psychoorganic syndromes. More often observed
There are paroxysmal states - falls, fainting, epileptic seizures.
This stage differs from the previous one and what, as a rule, is observed in patients
several quite pronounced syndromes. Patients with stage III dyscirculatory
encephalopathies turn out to be inoperative, their social and household
adaptation, i.e. dementia develops.
CT characteristics are changing from normal values or
minimal atrophic signs in stage I to more pronounced small focal
changes in brain matter and atrophic (external and internal) manifestations in II
stages to sharply marked cortical atrophy and hydrocephalus with multiple
hypodense foci in the hemispheres - in stage III. It should be noted, however, that this
the relationship is statistical in nature and full correspondence between the CT picture and
clinic is not always observed. High representation of atrophic changes
brain, according to CT, in patients with a clinical picture of dyscirculatory
encephalopathy, especially in stage II and III, in the absence of a clear connection with the severity
changes in the cardiovascular system, indicates the possibility of a combined
development of primary degenerative-atrophic processes in the brain and changes,
due to chronic discirculation. The same circumstance requires

the most clear argumentation of pathogenetically significant vascular pathology in diagnosis of discirculatory encephalopathy in the elderly and especially senile age.

In the picture of discirculatory encephalopathy, several main syndromes - cephalgic, vestibular-atactic, pyramidal, amiostatic, pseudobulbar, paroxysmal, psychopathological.

Treatment.

Treatment for discirculatory encephalopathy should include impacts aimed at the underlying disease, against which the discirculatory encephalopathy (atherosclerosis, arterial hypertension, vasculitis and etc.), elimination of neurological and psychopathological syndromes, improvement cerebral circulation, metabolic processes. Considering that most patients with discirculatory encephalopathy are in the elderly and senile age, it is necessary to carry out adequate therapy of concomitant somatic diseases, the course of which, by physiogenic or psychogenic mechanisms, has a significant effect on the neuropsychic status of patients. Highly undesirable excessive decrease in blood pressure, which can lead to acute or chronic decompensation of cerebral circulation. Treatment of atherosclerosis, arteries mental hypertension, concomitant somatic diseases should be carried out

together with the relevant specialists. Part of the therapy aimed at possible improvement of cerebral circulation, as well as therapy for neuro-mental manifestations of discirculatory encephalopathy, is within the competence neurologists and psychiatrists.

SPINAL CORD VASCULAR DISEASES

Spinal circulation disorders

There are a large number of etiological factors leading to vascular spinal cord injury. The vast majority of patients develop ischemic brain damage (myeloischemia) and only occasionally occur hemorrhages (hematomyelia). There are three main groups of reasons myeloischemia. The first group includes lesions of the cardiovascular systems: congenital (malformations of the spinal vessels - arteriovenous aneurysms, arterial aneurysms, varicose veins; coarctation of the aorta, hypoplasia of spinal cord vessels) and acquired (atherosclerosis of the branches of the aorta, arteritis, phlebitis, thrombosis and embolism, circulatory failure due to cardiac weakness activity with myocardial infarction, atrial fibrillation, with hypertensive disease). All these reasons are found in about 20% of all patients with myeloischemia. The second group (about 75%) consists of processes leading to compression vessels from the outside: compression of the aorta and its branches by tumors and tumor-like formations of the chest and abdominal cavity (enlarged due to pregnancy uterus, packs of lymph nodes with lymphogranulomatosis, tuberculosis, metastases tumors, etc.), (pressure of the radicular-spinal arteries and radicular veins, hernias intervertebral disc (the most common type of compression), epi- and subdural

tumor, vertebral fragments in trauma, epidural inflammatory
infiltrate thickened by soft and arachnoid membranes (including calcareous
plaques in them), etc.
The third group includes iatrogenic factors, when myeloischemia
occurs as a complication of surgical interventions (radiculotomy with intersection
radicular-spinal artery, prolonged clamping or plastic aorta with
switching off the intercostal or lumbar arteries, surgery in the paravertebral
area, etc.) and injection manipulations (epidural blockade, spinal
anesthesia, etc.).
Naturally, one patient may have a combination of different
pathogenetic factors, for example atherosclerosis of the aorta and its branches and vertebral
osteochondrosis.
An important role in the pathogenesis of myeloischemia is played by: 1) the state of collateral
circulation, which depends on the variant of vascularization of the spinal cord (with the main type, the number of blood inflows is small and shutting off even one channel is not
compensated by adjacent radicular-spinal basins); 2) variety
etiological factors; 3) the state of general hemodynamics. When you turn off a large radicular-spinal artery at the level of its main trunk (before dividing into
ascending and descending branches) ischemia in certain areas of the spinal cord can develop according to the principle of the "steal" syndrome (pathogenic compensation cerebrospinal circulation).
Hemorrhagic spinal stroke due to rupture

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arteriovenous (arterial) aneurysm or injury to the spine and spinal cord.
If the venous circulation is impaired, the development of hemorrhagic
spinal cord infarction.
Despite the significant number and variety of etiological and
pathogenetic factors, vascular lesions of the spinal cord have enough
a clear overall clinical picture.
Disorders of the cerebrospinal circulation can be divided into transient,
acute and chronic.

Transient disorders of cerebrospinal circulation

Transient (transient) myeloischemia includes only such forms, with
which focal spinal Symptoms disappear before 24 hours. Clinic of disorders
blood flow in the upper and lower arterial basin is different. Meet
the following options.

Falling drop syndrome. It is characterized by the sudden onset of a sharp
weakness of the arms and legs when turning the head quickly to the side or throwing the head back
posteriorly, when the patient suddenly falls, consciousness does not change, pain is often felt

in the back of the head and neck. After 2-3 minutes, the strength in the limbs is restored. Deep
reflexes on the hands are reduced or absent, on the legs they are evenly revitalized, maybe
evoke the Babinsky reflex from both sides. In a few tens of minutes
the neurological status is normalized. Paroxysms of tetraparesis are renewed with repeated sharp turns of the head. They arise with pronounced degenerative degenerative lesions of the cervical spine, sometimes in combination with atherosclerosis of the vertebral arteries and are associated with transient ischemia of the cervical segments
department.

Unterharnsheidt's syndrome.

Characterized by sudden onset
paralysis of the upper and lower extremities with switching off of consciousness for a short time
(2-3 minutes). When the consciousness of these patients is restored, they cannot move any
hands or feet. However, after another 3-5 minutes, voluntary movements resume.
in the limbs, patients experience general weakness and fear of repeated paroxysm. V interictal period there is a feeling of heaviness and dull pain in the cervical spine spine. Usually, such paroxysms occur with sharp turns of the head, as well as
in patients with falling drop syndrome, in contrast to the latter, to weakness
limbs are joined by loss of consciousness. Unterharnscheidt syndrome occurs with cervical osteochondrosis and is associated with ischemia of not only cervical thickening, but also cerebral
trunk (pool of vertebral arteries).

Myelogenous intermittent claudication. Comes with the onset of ischemia in lower arterial basin of the spinal cord. Clinically, it is characterized by the fact that with prolonged walking or other physical activity, weakness and feeling appear numbness in the legs, sometimes this is accompanied by an imperative urge to emitting and defecation. After a short rest 5-10 minutes) these phenomena
pass and the patient can continue to walk. In neurological status during the period weakness of the legs, a decrease in knee and Achilles reflexes, hypotension gastrocnemius muscles, fascicular twitching in them, sometimes Babinsky's reflex. However, these neurological disorders resolve quickly. Pulsation of the lower arteries limbs does not change. The patient is forced to stop because of weakness, and not from
sharp pain in the legs. These criteria are used to differentiate between

peripheral intermittent claudication (with atherosclerosis or endarteritis
lower extremities). Patients with myelogenous intermittent claudication often note the twisting of the feet when walking. This variant of lameness occurs due to lumbar osteochondrosis with the effect of a disc herniation on one of the lower radicular
spinal arteries (with a low variant of the Adamkevich artery or in the presence of

lower additional radicular-spinal artery). Therefore, myelogenous
lameness in such patients develops against the background of lumbodynia or lumboischialgia. Less often
the cause of this syndrome is spinal vasculitis or abdominal atherosclerosis aorta and its branches.

Caudogenic intermittent claudication. Occurs with congenital or acquired narrowing of the spinal canal at the level of the lumbar spine. Such patients when walking first appear painful paresthesias in the form of tingling, crawling creeps, numbness in the distal legs, soon these sensations
rise to the inguinal folds, spread to the perineum and genitals.
Overcoming these sensations and trying to continue walking in patients develops and weakness in the legs. After a short rest, such disorders disappear. Out of walking like
as a rule, there are no signs of damage to the cauda equina. It should be noted that myelogenous
Claudication is a clinical rarity, while caudogenic claudication is an everyday reality. Rarely, there is a combination of myelogenous and caudogenic intermittent lameness, then both paresthesias and weakness of the legs are pronounced.

Ischemic spinal stroke

Men and women get sick with the same frequency between the ages of 30 and 70 and older. During the course of the disease, several stages can be distinguished: 1) the stage of precursors
(distant and near), 2) stage of stroke development, 3) stage of reverse development, 4) the stage of residual phenomena (if complete recovery has not come).
Paroxysms are the harbingers of ischemic spinal stroke
transient spinal disorders (myelogenous, caudogenic or combined
intermittent claudication, transient pain and paresthesia in the spine or branching projections of certain spinal roots, dysfunction
pelvic organs).
The rate of onset of a stroke is different - from sudden (with embolism or traumatic compression of the vessels supplying the spinal cord) up to several hours and
even a day.
It has already been mentioned that spinal infarction is often preceded by pain in spine or along the individual roots.
The cessation or significant abatement of this pain after the development of myeloischemia. This occurs due to an interruption in the passage of pain impulses along
sensitive conductors at the level of the spinal cord ischemic focus.

Clinic. The clinic of ischemic spinal stroke is very polymorphic and
depends on the prevalence of ischemia both along the length and across the dorsal brain.
It should be noted that angiotopic diagnosis is always fraught with difficulties.
The reason for this is the large individual variability in the distribution of radicular arteries. As a result, even an accurate topical diagnosis of the lesion does not provide sufficient

criteria for determining which of the arteries has lost patency. Recognition
it is complicated, in addition, by the dynamism of clinical manifestations. It requires study

individual variants of the clinical picture, based on the prevalence of ischemia as along the length and across the spinal cord.

Diagnostics. When recognizing spinal cord ischemia,
precursors in the form of myelogenous intermittent claudication or transient paresis, dyscalgia, radiculalgia, etc. Great importance is attached to the rate of development of the disease
(acute or subacute), no signs of inflammation or acute compression
spinal cord. According to the clinical picture, one can, at least presumably, think about
defeat of one or another vascular basin. More often this applies to the front
spinal artery and its anterior radicular-spinal
trunks of different levels of the spinal cord.

According to the peculiarities of the clinical picture, it is possible to carry out differential
diagnostics
between
arterial
and
venous
radiculomyeloischemias.

Arterial radiculomyeloischemia develops acutely or subacutely usually after period of precursors and against the background of a hyperalgic crisis with the subsequent termination
or a significant reduction in pain. Symptom complexes of defeat are characteristic predominantly of the ventral half of the spinal cord diameter.

Additional research methods are of great help in diagnostics.
Occlusion of the aorta and its branches in some cases can be confirmed using angio-graphy. It should be noted that areas of atherosclerotic calcification of the wall
the aorta and its aneurysms are often found on lateral spondylograms. Defined
Comprehensive information about the condition of the spinal cord can be obtained with CT and MRI.

Compression factors in patients are specified using spondylography and pyelography. It is necessary to speak about complicity of ischemia in cases of detection of inconsistency
the consequences of the level of lesion of the spine with the border of the medullary focus, determined
according to clinical data. Research into cerebrospinal
liquids. Absence of subarachnoid block and normal composition
cerebrospinal fluid is present in a third of patients. However, often in the acute phase spinal stroke, there are significant changes in the fluid (increased

protein content from 0.6 to 2-3 g / l and even higher, sometimes this is combined with moderate
pleocytosis - from 30 to 150 cells in 1 mm). Especially altered cerebrospinal
the liquid happens when the venous outflow is disturbed. In the acute stage of a stroke, it is possible
detection of a block of the subarachnoid space, which is caused by edema and thickening of the spinal cord itself. With repeated lumbar punctures after 1-2 weeks cerebrospinal fluid usually normalizes and subarachnoid block
no.
Electrophysiological research methods can identify a violation
innervation of even those muscles in which there are signs of damage in the usual clinical
the study fails to find (sufficient muscle strength, no change in their tone).
Treatment. They are carried out in several directions. The first of them provides for improvement of local blood circulation due to the inclusion of collaterals and increase volumetric blood flow velocity. For this purpose, vasodilators are prescribed, improving cardiovascular activity, decongestants, antiplatelet agents, antihypoxants.
The second direction of therapeutic measures provides for the elimination occlusive process. With the thromboembolic nature of spinal stroke anticoagulants are prescribed (heparin, phenylin). Small doses of them are shown for

preventing the development of thrombus formation. In cases of compression-vascular spinal disorders, therapeutic tactics are aimed at eliminating the compression.
Most often we are talking about a discogenic disease. These patients are used as orthopedic (tight bed, spinal traction, wearing a corset, muscle massage
along the spine, exercise therapy), and physiotherapy. With absence
success from drug and orthopedic treatment establish indications for
surgical intervention. It is also performed for patients with intra- and extravertebral tumors. The choice of the method and the scope of the operation is decided on an individual basis.
together with neurosurgeons. Special tactics of therapeutic measures
adhere to lesions of the aorta (coarctation, atherosclerotic aneurysm).
It should be determined in conjunction with the surgeons.
All patients, including in the postoperative period, are prescribed nootropic preparations, vitamins, absorbents and biostimulants (aloe, vitreous
body, lidaza, etc.).
Regardless of the applied method of pathogenetic treatment in all cases
spinal infarction requires particularly careful patient care in order to
prevention of pressure ulcers and urosepsis.
The outcome of spinal cord infarction differs depending on the causative cause and method of treatment. In more than half of the patients, it is possible to obtain a favorable therapeutic
peptic effect: practical recovery and improvement with moderate
residual phenomena. Fatal outcome is observed with spinal stroke on

the soil of a malignant tumor, dissecting hematoma of the aorta and during the development
concomitant diseases and complications in the form of myocardial infarction, urosepsis.
As for the labor prognosis, it depends on the severity and prevalence neurological disorders in the residual stage. In addressing issues of employability The following expert criteria are accepted. First group of disability determined by patients with tetraparaplegia or deep paresis in combination with dysfunction of the pelvic organs, trophic disorders. These sick need outside care. The second group of disability is established by the patient with moderate paresis of the limbs and dysfunction of the pelvic organs. Such patients can do work at home. The third group of disability is assigned patients with mild paresis of the extremities without disorders of the function of the pelvic organs. These
patients need rational employment.

Hemorrhagic spinal stroke (hematomyelia)

Hematomyelia is a hemorrhage in the spinal cord.

Etiology. The most common cause is spinal injury and abnormalities the vascular system of the spinal cord (arteriovenous aneurysms, etc.). Less often hematomyelia develops with hemorrhagic diathesis, infectious vasculitis, etc.
The focus of hemorrhage is usually located in the gray matter of one or a number of adjacent
segments.
The clinical picture depends on the localization of the hematoma focus. Signs of defeat
spinal cord occur acutely at the "moment of injury or after physical overexertion (lifting weights, straining). Peripheral paresis appear corresponding muscle groups and segmental anesthesia. May be violated function of the pelvic organs.
With hematomyelia in the area of cervical enzymes to peripheral paresis of the hands and

central - the legs may be joined by a violation of the respiratory function (muscle paresis
diaphragm), which aggravates the course of the disease.
Hematomyelia must be differentiated from spinal cord infarction. With acute the development of a stroke often causes difficulties. Helps in diagnostics detection blood and cerebrospinal fluid. In some cases, spinal
subarachnoid hemorrhage, which initially manifests itself as radicular pain and spinal disorders. Meningeal symptoms soon join
(headache, nausea, muscle and neck stiffness).

Treatment. In the acute period of hematomyelia, treatment is carried out similarly to that
with hemorrhage in the brain. Constantly monitor the condition of the bladder, bedsores are prevented.

Chronic insufficiency of cerebrospinal circulation

Chronic insufficiency of the cerebrospinal circulation first proceeds in the form of transient disorders, but in the future there are signs of persistent, often progressive lesions of various structures of the spinal cord. Sometimes such variant of the clinical picture occurs some time after ischemic spinal stroke. The clinic is characterized by mixed para- or tetraparesis in combined with spotted hypesthesia, dysfunction of the pelvic organs. Sometimes one the syndrome of amyotrophic lateral sclerosis with a long course and jerky progression.

The principles and methods of therapy are similar to those for discirculatory encephalopathy.

DEGENERATIVE DISEASES WITH PREVIOUS IMPAIRMENT COGNITIVE FUNCTIONS

ALZHEIMER'S DISEASE

The disease is named after the famous German pathologist and psychiatrist A. Alzheimer, who described her pathomorphological picture in a patient in 1907, suffering from dementia. In the last 10-15 years, a lot of new information has been received about clinic, diagnosis and pathogenesis of Alzheimer's disease (AD). This form is characterized by breakdown of higher cortical functions, cerebral atrophy and the development of dementia in elderly and old age. Among women, the disease occurs 3-4 times more often, than among men. Genetic studies have made it possible to establish in familial cases dominant type of inheritance. The genes responsible for the development of AD are localized at 21 th and 19 th chromosomes. The average age of onset is 55 years. Duration illness 7-15 years.

Pathogenesis. Studies of neurotransmitter changes in AD reveal a significant decrease in the activity of choline acetyltransferase, acetylcholinesterase and co-retention of acetylcholine in the hippocampus and neocortex, which is associated with the loss of neurons in the Meinert nucleus, from where most of the cholinergic terminals. Similar processes affect noradrenergic and serotonergic system. A decrease in the concentration of glutamate is detected, substance P, somatostatin and corticotropin in the cortex and subcortical nuclei. It is not clear, are the described neurotransmitter disorders the primary factors in pathogenesis of the disease or they are caused by the loss of neurons. Introduction to patients cholinomimetics and acetylcholine precursors significant positive

has no effect on the course of the disease.

Clinic. In elderly and senile people, the disease is mild
course with gradually increasing dementia, and for patients aged 40-50 years characterized by a more intense progression of dementia. The disease begins imperceptibly. Usually, neither patients nor their loved ones can clearly identify this period.
Most often, at first there are memory impairments, mainly short-term;
the aggravation of this defect leads to a decrease in memorization, forgetting the names of loved ones
people, their appearance, names of objects, words (dysnomy). Memory impairment are a reflection of the degenerative-atrophic process in the hippocampus. Largely a mnestic defect is caused by gradually increasing speech disorders:
patients with difficulty find the right words, hardly perceive the meaning of what they read.
Partially due to the violation of spatial gnosiea and praxis, patients cannot
neither read nor write. Behavior becomes stereotyped, stereotyped, indifferent.
Retaining some occupational skills allows patients to maintain
some time at work, despite the development of noticeable manifestations of the disease.
Affective disorders usually join in the later stages of the disease.
Mood instability, irritability, anger,
episodes of psychomotor agitation, followed by apathy. Development is possible hallucinations, delusions (jealousy, damage), although paranoiac disorders do not occur
Often. In the final stage of the disease, there is a revitalization of the reflexes of the oral
automatism, gluttony, elements of the Kluver-Bussy syndrome (hyperphagia, hypersexuality). As already noted, in some cases, it is revealed
amiostatic hypokinetic-rigid syndrome, motor stereotypes are possible.
Epileptic seizures rarely occur.
The diagnosis of AD is based on excluding other possible causes of dementia,
the presence of two or more types of cognitive impairment, the gradual development of the disease
and its constant progression. Although nonspecific, they are essential for establishing the diagnosis of CT and MRI of the head, revealing with an expanded picture of the disease
levania significant atrophic changes in the brain. Single-photon emission CT and positron emission CT scans show biparietal and bitemporal decreases
regional cerebral blood flow and metabolism. The EEG records the deceleration background rhythm, slow activity.
Differential diagnosis with the exclusion of volumetric processes and hydrocephalus performed with clinically similar conditions - Pick's disease, multi-infarction dementia combined with asthma, as well as with other forms of presenile dementia and with

depressive conditions.
Treatment. There is no effective treatment. If possible, patients should stimulate feasible household and social activity, avoid premature and prolonged hospitalization. Given the defectiveness of cholinergic systems of the brain, if necessary, the use of antipsychotics and antidepressants drugs without pronounced anticholinergic properties (nortriptyline, haloperidol). The use of barbiturates is undesirable; if necessary, you can use small doses of benzodiazepia (sibazone, nosepam, etc.). Since at AD also has a monoamine brain defect, then complications are quite possible psychotropic therapy (development of amiostatic syndrome), which requires careful observation of patients and adequate selection of doses of drugs.
Attempts are being made to treat with a cholinomimetic drug - amiridine at a dose of 20-80 mg / day orally or intramuscularly; the effect is being studied

close to physostigmine - a drug - tacrine. According to reports, these funds effective in the relatively early stages of the disease. They can improve state of cognitive functions and slow down the progression of the disease. There are reports of beneficial effect of the calcium channel blocker nimodipine (90-120 mg / day) and large doses of cerebrolysin - 20-30 ml intravenously in isotonic sodium solution chloride for a month every day. The effectiveness of these and other drugs needs further study.

PEAK DISEASE

The disease was first described by the Czech psychiatrist and neuropathologist A. Pik in 1892 Characterized by progressive dementia, early personality breakdown and other symptoms due to relatively local brain atrophy. More often occurs in women. The pathogenesis is not well understood. In a number of patients, autosomal dominant type of inheritance. However, in most cases the hereditary nature of the disease has not been proven. Infectious diseases, injuries, probably only provoke or aggravate the pathological process. Average age the onset of the disease is 55 years old. The duration of the disease is from 5 to 10 years or more.
Clinic. Clinical symptoms are determined by pathomorphological changes in the brain. Dementia develops gradually. The defeat of the frontal and temporal lobes manifested by apathy, hypokinesia, gait disturbance, early enough the emergence of reflexes of oral automatism, grasping reflex, amiostatic-hyperkinetic, dysphasic disorders, turning into unrelated speech with a small set of words. Verbal perseverations are characteristic, palilalia, echolalia, movement stereotypes. The relatively rapid development of these manifestations distinguish Pick's disease from Alzheimer's disease. Also, for starters Pick's disease is also characterized by personality changes associated with localization pathological process. In patients with predominant convexital atrophy

parts of the frontal lobe, spontaneity, apathy appear. At the same time, it is characteristic
extreme distraction to external stimuli with minimal spontaneous
reactions. Predominant atrophy of the basal parts of the frontal cortex is accompanied by
disinhibition of behavior, bulimia, hypersexual behavior, euphoria
(moriey). Development of apraxia and agnostic disorders is possible. As the diseases, mental and neurological disorders are increasing,
amimia, akinesia, mutism. In the final stage, symptoms of pseudobulbar appear paralysis, cachexia, marasmus develops.
Diagnosis is based on the same principles as for Alzheimer's disease.
It is not always easy to differentiate these forms of demetia clinically. They are distinguished
characteristic of dementia, greater "focus" of the lesion (atrophy) and less
the presence of speech and apracto-agnostic disorders with an earlier
personality breakdown in cases of Pick's disease. Objective and reliable analysis is important
symptoms and study of the course of the disease. Differential diagnosis
performed with various atrophic processes associated with dementia
(Alzheimer's disease), Parkinson's disease, Huntington's chorea, frontal tumors
lobes and other causes of frontal dementia. Differential assistance
Methods for diagnosing the form of presenile dementia can be
neuroimaging examination.
Treatment is symptomatic.

INFECTIOUS AND PARASITIC DISEASES OF THE NERVOUS SYSTEM

Infectious diseases of the central nervous system are one of the most common forms of neurological pathology.
In recent decades, thanks to the introduction of new diagnostic technologies and powerful antibacterial and antiviral drugs, significant advances have been made in the treatment of non-
which more recently lethal diseases. At the same time, the range of pathogens has expanded infectious diseases of the nervous system, mixed, atypical forms have become more frequent, which can
create significant diagnostic difficulties.
The spectrum of microorganisms infecting the nervous system is very diverse.

Meningitis

Meningitis is an acute infectious disease with a primary lesion of the arachnoid and
soft membranes of the brain and spinal cord. The causative agents of the disease are most often bacteria and viruses, less often fungi, protozoa, mycoplasmas, helminths, rickettsia and amoeba. Bacteria
or viruses enter the body through the entrance gate - most often this is the nasopharyngeal cavity and intestines.
From here they enter the bloodstream (stage of viremia or bacteremia) and then hematogenously are introduced into the membranes of the brain.
Depending on the nature of the inflammatory process in the membranes of the brain and the resulting
The composition of the cerebrospinal fluid distinguishes between purulent and serous meningitis. Purulent meningitis
as a rule, caused by bacteria, and lymphocytic - by viruses. Exception to this rule
are tuberculous, syphilitic and some other serous meningitis.

According to the rate of development, acute, subacute and chronic meningitis are distinguished. With some
forms, in particular with meningococcal meningitis, the fulminant development of the disease is possible.
Morphological changes in meningitis are mainly observed in the arachnoid and soft
membranes, but the process may also involve ependyma and choroid plexuses of the ventricles of the brain.
The most conspicuous manifestation of damage to the meninges is headache. How
as a rule, it has a bursting character, it is felt in the whole head or mainly in the frontal,
temporal or occipital parts. The onset of headache is due to irritation of the brain
sheaths innervated by the branches of the trigeminal (V) and vagus (X) nerves, as well as
sympathetic fibers. The receptors of the membranes can be influenced by inflammatory
the process, toxic substances released during it and an increase in ICP. The latter is due to the fact
that inflammation of the choroid plexuses of the ventricles of the brain is accompanied by an increase in production
cerebrospinal fluid with a simultaneous violation of the processes of its absorption.
Vomiting is also a direct result of this. It arises as a result of direct or
mediated irritation of the trigger zone located in the bottom of the rhomboid fossa.
With most meningitis, general infectious symptoms are found: malaise,
increased irritability, facial flushing, increased body temperature, shift in blood count
to the left. Bradycardia often develops, then turns into tachycardia, followed by arrhythmia.
Breathing quickens, in severe cases it becomes pathological (Cheyne's breathing -
Stokes).
The most important objective signs of meningitis are the so-called meningeal
the symptoms that make up the meningeal syndrome. Rigidity is noted in almost all cases.
muscles of the neck. It is found during passive flexion of the patient's head. Due to spasm
the extensor muscles of the neck fail to fully bring the chin to the sternum. Pronounced degree
rigidity is accompanied by throwing the head backward, pressing it into the pillow
(opisthotonus). The stiffness of the neck muscles is often combined with the stiffness of the muscles of the back and limbs.
To identify the latter, use the method proposed by the St. Petersburg physician V.M. Kernig.
When examining Kernig's symptom, the patient lies on his back, his legs are alternately bent at first
at right angles in the hip and knee joints, and then try to passively straighten in
knee joint. In case of irritation of the membranes of the brain, the leg can only be straightened to a certain
angle due to the resulting tension in the muscles of the back of the thigh. It is the presence of the flexor
contractures ”(by definition V.M. Kernig) is a fundamental difference between this phenomenon and
pain symptom Lasegue. Somewhat less often than the stiffness of the muscles of the neck and
Kernig's symptom, are detected
symptoms of Brudzinsky. Brudzinsky's upper symptom is flexion of the thigh and lower leg during
an attempt to tilt the head of a patient lying on his back anteriorly. Lower Brudzinsky symptom
manifested by flexion of the contralateral leg when examining Kernig's symptom on the other leg.
For young children, the symptom of Lesage hanging is characteristic: when raising a child by

the armpits of his legs are involuntarily pulled up to the stomach and remain in
this position. The severity of meningeal symptoms can vary. With lightning speed
flowing forms of meningeal syndrome may be weak or absent altogether.
It should be borne in mind that meningeal symptoms may be mild in the elderly.
age.
In infants, bulging of the anterior fontanelle is determined, due to
increased ICP.
General hyperesthesia is an important sign of irritation of the meninges of the brain. Sick

painfully react to any external stimuli - bright light, loud sound, touching
skin.
Focal neurological symptoms in meningitis are highly variable and indicate
involvement in the process of the substance of the brain or spinal cord and the cranial roots
emanating from them
or spinal nerves. More often than others, the oculomotor nerves are affected, which is due to
the predominant localization of the pathological process on the basis of the brain in some types
meningitis (tuberculosis, fungal infections, carcinomatosis, etc.). Other cranial nerves
are affected much less frequently. Sometimes a change in deep reflexes is found, first in
upward and then downward. In some cases, the appearance of pathological
reflexes. The defeat of the membranes of the spinal cord and located near the roots can
cause the occurrence of pain at the spinal level.
Severe purulent meningitis in some cases is accompanied by a sharp
psychomotor agitation, delirium, epileptic seizures.
The identification of meningeal syndrome should in no way be identified with
a diagnosis of meningitis. For example, a bright symptom complex of irritation of the meninges -
a constant companion of subarachnoid hemorrhages, it is often observed in carcinomatosis
meninges and with intracranial hypertension due to volumetric
intracranial processes.
Of decisive importance in the diagnosis of meningitis is the study of the cerebrospinal
liquids. If the patient does not have pronounced signs of increased ICP (stagnant discs
optic nerves), lumbar puncture is completely safe and should be performed when
the slightest suspicion of the possibility of meningitis.
With a lumbar puncture, cerebrospinal fluid usually flows out under an increased
pressure, its appearance is determined by the number of cellular elements. With serous meningitis
the liquid is transparent or slightly opalescent, with purulent - turbid, yellow-green.
Pleocytosis is a characteristic feature of the acute stage of meningitis.
moderately high protein content (cellular-protein dissociation).
With purulent meningitis, neutrophils predominate, with serous meningitis - lymphocytes. Level
pleocytosis and the nature of the formed elements of cerebrospinal fluid to a certain extent
depends on the severity of the inflammatory process and its dynamics during the development of the
disease.
The favorable course of purulent meningitis is characterized by a decrease in the relative amount
neutrophils and an increase in lymphocytes. The combination of high neutrophilic pleocytosis with
a significant increase in the level of protein in the fluid is noted in severe disease and
is regarded as an unfavorable prognostic sign.
For the differential diagnosis of meningitis, research is of great importance.
glucose content in cerebrospinal fluid. A decrease in this indicator is typical for
tuberculous and fungal meningitis. A sharp decrease in glucose content in the CSF is characteristic
for purulent meningitis.
CSF test results - determination of cellular composition, protein and sugar levels - have
decisive for the differential diagnosis and the appointment of an etiotropic
treatment. The final etiological diagnosis is determined using bacteriological and
serological studies.
Agglutination and agglutination reactions widely used in the diagnosis of infectious diseases
complement fixation reactions are of limited use in the diagnosis of meningitis, since
they take a long time to complete. The sowing method has the same disadvantage.
pathogen on nutrient media and determination of its sensitivity to various antibiotics.
However, this study must be carried out in any case, since the
the results are needed in the future to correct the procedure for this patient.
drug therapy.

The most reliable and rapid diagnosis of the etiology of meningitis is provided by

immunological express methods for the study of cerebrospinal fluid - the counter method immunophoresis and the method of fluorescent antibodies. These methods allow you to determine the presence in
liquids of specific antigens - protein components of meningitis pathogens - using
group precipitating sera for several hours. Fluorescent method
antibodies allows you to diagnose both bacterial and viral meningitis and, especially
valuable, mixed bacterial-viral meningitis, the so-called “mixmeningitis”.
A patient with suspected meningitis is subject to mandatory and possibly earlier
hospitalization. It is not recommended to start antibiotic therapy in the prehospital period. Only in severe cases occurring with high body temperature, impaired consciousness, in the presence of signs of infectious toxic shock, it is necessary to inject 3,000,000 units intravenously
penicillin and 30-60 mg of prednisolone. Lumbar puncture before placing the patient in
hospital not shown.

Acute purulent meningitis

Bacteria can enter the subarachnoid space and ventricles of the brain by hematogenous route with septicemia or metastasis from infectious foci in the heart, lungs or other
internal organs. In addition, damage to the membranes can occur as a result of contact
spread of infection from a septic focus in the bones of the skull, spine, brain parenchyma (sinusitis, osteomyelitis, brain abscess). The entrance gate for microorganisms is
fractures of the bones of the skull, paranasal sinuses and mastoid, as well as the area
neurosurgical intervention. It is extremely rare for an infection to drift with the lumbar puncture. Pathomorphology, symptoms and clinical course of acute purulent meningitis
of various etiologies are largely similar.
Acute purulent meningitis can be caused by almost any pathogenic bacteria, but in
the overwhelming majority of cases it is caused by hemophilus influenzae, meningococcus and pneumococcus. V
In recent years, the number of cases in which the pathogen is not detected has increased. Today such patients form the fourth large group in the structure of purulent meningitis. This situation, likely to be a consequence of prescribing therapy prior to admission to hospital.
During the neonatal period, the most common etiological agents are E. coli and
group B streptococcus. In older children, 60% of all bacterial meningitis
falls on meningitis caused by Haemophilus influenzae. General lethality from bacterial meningitis is about 10%.

Meningococcal meningitis

Meningococcus is a gram-negative diplococcus (Weixelbaum's bacillus). He
easily detected by microscopic examination in leukocytes or extracellularly.
Meningococcal infection is transmitted by droplets. The source of infection is
a sick person or a healthy host. Meningococci are very unstable to external
factors: temperature fluctuations, insufficient air humidity, the influence of sunlight, and quickly die outside the human body. This, apparently, explains the relatively low contagiousness of the disease. Undoubtedly, the degree of susceptibility also plays an important role. macroorganism to meningococcal infection.
As a rule, the disease is sporadic, but sometimes there are small
epidemics. The infection is characterized by a rather pronounced seasonality - the greatest the number of outbreaks is recorded in the winter-spring period. The disease occurs in people of all ages,
however, children are more likely to get sick, especially young ones.
Meningococcal disease can manifest itself in various forms: asymptomatic
bacterial carriage, nasopharyngitis, arthritis, pneumonia, meningococcemia, purulent meningitis and meningoencephalitis.

Pathogenesis. After entering the body, meningococcus first grows in the upper
respiratory tract and can cause primary nasopharyngitis, which usually resolves quickly. Have persons less resistant to infection, meningococcus enters the bloodstream and spreads throughout the body.
In severe cases, meningococcemia develops, often accompanied by a characteristic

hemorrhagic rash. The resulting endotoxin triggers disseminated intravascular coagulation and endotoxic shock.

Pathomorphology. The subarachnoid space is filled with purulent exudate. Superficial the veins are dilated. The accumulation of pus is noted mainly on the convexital surface of the cortex,

the base of the brain, on the membranes of the spinal cord. From the meninges of the brain, the inflammatory process is
perivascular spaces are transferred to the substance of the brain. As a result, edema occurs, small purulent foci in the substance of the brain, small hemorrhages and blood clots in the vessels. Microscopically, the picture of inflammatory infiltration is determined in the membranes of the brain. In the initial
and in the advanced stages of the disease, it is predominantly neutrophilic in nature, later lymphocytes and plasma cells appear. The ventricles, often dilated, contain
cloudy liquid.

Clinic and diagnostics. The incubation period for meningococcal infection ranges from 2 up to 10 days (usually 5-7 days). As a result of the penetration of meningococcus into the membranes of the brain,
purulent meningitis. The disease usually develops suddenly. Its beginning is so sharp that the patient or others can indicate not only the day, but also the hour of the onset of the disease. Body temperature
rises to 38-40 ° C, a very sharp headache occurs, which can radiate to the neck, back and even legs. The headache is accompanied by vomiting, which does not bring relief. Appear general hyperesthesia, photophobia, meningeal symptoms: neck muscle stiffness, Kernig symptoms and Brudzinsky, - however, their severity may be different and does not always correspond to the severity
process. Often, at the onset of the disease, bradycardia up to 50-60 beats per minute is noted. During diseases, the pulse rate increases, in some cases arrhythmia occurs.
Consciousness is initially preserved, but in case of untimely treatment, confusion develops. There may be a sharp motor excitement, sometimes a delirious state. As
the progression of the disease, excitement is replaced by drowsiness and stupor, turning into a coma. The fundus of the eye remains normal, sometimes there is some expansion of the venous vessels. In children
infancy, the onset of the disease is manifested by general anxiety, a sharp, piercing cry (meningeal cry). They often have clonic-tonic seizures,
sometimes passing into status epilepticus. Very important for diagnosing meningitis in infants a symptom of bulging and tension of the anterior fontanelle. Often, on the 3-4th day of illness, there are
herpetic eruptions on the skin and mucous membranes of the mouth, lips.
Of the local neurological symptoms, damage to the oculomotor is more often noted. nerves, manifested by diplopia, ptosis, strabismus, anisocoria. Less commonly, there is a lesion other cranial nerves. Before the use of penicillin, the vestibular cochlear nerves often suffered (Viii), and deafness was one of the most common complications of meningitis. Currently irreversible damage to this nerve is rare.
A blood test reveals neutrophilic leukocytosis and an increase in ESR. but
cases of a disease with a normal blood picture are possible.
Cerebrospinal fluid in the first hours of the disease may not be changed, but already by 1 On the 2nd day, its pressure rises sharply (usually up to 200-500 mm of water column), it becomes cloudy,
sometimes acquiring a grayish or yellowish-gray color. The number of cells is dramatically increased and
reaches hundreds and thousands (usually 2000-10000) in 1 mm. Pleocytosis is predominantly neutrophilic, the number

lymphocytes are negligible. Meningococci can be found in cells. The amount of protein in the CSF increased, sometimes up to 10-15 g / l. The glucose content drops sharply.
The duration of the disease with adequate treatment is on average 2-6 weeks, however possible hypertoxic forms, proceeding with lightning speed and leading to death in during the day.
Complications. Severe forms of meningococcal infection can be complicated by pneumonia, myo- or pericarditis. The development of cerebral edema is especially dangerous. The latter is accompanied by
impaired consciousness, epileptic seizures, respiratory and cardiovascular activity, oculomotor disorders. In the outcome, there is often a wedge in tentorial or foramen magnum. Acute cerebral edema may occur as in the beginning fulminant form of meningitis, and in the midst of the disease against the background of antibiotic therapy.
A characteristic clinical feature of meningococcal infection is the appearance on the skin hemorrhagic rash, usually in the form of asterisks of various shapes and sizes, dense on touch protruding above the level of the skin. More often, the rash is located in the buttocks, thighs, and on the legs.
It should be remembered that a hemorrhagic rash can sometimes be observed with meningitis caused by H.
influenzae, pneumococcus, in acute bacterial endocarditis caused by Staph. aureus, vasculitis and other conditions. The joints are sometimes affected. Meningococcemia may be accompanied by damage to the membranes of the brain, but can occur (in 20% of cases) and without the phenomena of meningitis.
Another severe manifestation of generalized meningococcal infection is bacterial (endotoxic) shock, which in most cases develops in childhood.

The disease occurs acutely. The body temperature suddenly rises, chills are noted. Soon a profuse hemorrhagic rash appears, at first small, and then larger, with necrotic plots. Pulse quickens, blood pressure decreases, heart sounds become muffled, uneven breathing. Convulsions sometimes occur. The patient falls into a comatose condition. Often the patient dies without regaining consciousness. For a long time, such an outcome was associated
with destruction of the adrenal cortex (Waterhouse-Friederiksen syndrome). At present time it is believed that the cause of such a severe course of the disease is mainly endotoxic shock resulting from damage to small vessels and an increase blood clotting with the formation of a large number of microthrombi (DIC syndrome). Defeat the adrenal glands can be detected in all cases.
Haemophilus influenzae meningitis
It is currently the most common (about 50%) form of acute bacterial meningitis. Mostly newborns and young children are ill: in 90% of cases the disease develops in the first 5 years of life. In newborns and young children, meningitis, caused by Haemophilus influenzae, usually primary. In adults, this is usually secondary lesion resulting from acute sinusitis, otitis media, bone fractures skull. The development of meningitis can be promoted by cerebrospinal fluid rhinorrhea, immunodeficiency, sugar
diabetes, alcoholism. There is a seasonality of the occurrence of meningitis with an increase in the incidence
in autumn and spring and its decline in the summer months.
The pathomorphology and clinical picture of meningitis caused by Haemophilus influenzae does not differ from
other forms of acute purulent meningitis. With a protracted course of the disease, local foci of infection in the membranes or cortex, internal hydrocephalus, damage to the cranial nerves,

thrombosis of cerebral vessels.
The duration of the disease is usually 10 to 20 days. In some cases, it can develop
lightning fast, sometimes dragging on for weeks or months.
Changes in cerebrospinal fluid are similar to those in purulent meningitis.
other etiology. Microorganisms can be isolated from both liquid and blood, in the latter
In this case, the cultures are often positive in the initial stage of the disease. In the absence of treatment for a long time
the current disease often develops paralysis of the external muscles of the eye, deafness, blindness, hemiplegia,
seizures, dementia. In untreated cases, neonatal mortality from meningitis,
caused by Haemophilus influenzae, is more than 90%. For adults, the prognosis is more favorable. They often recover spontaneously. Against the background of adequate treatment, mortality decreased
almost up to 10%, but complications are still not uncommon.

Pneumococcal meningitis

Pneumococcus (Streptococcus pneumoniae) as a cause of meningitis is as common as meningococcus. Meningeal infection is usually a complication of otitis media, mastoiditis, sinusitis, fractures of the bones of the skull, infectious diseases of the upper respiratory tract and lungs.
The factors predisposing to the development of pneumococcal meningitis are alcoholism, poor nutrition, sickle cell anemia. The disease can occur at any age, but more
50% of cases are patients under one year old or over 50 years old.
Body temperature is often elevated, but it can be subfebrile and normal. Quickly
shell symptoms appear, but in a coma, they can disappear. Often in
the process involves the brain, cranial nerves. A picture of acute swelling and edema is possible the brain with its insertion. On the 3-4th day of illness on the mucous membrane of the mouth, less often on the arms and trunk
herpetic eruptions appear, small hemorrhagic rash, bradycardia and
lowering blood pressure. In the blood - pronounced inflammatory changes. Pneumococcal
the etiology of meningitis is confirmed by the detection of the pathogen in the cerebrospinal fluid or blood, positive serological reactions.
Without treatment, patients die on the 5-6th day. The disease can take a protracted course. Before introduction of sulfa drugs into practice detail from pneumococcal meningitis
was almost 100%. Now it is about 20-30%. Better prognosis in
recovery plan is observed with meningitis complicating a fracture of the skull bones or unknown source of infection. In contrast, mortality is high in cases where meningitis develops as a result of pneumonia, empyema, lung abscess, or persistent bacteremia, indicating the presence of bacterial endocarditis. Exceptionally high mortality rate

accompanied by a triad, including pneumococcal meningitis, pneumonia and bacterial endocarditis (Austrian syndrome).
If possible, the primary focus of infection is removed surgically. Liquor
fistulas arising from fractures of the skull bones are closed by craniotomy and suture the hard shell of the brain. Otherwise, relapses almost always occur.

Listeria meningitis

Listeriosis is a disease caused by Listeria monocytogenes, in which, in addition to meningitis, subacute sepsis, endocarditis, skin lesions are possible. The disease is characterized by an acute onset, an increase in body temperature, a kind of rash on the face in the form of a "butterfly", enlargement of the liver, spleen, lymph nodes.
Listeria meningitis, as a rule, occurs in debilitated patients: in young children,
especially newborns, and in the elderly, suffering from neoplastic diseases or
the background of immunosuppressive therapy. Clinical manifestations of the disease can develop in the form of

acute infection, and subacute. Unlike other meningitis in patients with listeria meningitis often does not show a stiff neck. In the cerebrospinal fluid, the amount cells can vary significantly: 50-1000 mm. The glucose concentration is normal or slightly lowered. The protein is slightly increased. Disease verification is carried out by isolating listeria from cerebrospinal fluid, throat washes, amniotic fluid.
The clinical diagnosis of listeria meningitis is based on the detection of common symptoms listeriosis, especially jaundice. In their absence, they resort to searching for the pathogen and conducting
serological reactions.
Treatment - intravenous penicillin up to 10,000,000-12,000,000 U / day for 8-10 days. V in severe cases, this therapy is supplemented by oral prednisolone at 40–60 mg / day. Exodus diseases in most cases are favorable.

Amebic meningitis

Amoebic meningitis is more common among young people and children when bathing in polluted reservoirs in the summer. The onset of the disease is manifested by a headache, an increase body temperature, the occurrence of meningeal symptoms, impaired sense of smell. Last thing due to the fact that Naegleria fowleri penetrates into the mucous membranes of the nose, and then through the ethmoid
the plate - into the cranial cavity, affecting primarily the olfactory nerves (I). Often focal neurological symptoms develop, indicating the spread of pathological gichesky process on the substance of the brain. Cerebrospinal fluid contains neutrophilic pleocytosis, a significant number of erythrocytes, the glucose content is reduced. In a number of patients it is possible
detect amoebas in the cerebrospinal fluid, which confirms the diagnosis. The disease quickly progresses.
progresses and in the absence of treatment is fatal within 1-2 weeks.
Treatment: Amphotericin B is prescribed at 250 U / kg, if necessary, the dose is increased to 1000 UNIT / kg. The drug is administered intravenously every other day. Treatment lasts 4-8 weeks. A drug
nephrotoxic. It is possible to save patients only with the early initiation of amphotericin therapy.

Recurrent bacterial meningitis

Recurrent episodes of bacterial meningitis indicate either anatomical defect, or about violations of the mechanisms of immunological protection. Often recurrence of meningitis
observed after traumatic brain injury, while the first attack of meningitis may develop and after few years. As a rule, the causative agent in this case is pneumococcus. Into the subarachnoid the space the bacteria can enter through the ethmoid plate, the site of the fracture bones of the base of the skull, eroded bony surface of the mastoid process, as well as penetrating head wounds or neurosurgical interventions. Often there is a cerebrospinal fluid rhinorrhea or otorrhea, which may be transient. It is revealed by being discovered in secrets from the nose and ear glucose in significant concentration. Rhinorrhea is often misinterpreted as rhinitis.
An old diagnostic sign is the “teapot” symptom: when the head is tilted forward, the outflow from the nose is getting worse. Treatment for recurrent meningitis is the same as for the first episodes of the disease.
A radical solution to the problems of recurrence is the surgical closure of the cerebrospinal fluid fistula. For
detecting the site of the outflow of cerebrospinal fluid using radionuclide techniques with preliminary introduction of contrast agents into the intrathecal space.

Sometimes bacterial meningitis mimics nonbacterial recurrent meningitis (Molar's meningitis, meningitis with Behcet's syndrome, etc.).

Complications of acute purulent meningitis

Neurological complications associated with acute bacterial meningitis are divided
early and late. Early ones include: increased ICP, epileptic seizures, arterial or
venous thrombosis, subdural effusion, hydrocephalus, sensorineural deafness. The late ones include
intellectual impairment, permanent focal neurological deficit, epilepsy. Below
the most common complications are described.
Increased intracranial pressure. ICP is an important determinant of outcome
diseases. Fluttering with pronounced cerebral edema, an insertion is found in 4-8% of patients.
With severe cerebral edema, manifested by impaired consciousness, pupillary anomalies, focal
neurological symptoms, mannitol is used (0.5 mg / kg, injected in 30 minutes), with
need for longer
of treatment add dexamethasone intravenously.
The feasibility of using corticosteroids for uncomplicated acute bacterial
meningitis is problematic as it reduces the penetration of antibiotics into the central nervous
system. V
mild cases, to reduce cerebral edema, it is enough to limit fluid intake to 1.2-
1.5 l / day
Convulsive syndrome. Partial or generalized seizures occur in 30%
patients with meningitis, more often in children. Seizures usually develop in the first 48 hours after
the onset of the disease.
Possible causes of seizures include fever, hyponatremia, cerebral infarctions,
subdural empyema, toxic encephalopathy due to effects on the nervous system
decay products of leukocytes and bacteria. As a late complication, epilepsy is rare.
Indications for long-term anticonvulsant therapy after bacterial cure
meningitis is not precisely defined. Patients with a mild course of the disease who do not have
significant
neurological abnormalities and epileptic changes on the ECG, anticonvulsant therapy
can be discontinued after discharge from the hospital.
Brain infarctions. This complication is most common in children when the pathogen
the disease is Haemophilus influenzae, however, it can also be observed with meningitis of a
different etiology.
Damage develops secondarily, usually due to vasculitis.
Subdural effusion. CT significantly increased the detectability of subdural effusions. They
found in 24-30% of cases of neonatal meningitis. For most children, they do not give
symptoms and do not require intervention. The development of subdural effusion in newborns is the
most
characteristic of meningitis caused by Haemophilus influenzae. Persistent vomiting, bulging
fontanelles,
seizures, focal neurological symptoms and persistent fever allow
suggest the presence of this complication. Large effusions lead to displacement of the brainstem.
Their evacuation by puncture of the fontanelle is usually accompanied by a rapid disappearance of
symptoms.
Subdural empyema. It is observed in 2% of cases of bacterial meningitis in children. Analysis
subdural fluid allows you to differentiate empyema from the more common subdural
effusions. Both effusion and empyema are characterized by an increase in white blood cell count and
concentration
protein, but with empyema, the fluid is more purulent and contains mainly neutrophils.
Long-term or recurrent fever without an increase in meningeal
symptoms may be due to the formation of extracranial foci of infection or the action
used medicines.
Hydrocephalus. Most often complicates the course of the disease in children, but can occur in any
age. In newborns, hydrocephalus is observed in 30% of cases of meningitis. Great danger
represents the spread of the inflammatory process to the ventricles of the brain and the development
of ventriculitis.
Sensorineural deafness. This is a rather rare consequence of meningitis, observed in 5-9%
sick. If hearing impairment is detected, an audiological assessment should be carried out and how

you can start rehabilitation earlier.

Treatment of acute purulent meningitis

Antibiotic therapy. The empirical choice of antibiotic for initial treatment is the probable nature of the pathogen, which to a certain extent depends on the age of the patient. Combinations of antibiotics should be avoided unless proven to be synergistic. Doses and frequency drug administrations must be carefully verified; this is especially important when treating newborns and children.

For meningitis of newborns, treatment begins with ampicillin and cephalosporins of the third generations (cefotaxime, claforan, ceftriaxone, ceftazidime); the latter are active against E. coli. If the causative agent is group B streptococcus, the drug of choice is penicillin. With meningitis, caused by Haemophilus influenzae, it is recommended to start therapy with a combination of ampicillin and chloramphenicol. An alternative agent is chloramphenicol or cephalosporins of the third generations. Streptococcus is the most common cause of meningitis in adolescents and adults. pneumoniae and Neisseria meningitidis, which are sensitive to penicillin. If you are allergic to penicillin the drug of choice for pneumococcal and meningococcal meningitis is chloramphenicol. In immunodeficiency states, meningitis can be caused by fungi, other species bacteria and toxoplasma.

It should be borne in mind that it is often not possible to identify the type of pathogen. It could be due to the fact that before hospitalization they received antibiotics, for the most part in insufficient dose. In these cases, consultation with an infectious disease specialist is necessary.

Duration of intravenous antibiotics for bacterial meningitis usually 7-10 days after normal body temperature is established. Expressed the opinion that with a good response to antibiotic therapy in the case of meningitis caused by meningococcus or H. influenzae (but not pneumococcus!) end of treatment. Longer treatment is necessary in severe cases, especially when prolonged high fever. Prolonged or repeated fever may be caused by phlebitis, metastatic foci of infection, nosocomial infection (Greek nosokomio - hospital); rarely, untreated meningitis is the cause. Until all possible the reasons for the increase in body temperature, drug fever cannot be diagnosed. If, despite adequate therapy, the increase in temperature persists for no apparent reason, antibiotics can be canceled with a positive dynamics of changes in cerebrospinal fluid.

The fate of patients with purulent meningitis is decided in the first days of the disease. General treatment principle is to create "antibiotic protection" as early as possible. Therefore it is necessary strive for the earliest possible diagnosis and early initiation of therapy antibiotics. Unfortunately, the specific pathogen is far from always known, and for its laboratory verification requires a certain time, the loss of which can lead to serious consequences. Therefore, at the onset of the disease, purely clinical signs are used, which which to some extent can guide the doctor in determining the nature of the infection.

In the absence of accurate data on the nature of the pathogen, it should be assumed that the most penicillin has a wide range of effects on bacterial agents. Penicillin is injected intramuscularly or intravenously at the rate of 200,000-300,000 U / kg per day, i.e. 18,000,000-24,000,000 IU per day in equal doses after 3-4 hours.Approximately 10-12 hours after the start of penicillin administration the patient's condition is gradually improving, consciousness clears up, headache decreases. TO at the end of the 1st or 2nd day of treatment, a decrease in body temperature is noted. Decreases gradually the severity of meningeal symptoms, they disappear on the 4-10th day of treatment. Reorganization in progress

cerebrospinal fluid. An indication for the cancellation of penicillin is a decrease in cytosis to less than
100 cells in 1 mm and the predominance of lymphocytes in the cellular composition (more than 75%). In the absence
effect and with initially severe forms of meningoencephalitis, the daily dose of penicillin increases up to 48,000,000 UNITS If the patient is in a coma or treatment is started late,
the dose of administered penicillin is increased to 800,000-1,000,000 U / kg of body weight per day. In these cases
only intravenous penicillin should be used. You can also apply
semi-synthetic penicillin - ampicillin in the highest daily dose of 12-15 g. It should be noted that in general, in the treatment of infectious lesions of the central nervous system, especially in severe cases, antibiotics
should be administered intravenously; intramuscular injection is technically simpler and can be used in
mild cases. Endolumbar administration of antibiotics, with rare exceptions, is not indicated.
Symptomatic therapy. Adequate symptomatic therapy is possible with constant
control of vital functions of the patient's neurological status. With pronounced
respiratory disorders are performed by mechanical ventilation. Careful correction of water electrolyte imbalance (especially hyponatremia). With hypovolemia, you first need
replenishment of blood volume using intravenous administration of crystalloid and colloid solutions. After correcting hypovolemia, it is necessary to limit the introduction of fluid to 1/3 of usual (1.2-1.5 l / day), since there is a high risk of developing cerebral edema and increased secretion antidiuretic hormone (ADH) in the early stages of meningitis. If ADH hypersecretion is detected (hyponatremia less than 120 mEq / L, plasma hypoosmolarity), a longer duration is indicated

reducing the introduction of fluid, in severe cases - the introduction of a hypertonic solution or saline in combination with furosemide (under the control of sodium levels, which should
be increased to 125 meq / l). In case of persistent arterial hypotension, vasopressors are prescribed (dopamine). With the development of DIC syndrome against the background of septic shock, which often complicates
bacterial meningitis, administration of heparin is indicated.

Acute lymphocytic meningitis

Meningitis caused by Coxsackie viruses and ECHO

Epidemiology. Coxsackie viruses were first isolated in 1948. in the village of Koksaki state New York in the USA, Somewhat later in 1951, viruses were discovered, which were designated as "Orphan viruses", as it was not known what diseases they cause. Name of this group
comes from the initial letters of the English term ECHO (Enteric Citopatogenic human orphan - literally an orphan virus that infects cells of the human small intestine). Later it was suggested
to combine poliomyelitis viruses, Coxsackie viruses and ECHO into the enterovirus group. Coxsackie and ECHO viruses are widespread among the population of the entire world. Sporadic diseases have been reported since the early 1930s, and epidemic outbreaks since 1958, when the incidence of poliomyelitis began to decline. There is a certain seasonality in their occurrence with a maximum in the summer-autumn period. Mostly children are ill. High contagiousness and massiveness of diseases suggest an airborne method
transmission of infection. However, one cannot completely exclude the possibility of an enteral route of its distribution.
wandering. The incubation period for enteroviral diseases ranges from 2 to 7 days. From
More than 40 strains of the currently known enteroviruses are undoubtedly pathogenic. They can cause a wide variety of diseases, including myalgia and spinal cord injury,
resembling polio. The same viruses can infect the lining of the brain, causing acute
serous meningitis.
Enterovirus infection is highly contagious and therefore caused by it
diseases often arise in the form of epidemic outbreaks.

Clinic. Serous meningitis caused by enteroviruses usually has an acute onset
accompanied by a sharp rise in body temperature, severe headache, repeated vomiting.
The high temperature lasts about a week, sometimes a little less, and then decreases to normal.
The general appearance of the patient is very typical: the face is hyperemic, against this background stands out pale,
nasolabial triangle. Many have severe conjunctivitis, vascular injection of the sclera.
Pharyngitis, herpetic eruptions on the lips and in the nose, herpetic
angina. In the blood, quite often there is a shift in the leukocyte formula to the left, an increase in ESR up to
25-40 mm / h.
Leading in the clinical picture are signs of intracranial hypertension - headache
pain and vomiting. A decrease in ICP by lumbar puncture is accompanied by a decrease
headache and cessation of vomiting.
Meningeal symptoms do not appear immediately, but on the 2-3rd day of the disease. When conducting
lumbar puncture, cerebrospinal fluid is clear, flows out under increased pressure,
there is lymphocytic pleocytosis, the protein content is within normal limits or slightly increased.
The glucose content does not change.
Sanitation of cerebrospinal fluid occurs gradually, starting from the 10-12th day, and
it normalizes by the end of the 3rd week.
The course of enteroviral meningitis is favorable, the disease ends in complete
recovery. However, one should bear in mind the tendency to relapse, the frequency of which ranges from
10 to 40% of cases. Clinically, a relapse is manifested by a deterioration in the general condition,
body temperature, increased headache, sometimes vomiting. Are re-discovered
changes in cerebrospinal fluid. Relapse is possible at different times - from 2 weeks to 1 month.
Despite the presence of relapses, the outcome of the disease remains favorable.
Diagnostics. Recognizing meningitis is not particularly difficult. Sharp start
diseases, headache, meningeal symptoms and, finally, changes in cerebrospinal
fluids indicate the presence of serous meningitis. It is much more difficult to establish it
etiology. The main property of meningitis of enteroviral origin is their high
contagiousness and hence the massive defeat of significant groups, especially children. Should
constantly remember that this particular type of meningitis is, along with mumps, the most frequent

a form of acute lymphocytic meningitis. Clinical signs of the disease, as with others
forms of aseptic meningitis are not very informative. To obtain reliable diagnostics
virological and serological studies are required.
Treatment. Symptomatic treatment: analgesics and diuretics.
Mumps meningitis
Mostly preschool and school children are prone to mumps.
age. Among young children, the disease is much less common. Boys get sick
somewhat more often than girls. Inflammation of the salivary glands is accompanied by swelling.
The onset of meningeal symptoms is possible at different times. Most often they appear
3-5 days after swelling of the glands, but other time relationships are possible, when
meningeal signs develop much later, for example, a month after swelling of the glands,
or precede it. Significant diagnostic difficulties arise when
mumps meningitis develops without clinical manifestations of mumps.
As with other viral lesions, mumps infection is characterized by a period of viremia, during
the time during which the virus from the blood enters the nervous system, in particular into the membranes of the brain. Like
pathogenesis is probably inherent in early forms of the disease, which are related to primary lesions nervous system. The late forms of mumps meningitis seem to be based on
neuroallergic process.

It should be emphasized that during the period of viremia, the mumps virus can penetrate, in addition to salivary
glands and membranes of the brain, and other organs, such as the pancreas and testicles in boys, causing pancreatitis or orchitis.
It is assumed that the mumps virus is transmitted by airborne droplets. Incubation
the period lasts up to 3 weeks, after which clinical symptoms of the disease appear, in the first place swelling of the salivary glands. Within 10 days after the first symptoms of the disease appear
the patient is a source of infection and is a danger to others.
Clinic and diagnostics. Mumps meningitis develops acutely, accompanied by a sharp
increased body temperature (30-40 ° C), intense headache, repeated vomiting.
Meningeal symptoms appear early but are moderate. The most constant is
stiff neck muscles; Kernig's and Brudzinsky's symptoms are noted only in some patients. In children young age develop general weakness, drowsiness, weakness, less often a state of excitement, delirium, hallucinations. In rare cases, impaired consciousness, generalized
convulsive seizures. Mumps virus is the most common cause of viral meningitis, except in countries where
vaccination against mumps is carried out.
It should be borne in mind that mumps infection, affecting the nervous system, can proceed without meningeal symptoms. Mumps meningitis is characterized by a wide range of clinical manifestations: serous meningitis, meningism without meningitis, clinically asymptomatic meningitis.
Therefore, the study is of great importance in the diagnosis of mumps meningitis.
cerebrospinal fluid. Correct diagnosis plays an important role in the treatment and
prevention of possible complications, most often intracranial hypertension.
With a lumbar puncture, cerebrospinal fluid flows out under increased pressure.
It is colorless, transparent, sometimes only slightly opalescent; pressure 250-300 mm of water Art. Cytosis in
within hundreds, sometimes several thousand cells. In the first days of the disease, lymphocytes predominate, but
possibly a small amount of polynuclear cells. Over time, cytosis becomes excluded
strongly lymphocytic. The amount of protein is slightly increased (0.6-1 g / l), but may be within norms. The glucose content is not changed. The clinical picture is mainly determined not by the magnitude
cytosis, and the degree of increase in ICP.
In the process of recovery, the cerebrospinal fluid is sanitized within 2 weeks, less often in longer terms. Clinical symptoms disappear much earlier than sanitized
cerebrospinal fluid. Their disappearance is noted on the 7-10th day. Signs of defeat
brain substances are found in 1-5% of cases. Of the complications that may arise during diseases, one should remember about pancreatitis and especially orchitis (in boys over 10 years old). Pancreatitis occurs in about 15% of patients. Clinically, it presents with abdominal pain with predominant localization in the upper left quadrant with irradiation to the back, repeated vomiting. The level of amylase in the urine rises to 150-250 units. Orchitis is painful and swelling of one or both testicles, hyperemia and swelling of the scrotum, fever
body. These phenomena decline after 3-5 days, and disappear completely by the 10-12th day. However, one should

keep in mind that severe orchitis, transferred during puberty, can subsequently
lead to infertility.
The most operative method for establishing the etiology of mumps meningitis is the method fluorescent antibodies. Significantly longer time is required to determine antibody titers
by RTGA in paired samples of blood serum and cerebrospinal fluid.
Treatment. With mumps, treatment is symptomatic. Bed rest
it is prescribed depending on the patient's condition, since it does not have a noticeable effect on

the frequency of complications. The diet should be consistent with the patient's ability to chew. Lumbar
punctures relieve headaches. For orchitis, bed rest and local treatment are recommended.

Acute lymphocytic choriomeningitis

A rare form of neuroinfection. The reservoir of the virus is gray domestic mice. Infection occurs due to contact with objects contaminated with excrement or nasal mucus of these animals. When it enters the body, the virus is hematogenously spread throughout the body. The disease can take on the nature of small epidemic outbreaks, but more often
sporadic cases occur. Mostly people aged 20-35 are sick.

The incubation period of the disease lasts 1 - 2 weeks. The onset of the disease is characterized by general malaise, weakness, headache, catarrhal symptoms. In the future, it is enough
the body temperature rises sharply to 39-40 ° C, vomiting occurs, the intensity of the head increases pain. Within a few hours, meningeal signs develop. Often, patients are worried about pain in the eyeballs, a feeling of pressure in them, photophobia. Possible unstable dysfunctions oculomotor nerves, paresis of facial muscles. 1/4 of patients have symptoms,
indicative of the involvement of the brain substance: pathological foot marks, anisoreflexia, loss of coordination.

Along with this, abortive forms are also possible, manifesting only
general infectious signs.

To establish a diagnosis, it is necessary to study the composition of the cerebrospinal fluid. When a lumbar puncture is performed, fluid is released under increased pressure, it transparent, contains an increased number of cellular elements - lymphocytes. Their number reaches 1000 in 1 ml and above, the content of protein and glucose is usually not changed.

The course of the disease is benign. 7-10 days after the onset of the condition of the patients improves - cerebral and meningeal symptoms disappear. Complete sanitation of the cerebrospinal liquid is tightened up to 1.5-2 months. During the period of convalescence, pain in the joints of the hands is characteristic,
which are accompanied by edema. On the 2-3rd week from the onset of the disease, it is possible to develop
generalized alopecia, orchitis.

Diagnosis of acute lymphocytic choriomeningitis is based on the presence of moderate pronounced meningeal syndrome against the background of an increase in body temperature, characteristic changes
in the cerebrospinal fluid and the benign course of the disease. Etiology of the disease established by isolating the virus from the blood and cerebrospinal fluid, and
studies of the reaction of neutralization and binding of complement. Urgent diagnosis of etiology meningitis is diagnosed using the fluorescent antibody method.

Treatment is symptomatic.

Herpes zoster meningitis

The group of herpes viruses includes 80 species. One of the main features of the family - the ability for lifelong persistence in the host's body. Varicella-Zoster virus causes chickenpox in children and shingles in adults. The incidence of herpes zoster is 1-2%. Aseptic meningitis presents with fever, headache, very mild
meningeal signs. The onset of the disease is acute, body temperature sometimes reaches 38-39 ° C. Confusion, stupor, coma are extremely rare. Meningeal signs are barely outlined. Liquor Zoster meningitis syndrome is characterized predominantly by lymphocytic pleocytosis (from 25 up to 150 cells in 1 mm). Pleocytosis decreases from the first days to the 6th week of the disease. Compared with
with other aseptic meningitis, herpetic meningitis is characterized by longer periods of sanitation cerebrospial fluid. Lymphocytic pleocytosis occurs in 40-80% of cases of manifest zoster infection. Such data suggest that asymptomatic meningitis with
shingles is not a complication, but an almost obligatory component of the disease. Shown the appointment of acyclovir.

Alternating delayed hemiparesis

Among the complications of shingles, which usually include ocular apples, postherpetic neuralgia, cranial nerve palsies, myelitis, meningoencephalitis, recently a new symptom complex was identified - contralateral hemiparesis associated with herpes zoster ophthalmicus (HZO).

Its development is based on damage to the vessels of the brain, causally associated with a viral infection, which was verified by histological examination of temporal artery biopsies.

A characteristic finding is necrotizing granulomatous angiitis of the meningeal and cerebral arteries, ipsilateral cerebral infarctions.

Considering that there is a time interval between the occurrence of herpes zoster and a motor defect that the trigeminal node (gasser node) is close anatomically to the proximal department of the middle cerebral artery and there are phenomena of ganglionitis in it and that viruses are determined in the adventitial membrane of the arteries, we can assume the spread of the virus from the node to the vessel tactfully. Possible cerebrospinal fluid and hematogenous pathway.

The development of cerebellar and occipital lobe infarction with HZO and cerebral stem infarction is described with cervical localization of herpes.

The clinical block described is referred to as HZO hemiplegia delayed syndrome, or alternating zoster syndrome. The average age of patients is 58 years. 75% of patients are over 40 years old.

The average interval between the appearance of HZO and the development of contralateral hemiparesis is 7.3 week Sometimes this interval is reduced to 1 week, and in some cases it increases to 6 months.

Half of the patients have a severe course of the disease: somnolence, stupor, confusion. Mortality reaches 20%. The development of hemiparesis is often acute, but it is also possible a slowly progressive increase in the motor defect. In some patients, the disease proceeds benign with satisfactory recovery. More often described as isolated hemiparesis of varying severity. Only in isolated cases is it supplemented with hemianesthesia, hemianopsia, cerebellar signs.

One gets the impression that the occurrence of hemiparesis is not accompanied by additional changes in the composition of the cerebrospinal fluid. Inflammatory changes are found in 1/3 punctured patients, in 1/3 of patients CSF at the time of the development of hemiparesis is normal.

Meningitis with infectious mononucleosis

Infectious mononucleosis - a disease caused by the Epstein-Barr virus and manifested by damage to the lymph nodes, liver, spleen and skin. Mononucleosis is recorded mainly in children and young people. A typical clinical picture constitute malaise, fever, headache, tonsillitis, enlargement of the cervical lymph nodes, hepatosplenomegaly, as well as a specific change in the hemogram. Involvement of the nervous system occurs in no more than 1-5% of cases. Lymphocytic pleocytosis in cerebrospinal fluid is found in the absence of any neurological symptoms. Severe headache and stiffness of the cervical muscles can be the initial and only manifestations of CNS damage (aseptic meningitis). Signs of encephalitis (delirium, convulsions, coma, focal symptoms) are rarely observed. Described optic neuritis (II), facial lesions (VII) and others cranial nerves, acute autonomic neuropathy, Guillain-Barré syndrome, transverse myelitis, acute cerebellar ataxia.

The prognosis is favorable with complete regression of neurological symptoms, with the exception of cases where, against the background of severe polyneuropathy, paralysis of the respiratory muscles develops.

In laboratory diagnostics, the detection of leukocytosis in the blood with

an increase in the number of lymphocytes and the appearance of abnormal mononuclear cells (atypical
lymphocytes). There is no specific therapy. The appointment of glucocorticoids is shown only in cases of severe complications.

Meningitis with cat scratch disease

The leading manifestation of the disease is chronic regional lymphadenopathy,
occurring in the area of scratches caused by a cat (93%), less often a dog or other animals.
The disease is usually benign, and the adenopathy resolves spontaneously within a few
weeks or months. The causative agent is not exactly identified, it is assumed that it is
gram-negative pleomorphic bacillus. In 60% of cases, the disease develops at the age of 5-21 years.
General infectious symptoms appear 3-10 days after scratching, after 2 weeks

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regional lymphadenopathy joins.
The defeat of the nervous system is observed in the form of serous meningitis, radiculopathy,
polyneuropathy, myelopathy. Neurological symptoms usually come on suddenly, with
fever, 1-6 weeks after the onset of adenopathy. The cerebrospinal fluid may contain
moderate pleocytosis and increased protein content. The acute period lasts 1-2 weeks, then within
After 1-6 months, there is a gradual recovery, which is complete in 90% of cases.
The effectiveness of antibiotic therapy has not been proven.

Vogt-Koyanagi-Harada syndrome (uveomeningitis)

Systemic disease manifesting meningitis, eye damage (bilateral
chronic uveitis, retinitis), alopecia, vitiligo, gray eyelashes (poliosis),
hearing (tinnitus, disacusion).

Mollare benign recurrent meningitis

The disease is described in 1944. Characterized by spontaneous, short-term,
remitting headaches and stiff neck muscles. Duration of an attack
2-3 days, most of them proceed as mild aseptic meningitis. When examining
symptoms of irritation of the meninges are revealed. Transient neurological
disorders in the form of epileptic seizures, diplopia, dysarthria, imbalance,
damage to the facial nerve (VII), anisocoria, foot signs and coma. Fever is usually mild, but
can reach high numbers (40 ° C). The disease occurs at any age. Exacerbations are observed
usually within 3-5 years. In cerebrospinal fluid in the advanced stage of the disease
detect lymphocytic pleocytosis (from 200 to several thousand cells per 1 mm), small
increased protein, normal glucose concentration. In the early stages of the disease in
cerebrospinal fluid find large endothelial cells with reduced osmotic
resistance, however, their presence is variable and is not necessary to establish
diagnosis. Among the etiological factors are called herpes simplex type I, epidermoid cysts,
histoplasmosis, but none of them occurs with obvious consistency.
Mollare meningitis is differentiated from recurrent bacterial, viral and
fungal meningitis and sarcoidosis, hydatid (echinococcal) cysts, intracranial
tumors, Behcet and Vogt-Koyanagi-Harada syndromes. The last two diseases, among others
signs, characterized by severe lesions of the skin and eyes.
The recovery of patients with Mollare meningitis usually occurs without specific treatment.
The exacerbation is controlled by the appointment of colchicine, glucocorticoids or non-steroidal
anti-inflammatory drugs.

Chronic meningitis

In addition to tuberculous, cryptococcal and brucellosis meningitis, subacute and
the chronic course of meningitis (meningoencephalitis) is observed with syphilis, leptospirosis,
borreliosis, actinomycosis, Whipple's disease, AIDS, toxoplasmosis, cysticercosis, sarcoidosis,
vasculitis, neuroleukemia, carcinomatosis, chemical meningitis, breakthrough of cysts with
cholesteatomas
and craniopharyngiomas.

Tuberculous meningitis

Tuberculous meningitis is most often a manifestation of hematogenous disseminated tuberculosis. The primary focus is usually localized in the lungs or bronchial lymph nodes, but its localization is also possible in other organs. In many cases, the primary focus remains unrecognized. The infection enters the lining of the brain by a hematogenous route. It is assumed that tubercle bacilli enter the central nervous system through the choroid plexuses of the ventricles of the brain into the cavity
the latter, causing an inflammatory process in the pia mater. Great value in
the development of tuberculous meningitis is attributed to allergic factors.
In the recent past, it was believed that the disease mainly affects children, but in the latter years, tuberculous meningitis is equally common in both children and adults.
Pathomorphology. The most characteristic morphological manifestations of tuberculous lesions of the soft membranes of the brain are the rash of miliary tubercles and the appearance in subarachnoid space of serous-fibrinous exudate. The meninges lose their transparency and become cloudy, covered with a jelly-like effusion. This process is most intensive

expressed at the base of the brain. Tuberculous tubercles can also be located on the outer surface brain, in the area of the lateral (Sylvian) groove, along the cerebral vessels, but here their number significantly less than at the base of the brain. Bumps the size of a grain of millet or a few less at first have a grayish color and are hardly noticeable, but then as a result of caseous decay acquire a yellowish color and become well visible. Inflammatory changes in acute
stages of tuberculous meningitis are pronounced exudative in nature.
Microscopically, diffuse infiltration of the pia mater is detected
lymphocytes and macrophages, polynuclear cells are less common. The tubercles have a characteristic
epithelioid structure, they contain giant cells. In the center of some tubercles there is
caseous decay. Infiltrates of lymphocytes and plasma
cells. As a rule, with tuberculous meningitis, not only the membranes but also the substance of the brain are affected.
The ventricles of the brain are distended, contain a cloudy liquid.
In the course of treatment, exudative inflammatory changes can completely undergo
reverse development. In the case of insufficiently intensive or late started treatment, the disease becomes chronic. Exudative changes turn into productive ones, appear
scars, adhesions and adhesions between the membranes. Significant changes occur in the vessels of the membranes
and the substance of the brain. Development of the sclerosing process in the ependyma and choroid plexuses
ventricles of the brain causes severe violations of cerebrospinal fluid and promotes the development hydrocephalus. Symptoms of blockade of the subarachnoid space are often found. In the later
During periods of illness, petrification may occur, typical for other forms of tuberculosis.
Clinic and diagnostics. Unlike purulent and serous meningitis of other etiology
tuberculous meningitis develops slowly in most cases. The appearance of meningeal
symptoms are preceded by a period of precursors: general malaise, increased fatigue, increased appetite, sleepiness during the day, restless sleep at night. These symptoms are complemented by
low-grade fever, often rising in the evening, a slight headache.
The patient loses weight, becomes pale, lethargic, loses interest in others, strives for solitude.
Sometimes vomiting appears for no apparent reason. Gradually, the severity of these symptoms increases,
headache intensifies, vomiting becomes more frequent, sleep becomes restless, patients are persecuted
nightmares. In adults, distinct mental changes develop.
The manifestations of psychosis may precede the symptoms of meningitis.
The slow onset of all these symptoms is due to toxic effects.

tuberculous process developing in the membranes of the brain. The prodromal period may last 2-3 weeks. Then there are signs of irritation of the meninges, muscle stiffness neck, a symptom of Kernig, Brudzinsky, etc. Body temperature rises to 38-39 C. General condition patients sharply worsens, headache increases. Vomiting becomes more frequent, anorexia develops. Head
the pain becomes so severe that the patients grab their heads with their hands and scream. In children young people often have seizures - general or local. The overall
hyperesthesia. An important symptom in early childhood is protrusion of the anterior fontanelle.
Gradually to symptoms of irritation of the membranes, which may not be so pronounced, symptoms of prolapse join - most often in the form of paresis and paralysis of the cranial nerves: abductor (VI), oculomotor (III), facial (VII). Other cranial nerves are less commonly affected, but dizziness, tinnitus, hearing loss may occur, indicating involvement
vestibular cochlear nerve (VIII). Possible rapid loss of vision due to the development of neuritis optic nerves or congestion in the fundus. Loss of vision due to secondary
atrophy of the optic nerves after stagnation, is irreversible.
Gradually, patients develop bradycardia, followed by tachycardia, increases blood pressure. In the later stages, local symptoms appear - the asymmetry of deep reflexes, pathological signs, mono- or hemiparesis, indicating damage to the substance brain. Consciousness darkens, convulsions appear, the patient's head is thrown back, legs bent at the hip and knee joints, the abdomen is pulled in.
Cerebrospinal fluid is clear, colorless, and flows out under increased pressure.
The amount of protein is increased to 1-5 g / l and more, pleocytosis is up to 100-300 cells per 1 mm. Mixed cytosis,
70-80% are lymphocytes.
A very important diagnostic sign is a decrease in the glucose content in the fluid.
The blood picture does not change significantly.
The slow development of the disease significantly complicates early diagnosis. Additional

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difficulties arise in connection with the widespread use of antibiotics for febrile conditions any etiology, which significantly changes the traditional deployment of a tuberculosis clinic meningitis and leads to delayed lumbar puncture and, therefore, the establishment correct diagnosis. This significantly worsens the prognosis and often leads to the development chronic and recurrent forms of the disease, unknown in the pre-reptomycin era. Relapses meningitis usually occurs against the background of an exacerbation of the main tuberculous process resistant to
treatment or insufficiently intensively treated.
Chronic forms of tuberculous meningitis are accompanied by a number of complications, in significantly aggravating the overall prognosis, the most common of which is hydrocephalus.
In some cases, tuberculous meningitis can occur with purely spinal manifestations. This is usually seen in adults. It can be acute - with a rapidly increasing paraplegia, radicular pain, fever, resembling ascending myelitis in the clinic, or chronically with slowly increasing paraplegia, sensory conduction disorders, which requires a differential diagnosis between spinal cord compression. Possibility a similar course of the disease without a typical clinic of basal tuberculous meningitis causes diagnostic errors.
Treatment. They use "triple" therapy: isoniazid, rifampicin, pyrazinamide. This block the most powerful tuberculostatic drugs last 2-3 months; the next 7 months - isoniazid and rifampicin. Isoniazid is prescribed at a dose of 15 mg / kg per day 1-3 times orally after meals. Isoniazid
induces a deficiency of pyridoxine (vitamin Bb). At a daily dose of 300 mg, isoniazid

polyneuropathy occurs in 2% of patients, and with an increase in dose - in 10%. In this regard, the appointment
isoniazid requires the obligatory ingestion of 25-30 mg of pyridoxine during the entire treatment isoniazid. There are no situations in which isoniazid would be prescribed without “covering” pyridoxia.
In addition to polyneuropathy, isoniazid can cause nausea, vomiting, skin rashes, arthralgias, euphoria, psychotic reactions, convulsions, hepatitis, pellagra. It is worth mentioning that unlike from the generally accepted practice of prescribing maximum doses of isoniazid (up to 900 mg or more) foreign
the guidelines of recent years recommend 300 mg as the optimal dose, since at the same time the content of isoniazid in the cerebrospinal fluid significantly exceeds the minimum
the level of the tuberculostatic effect of the drug.
Rifampicin is prescribed 600 mg once a day 30 minutes before meals. Possible side effects: fever, skin rash, hepatitis, nausea, vomiting, diarrhea, thrombocytopenia, decoloration of mild contact lenses and orange-red coloration of saliva and urine.
Pyrazinamide is prescribed at a dose of 30 mg / kg per day (1.5-2.5 g) orally after meals in 2 divided doses. Side
effects: arthralgia, hyperuricemia, hepatitis (rare).
In case of insufficient effect (resistant strains of M. tuberculosis) add a fourth
drug - streptomycin 1 g intramuscularly 1 time per day or ethambutol 25 mg / kg per day for months, and then 15 mg / kg per day by mouth in 1 dose after meals. Treatment with streptomycin should not
last more than 3 months due to its ototoxicity. Possible side effects of ethambutol - defeat optic nerve and hepatitis.
Glucocorticosteroids (prednisone 30-60 mg) are indicated in patients with spinal subarachnoid block, as well as with the development of focal neurological symptoms or increased ICP. The risk of using corticosteroids is high.
The duration of treatment can be 18-24 months.
Forecast. The overall mortality rate of patients with tuberculous meningitis remains very significant (about 10%), especially in children of the first year of life and in the elderly. Early diagnosis and early initiation of treatment increase the chances of recovery. 20-30% of survivors patients persist in varying degrees of severity residual defect in the form of a delay psychomotor development, mental disorders, epileptic seizures, oculomotor
disorders, deafness and hemiparesis.
Neurobrucellosis
The natural reservoir of Brucella is animals, especially goats and sheep. Often
infection occurs as a result of eating raw milk and dairy products.
The transmission of the disease can also occur by contact or by air. More often faces are affected having contact with animals (veterinarians, workers of livestock farms).
Pathogenesis. Pathogen through the mucous membranes of the gastrointestinal tract or through

damaged skin enters the lymph nodes, and from them spreads throughout the body.
An important role in the pathogenesis of brucellosis, in addition to the direct effect of an infectious agent,
play autoimmune reactions.
Clinic and diagnostics. The duration of the incubation period ranges from
1-3 weeks, sometimes it can take up to several months. The clinical picture of brucellosis characterized by polymorphism of symptoms and a remitting course.
Distinguish between subclinical, acute, subacute and chronic forms. The initial stage of acute forms: rise in body temperature, malaise, chills, severe sweating, joint pain.
There is pain and swelling of the joints, often of the knee and shoulder. The sizes are increasing liver and spleen, lymph nodes. Undulating fever is characteristic. V
blood is marked by hypochromic anemia, leukopenia, lymphocytosis.

The emergence of brucellosis spondylitis can be complicated by radicular syndrome.
The defeat of the nervous system is extremely polymorphic. Everyone can be involved departments of the central and peripheral nervous system. Often the patient may develop simultaneously two or three different neurological syndromes. Acute, subacute or chronic meningitis, meningoencephalitis, acute or subacute myelitis, polyradiculopathy, polyneuropathy.
Subacute or chronic meningitis lasts for weeks. Episodic exacerbations are characteristic, involvement of the brain substance (meningoencephalitis) and cranial nerves. Increased ICP (possible
stagnant discs) mimics the volumetric intracranial process. In cerebrospinal fluid there is lymphocytic pleocytosis, often very high protein content, possibly a decrease glucose concentration, xanthochromia.
In general, brucellosis meningitis is very similar to tuberculous and fungal. At the heart of diagnosis - anamnestic data about the acute period with characteristic somatic signs, isolation of brucella and serological studies.
Treatment. Use a combination of tetracycline 500 mg 4 times a day by mouth for 6 weeks and streptomycin 1 g per day intramuscularly for 3 weeks or a combination of doxycycline 200 mg 1 time per day by mouth and rifampicin 900 mg by mouth 1 time per day for 6 weeks. For chronic forms usually require repeated courses of antibiotics due to the possible persistence of infection.
Cryptococcal meningitis
Medical mycology is a discipline that is poorly known not only by neurologists and general practitioners.
practitioners, but also infectious disease specialists. This determines the difficulties in diagnosing fungal lesions of the nervous
systems.
Fungal diseases of the nervous system often develop in patients with weakened immune protection after prolonged antibacterial, corticosteroid, immunosuppressive or cytostatic therapy, as well as in patients suffering from malignant tumors, hemoblastosis, diabetes mellitus, AIDS, uremia. Clinical symptoms are mainly is determined not by the specificity of the pathogen, but by the intensity and prevalence of the infectious
process. Diagnosis of a fungal infection is complicated by the presence of the previous main problem.
illnesses. At first, fungal meningitis is more common, mainly of basal localization.
Symptoms indicating damage to the brain substance - meningoencephalitis or encephalitis, usually develop in the later stages of the disease.
The chronic nature of the course of the disease, the similarity of the clinical picture, lymphocytic cytosis in the cerebrospinal fluid makes it necessary in each case to conduct differential diagnosis with tuberculous meningitis.
The most common fungal disease in our country is cryptococcosis, caused by Cryptococcus neoformans.
Pathogenesis. The pathogenesis of this disease is in many ways similar to tuberculosis. Penetration pathogen in the lining of the brain occurs against the background of a decrease in the protective properties of the body and a violation
permeability of the blood-brain barrier. The defeat of the membranes manifests itself in the form of serous
productive meningitis with punctate hemorrhages in the hard and soft shell of the brain. Thickness shells increases, they acquire a cloudy color, appear on their surface
numerous small tubercles, resembling tubercles, developing with tuberculous meningitis. Similar changes occur in the basal parts of the brain, it is not excluded the spread of the process to the brain stem and spinal cord.
Clinic and diagnostics. Clinically, cryptococcosis can occur as subacute or

chronic infection, less often there is an acute course. The development of the disease is accompanied by
an increase in body temperature to 37.5-38 ° C. This temperature can be kept for a long time, decreasing
and rising without a definite pattern. Against this background, lesions of the lungs, skin, ears, nasopharynx.
Damage to the nervous system most often occurs in the form of meningitis or meningoencephalitis. Headache, dizziness, sometimes vomiting, general weakness occur. They are joined by stiff neck muscles, Kernig's symptom. In some cases, stagnant discs are found and focal neurological symptoms, which may suggest a volumetric intracranial process.
Cerebrospinal fluid can be clear, xanthochromic, or cloudy. In it the amount of protein and cells, mainly lymphocytes, gradually increases, usually no more 200 in 1 mm. The glucose content is reduced in 75% of patients.
For diagnosis, detection in the cerebrospinal yeast cell fluids (which is possible in about half of patients) or their sowing and growing on a special nutrient medium.
Cryptococcosis must be differentiated from other forms of subacute and chronic meningitis, especially tuberculous, and from carcinomatosis of the membranes.
In half of cases, cryptococcosis is due to immunodeficiency diseases, which makes the prognosis is absolutely bad. In the other half, cases with an acute onset and high titer of antigen in cerebrospinal fluid.
Treatment. Amphotericin is used at a test dose of 100 U / kg intravenously (the drug is very toxic), and then 250-1000 U / kg intravenously daily or every other day. In severe cases, or in case of toxic damage to the kidneys, they resort to endolumbar administration. The effect improves additional administration of fluorocytosine. The treatment cycle is 6 weeks. Repeated cycles are often needed.
Neuroleukemia
Neurological complications occur in 2-4% of patients with leukemia. CNS damage develops mainly due to infiltration of leukemic cells, but it can be and the result of hemorrhages, infections, drug or radiation therapy, electrolyte devices or disorders of cerebral circulation due to increased blood viscosity. Most a common form of neuroleukemia is meningitis, manifested by headache, vomiting, drowsiness or irritability, epileptic seizures, in severe cases, coma.
Due to infiltration of the ventricular system, obstructive or communicating hydrocephalus, stagnant discs of the optic nerves are often observed.
Leukemic infiltrates can compress or infiltrate the cranial nerves. Most often nerves II, III, VI, VII and VIII are affected. Oculomotor nerve palsy can sometimes occur with preserved pupillary reactions.
The diagnosis of leukemic meningitis is established by detection in the cerebrospinal leukemic cell fluids. In 90% of cases, the number of leukocytes in the CSF increases, and with centrifugation reveals blast cells. The pressure is usually increased, sometimes it is observed a decrease in glucose with an increase in protein. In 10% of cases, changes in CSF absent.
Leukemic meningitis is usually observed with acute lymphoblastic leukemia, less often with acute non-lymphoblastic leukemia, chronic lymphoblastic leukemia and myeloid leukemia.
The possibility of developing neuroleukemia must be taken into account when determining treatment tactics. Neuroleukemia can develop both during remission and in the acute phase. diseases. In these cases, methotrexate is administered endolumbar in combination with cytarabine up to disappearance of neurological symptoms and rehabilitation of cerebrospinal fluid.
The latter is especially important, since neuroleukemia can occur subclioically and to determine in the subsequent relapses of the disease. According to A.I. Vorobyov (1990), "Neuroleukemia is not a clinic, but cytosis."

In addition to meningitis, leukemia can cause damage to the brain matter. Depending on
from the localization of leukemic masses, various symptoms are observed: hemiplegia, aphasia,
hemianopsia, ataxia, cortical blindness, epileptic seizures. Sometimes there is a defeat
hypothalamus, manifested by eating disorders, drowsiness, diabetes insipidus,
obesity. Less commonly, the spinal cord is affected, usually at the thoracic level, with the
development of
complete transverse lesion syndromes, Brown-Séquard syndrome, anterior or posterior

spinal syndromes. Peripheral neuropathy in leukemia is rare, usually in
in the form of isolated or multiple mononeuropathy. In severe cases, usually against the background
of DIC
syndrome, intracranial hemorrhage may develop. With chronic myeloid
leukemia and other types of leukemia often develop a syndrome of high blood viscosity,
manifested by headache, drowsiness, lethargy, hearing impairment, transient
cerebral circulation disorders.
Viral, bacterial and fungal lesions of the central nervous system are often observed. Cases described
progressive multifocal leukoencephalopathy developing against the background of endolumbar
administration of methotrexate and radiation therapy.
Patients with severe thrombocytopenia with lumbar puncture may develop
spinal subdural hematoma, compressing the spinal cord or cauda equina.

Chemical meningitis

Chemical meningitis can occur with endolumbar administration of various substances, with
penetration of the contents of epidermal cysts into the subarachnoid space,
craniopharyngiomas, cholesteatomas. Often, developing meningitis resembles bacterial and
characterized by neutrophilic pleocytosis, a decrease in glucose in the CSF. Described
several cases of serous meningitis caused by treatment with ibuprofen, sulindac,
tolmetin, cotrimoxazole.

Optic-chiasmal arachnoiditis

The question of optic-chiasmal arachnoiditis remains controversial and confusing.
Most cases seem to be secondary and develop as a consequence of a previous injury,
meningitis, encephalitis, hemorrhage, arachnoid cyst, familial optic neuropathy
(Leber type).
Reliable cases of optic-chiasmal arachnoiditis, in which it was not found
the reasons leading to the development of the disease are very rare. It should be added that currently
in
As a result of the development of neuroimaging methods, a significant narrowing of this blurred
diagnostic category.
Monitoring of patients operated on in the past for optic-chiasmal
arachnoiditis, shows that in a considerable number of cases, after a few years, patients develop
detailed picture of multiple sclerosis. In other words, it is precisely polyslerosis that debuts
bilateral visual impairment is the most common reason for erroneous diagnosis of optical
chiasmal arachnoiditis. Equally, little awareness of Leber's disease also leads to
to the erroneous interpretation of bilateral central cattle as a consequence of arachnoiditis.
Given the special tropism of sarcoidosis to the involvement of the hypothalamus and the pituitary
area, it becomes clear why sarcoidous infiltration of the optic chiasm is
one of the main non-oncological causes of chiasmal syndrome.
Antibiotic therapy and microsurgical lysis of adhesions in the cisterna of the chiasm
(chiasmatic cisterna) can sometimes improve vision.

Purulent focal lesions of the brain and its membranes

Brain abscesses

Epidemiology. It is estimated that brain abscesses are diagnosed with a frequency of 1 / 100,000

hospitalizations, and this number has not changed since the middle of the last century, despite the use antibiotics. But the proportion of secondary abscesses has changed: after a chronic ear infection this complication has become less common, however, there is an increase in secondary abscesses with
congenital heart defects and injuries. Abscesses are six times less common than bacterial meningitis.
Persons of all ages can get sick, but a quarter of them are children under 15 years old, since in At this age, congenital heart defects of the blue type are observed more often. In men, the disease occurs 2-3 times more often.
Etiology. Abscesses arising from the paranasal sinuses account for 5-10% of cases, with the location of the primary focus in the frontal sinus, they are usually localized in the frontal lobes, with
ethmoiditis - in the deep parts of the temporal lobes, the causative agent is often streptococcus alone or in combination with other organisms. Otogenic abscesses are usually localized in in the temporal lobe or in the cerebellum, anaerobic flora is found in them in combination with aerobes.
Post-traumatic and postoperative abscesses are usually caused by golden staphylococcus. A very wide range of microorganisms can cause a hematogenous abscess. Sometimes
it occurs in patients with Nocardia asteroides infection of various organs. Brain abscess occurs either by hematogenous route, or due to the spread of infection through contact. Intact parenchyma the brain is relatively resistant to hematogenous infection. Hematogenous abscesses usually occur on the junction of white and gray matter containing terminal arterioles. Relatively weak vascularization of this zone contributes to the emergence of ischemic foci here with occlusion of small
vessels with infectious emboli. In 10% of cases, abscesses occur in persons with congenital defects hearts. Patients with blue heart defects with right-to-left shunt are particularly susceptible to the development of a brain abscess after the age of 2 years, since the lungs do not filter infectious emboli,
coming from venous blood. Abscesses in such patients can proceed without the encephalitis stage. in a pseudotumorous type, which usually leads to an erroneous assumption about a brain tumor. Pulmonary abscesses are a very common source of infected emboli. In general, abscesses hematogenous origin can be the result of an abscess of any localization. Abscesses arising from infections of the paranasal sinuses or ears, are formed due to contact spread of infection through the bone, membranes and intershell spaces. In addition, near the primary focus may develop thrombophlebitis, followed by retrograde spread infections in the brain parenchyma. Microorganisms can be introduced by penetrating trauma or by surgical intervention. The growth of microorganisms in the brain tissue first leads to localization bath encephalitis (cerebritis) with edema, hyperemia and petechial hemorrhages. Then the center the focus undergoes purulent decay, an unencapsulated abscess is formed, surrounded by inflammatory tissue. The abscess tends to spread into a white matter followed by breakthrough into the ventricles and the occurrence of severe ventriculitis. Gradually over the course of several
During these weeks, fibroblasts proliferate around the abscess, forming a capsule.
Clinic. Symptoms of a brain abscess usually correspond to those of an intracranial volumetric process, the headache is often very pronounced. General infectious manifestations are characteristic of the encephalitic stage. As the abscess capsule forms, they can reduce and by the time the diagnosis is established, they are absent in about half of the patients. Perifocal edema quickly leads to an increase in ICP, nausea, vomiting, headache pain, sometimes stunnedness; partial and generalized seizures are frequent. If ICP is not very high, the pulse and respiratory rate remain within normal limits. In many patients it is possible to identify
swelling of the optic nerve head. The asymmetry of the disc edema is not topical. Occasionally you can observe damage to the oculomotor (III) or abducens (VI) nerve as a result intracranial hypertension. Frequent focal symptoms include hemiparesis or

hemiplegia arising from damage to the cerebral hemispheres; spontaneity and confusion, observed mainly with damage to the frontal lobes. Involvement of the temporal and parietal the occipital regions are usually manifested by hemianopsia and aphasisea. A cerebellar abscess leads to
the occurrence of tremor, ataxia, nystagmus and other manifestations of cerebellar dysfunction. Often an abscess is manifested only by signs of intracranial hypertension. Brain stem abscess is rare and its diagnosis is often the lot of the pathologist. Along with neurological symptoms, they can

be signs of a primary disorder, such as heart disease, mastoiditis, sinusitis, pulmonary infections.
If brain abscess is suspected, lumbar puncture is not recommended due to high risk temporal or cerebellar implantation. However, if puncture is still performed, usually increased cerebrospinal fluid pressure, minimal pleocytosis, moderately increased protein content. The glucose concentration is usually normal and the fluid is sterile. Frequent pathological
EEG changes, for example, focal δ -waves with localization of an abscess in the cerebral hemispheres.
MRI and radioisotope scintigraphy are very informative, they can be used to visualize the encephalitic stage of the process, which does not allow CT. However, these methods do not always give
the ability to differentiate an abscess from a necrosis focus with localized herpetic encephalitis, heart attack, or brain tumor. CT and MRI give a clear, detailed picture already formed or encapsulated abscess. At the onset of the disease, encephalitis (cerebritis) manifests itself on CT as a zone of reduced density. Formed encapsulated abscess has a non-uniform density: the central zone of low density is surrounded by a rim of more high density (which corresponds to the area of cell accumulation formed along the periphery and vascular hyperplasia and does not necessarily indicate the presence of a formed fibrous capsules), around this there is an area of reduced density associated with edema of the surrounding fabrics. This structure becomes especially clear after the introduction of contrast. A similar picture can be observed with metastatic and cystic tumors. The angiogram reveals a volumetric process. Often there is simultaneously a primary focus of infection outside the central nervous system, so they can
required hematological, X-ray and bacteriological studies.
Diagnostics. A brain abscess should be suspected in a patient with a focus of chronic infections with the appearance of headache, fever, lethargy, drowsiness, focal neurological symptoms, epileptic seizures. If suspicion arises,
perform an x-ray of the skull and one of the following examinations: MRI, CT, radioisotope scintigraphy or angiography. The above symptoms can lead to false
the assumption of the presence of meningitis, which inevitably leads to . carrying out dangerous in the presence of
large formation of lumbar puncture. When pus is received at the operation, it should be sown on various environments for the detection of aerobic, anaerobic flora, mycobacteria or fungi. At suspicion of the presence of Nocardia asteroides, the study may be delayed, since a long incubation. This microorganism should be suspected in the presence of a cavity or nodular focus in lung, especially if nocardia is sown in sputum. Nocardia is often found in patients with damage to the immune system.
Pronounced changes in the clinical analysis of blood are noted in no more than 20% of patients.
Treatment. Following the diagnosis, broad-spectrum antibiotics are prescribed.
Drugs are selected taking into account their ability to penetrate into the abscess and into the surrounding
parenchyma, as well as the spectrum of their activity. Levomycetin is effective against a large number of microorganisms
organisms, including most gram-positive and gram-negative aerobic bacteria and

most of the anaerobic. It penetrates well into the brain parenchyma and can accumulate here, reaching a concentration higher than in blood plasma. However, the degree of its penetration into the abscess
unpredictable. Penicillin works well against most gram-positive
microorganisms, with the exception of many strains of Staphylococcus aureus. The extent of it brain penetration and abscess are also variable. If you suspect the presence of golden staphylococcus usually use semi-synthetic penicillins and cephalosporins of the third generations. Sulfanilamide drugs are effective against nocardia. Metronidazole is well administered nicks inside the abscess and has a fast bactericidal effect on anaerobic microorganisms.
It is especially useful in the treatment of otogenic abscesses. Antibiotic therapy is not carried out less than 6 weeks However, it must be borne in mind that microorganisms can continue to grow inside the abscess.
even if there are such concentrations of antibiotic that lead to the death of microbes when lack of pus. The presence of an abscess is an absolute indication for surgery - drainage or removal of the abscess. Exceptions include the following situations; process detection at stage encephalitis; deep location of the abscess, which creates technical difficulties and danger significant postoperative disability; the presence of multiple abscesses that
impossible to drain; location of the abscess in critical areas. The operation can be performed immediately after diagnosis if CT shows signs of abscess encapsulation.
During the intervention, it is very important to obtain material for sowing. If satisfactory the condition of patients, the operation can be delayed until encapsulation. There are reports of successful

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conservative treatment by prescribing antibacterial drugs. When conducting
Conservative treatment requires repeated CT to monitor the adequacy of therapy.
In addition to the usual surgery for excision of an abscess, a technique for aspiration and drainage of the abscess by CT-guided percutaneous puncture. When localizing
abscesses in deep sections or functionally significant areas puncture aspiration and introduction in an abscess, antibiotics are sometimes the only method that can save the patient's life.
Forecast. The mortality rate is 15-25%. The lowest mortality is observed in patients who received antibiotics and underwent surgery, that is, with combined treatment. 50% survivors, there are residual effects in the form of focal disorders and epileptic seizures.

Infectious lesions of the dura mater of the brain (epidural abscess, subdural empyema, septic sinus thrombosis)

Etiology and pathogenesis. Most epidural brain infections originate from
external bone structures: paranasal sinuses (usually frontal or ethmoid), mastoid process, middle ear, skull bones. A hematogenous route of infection is possible, as well as its occurrence as a complication of surgical interventions and penetrating trauma. When
external bone structures are the source of infection, the dura mater is the first
a barrier to microorganisms and can contain the infection for some time
within the epidural space. Due to the fact that the hard shell is tightly attached to periosteum, the focus occupies a small volume, representing an accumulation of pus that separates the periosteum from the hard shell. This type of lesion is defined by the term “epidural abscess ".
Through veins that perforate the hard shell, pathogens can enter
subdural space, through which the infection spreads widely until meeting with
structures limiting it, for example, the tentorium of the cerebellum, forming a subdural empyema, i.e. accumulation of pus between the hard and arachnoid membranes. In some cases, a breakthrough of pus is possible
inside the parenchyma of the brain with the formation of a brain abscess.
More often than others, epidural abscess is caused by aerobic and anaerobic streptococci (in 50% cases), other anaerobes (40%), staphylococci (10%). With suppuration of subdural hematoma in the infectious agent is usually gram-negative bacteria.

Clinic. Clinical manifestations are determined by the stage of the process. The beginning may be gradual. Against the background of subsiding symptoms of acute sinusitis or otitis media, or in the process of recovery
after surgery or injury, fever, headache, vomiting, meningismus, disorder
consciousness, generalized epileptic seizures. Symptoms of a primary infection (sinusitis, otitis media,
osteomyelitis) can mask the manifestation of an epidural abscess. Antibiotics are capable
delay the onset of the disease or lubricate the symptoms, but on their own they rarely lead to
cure epidural abscess and subdural empyema. As the infection spreads through
subdural space, ICP rises, the underlying membranes and brain matter are involved,
which leads to the appearance of focal symptoms: hemiparesis, aphasia, partial seizures,
damage to the oculomotor nerves. They may be due to compression of the adjacent cortex.
or a heart attack that occurs with thrombophlebitis of the cerebral veins.
Leukocytosis and increased ESR are common. Sinusitis, mastoiditis, cranial osteomyelitis can
be detected on an x-ray or CT scan. A lumbar puncture can be dangerous due to
increased ICP. Several hundred leu-
cocytes with a predominance of segmented. Protein content may be increased, glucose sometimes
reduced, Cerebrospinal fluid is usually sterile. CT and radioisotope scintigraphy -
the main methods of visualization of the focus of infection, allowing to determine its localization and
length. MRI in the diagnosis of subdural empyema is inferior to CT, since the intensity
the signal from the contents of the empyema and the brain substance may be the same. In some cases, effective
cerebral angiography.
Diagnostics. Combination of meningeal signs and changes in cerebrospinal fluid
can lead to misdiagnosis of meningitis. To avoid this error, remember that
sinusitis, especially frontal sinusitis, usually leads to infection of the hard membrane rather than to
meningitis. In addition, focal symptoms combined with minor changes
cerebrospinal fluid is rare in meningitis, but is more common in subdural

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empyema. Encephalitis, brain abscess, intracranial thrombus
bophlebitis. When conducting differential diagnostics, the most valuable methods are
neuroimaging. In many cases, an epidural abscess is found during surgery
about subdural empyema and brain abscesses.
Treatment. Treatment consists of surgical drainage and antibiotic therapy. Tactics
local irrigation of the subdural space with antibiotics was unsuccessful. Needed
sowing pus on aerobic and anaerobic media to isolate the pathogen and determine the sensitivity
antibiotic resistance. Because of the frequent occurrence of streptococci, it is usually given intravenously
penicillin at a dose of 24,000,000 U per day for adults and 320,000-480,000 U / kg per day for children. When
mixed infection, you need to select antibiotics for each microorganism. Treatment
lasts at least 4 weeks, it is longer in the presence of osteomyelitis.
Mortality in subdural empyema ranges from 25 to 40%. The main cause of fatal
outcomes - delayed diagnosis. Immediate causes of death: cerebral thrombophlebitis
veins and sinuses of the dura mater of the brain, extensive cerebral infarctions, fulminant
meningitis, multiple brain abscesses, progressive cerebral edema.
When the infection passes to the sinuses of the dura mater, their thrombosis occurs. Often
in this case, paired sinuses are affected (lateral, cavernous, stony), while
non-infectious thrombosis (during pregnancy, malignant neoplasms, blood diseases,
use of contraceptives, collagen diseases) more often occur in unpaired sinuses. Infection
sinus can occur by contact (from otorhinogenic foci), with periphlebitis, and
by septic venous embolization. From the sinuses, the infection can spread to epi- and
subdural spaces, pia mater, adjacent brain matter, as well as -

by hematogenous route - to get into various organs and tissues.
Sinus thrombosis leads to increased intracranial pressure, multifocal ischemia
brain (due to impaired venous outflow) and the formation of multiple heart attacks.
Lateral sinus thrombosis usually complicates acute or chronic otitis media, mastoiditis. More often he
observed in childhood. Clinically, the disease is manifested by fever, headache,
vomiting. Pain, usually bursting in nature, is localized in the ear. In the area of the mastoid
appendix often observed venous plethora, edema, hyperemia, tenderness of the veins on palpation,
sometimes inflammation can go to the jugular vein, leading to a limitation of neck mobility.
Intracranial pressure is increased, especially when the larger right sinus is affected.
Bilateral edema of the optic discs is usually observed. Drowsiness develops
gradually turning into a coma. Convulsive epileptic seizures may occur. Focal
manifestations usually do not occur.
When the infection spreads to the lower stony sinus, lesions of VI and V develop
nerves (Gradenigo syndrome), with damage to the jugular bulb, damage occurs IX, X, XI
cranial nerves.
Cavernous sinus thrombosis usually occurs when there are primary foci in the orbit,
sinuses, ear cavity, upper half of the face. The infection can enter the anterior part of the sinus (by
orbital veins supplying the orbit, frontal sinus, nasal cavity, upper face),
in the middle part (through the wedge-shaped sinus, pharyngeal and pterygoid plexuses, blood supply
pharynx, upper jaw, teeth), into the back (through the stony sinus, sometimes through the ear veins and
through the lateral sinus). In the first case, the most acute course of the process is noted, with the posterior path
spread is more chronic. The causative agent is most often Staphylococcus aureus.
The condition of patients is usually severe, there is a high fever, headaches, vomiting,
lethargy, epileptic seizures, tachycardia. Local changes include: chemosis, edema, and
cyanosis of the upper part of the face, especially the eyelids and the base of the nose, due to impaired outflow along
orbital veins. The superficial veins in the forehead are dilated. Hyperemia of the con-
junctiva, photophobia, ophthalmoplegia, initially associated with damage to the VI nerve, dilated pupils
due to the predominant damage to parasympathetic fibers or constriction of the pupils with
simultaneous involvement of sympathetic and parasympathetic fibers. The first may be amazed
branch of the V nerve. Hemorrhages in the retina, edema of the optic discs are observed. Visual acuity
normal or slightly reduced.
Thrombosis of the superior sagittal sinus occurs less often as a complication of a purulent infection.
Infection usually occurs from the nasal cavity or by contact with osteomyelitis,
epidural or subdural infection. Clinically, in addition to cerebral and general infectious
symptoms, there is swelling of the forehead and anterior parts of the scalp, sometimes plethora

scalp veins. In some cases, epileptic seizures, paralysis (hemiplegia, lower
paraplegia), homonymous hemianopsia, alexia, apraxia, etc.
Diagnostics. Craniograms may show signs of osteomyelitis, otitis media,
sinusitis, etc. Cerebral angiography, radioisotope
scintigraphy. CT scan often reveals foci of hemorrhage with increased density, in
sine projection. Contrast-enhanced CT may show a defect in the case of thrombosis
filling.
Treatment. Antibiotics in high doses. Since the disease is more often caused
staphylococcus, it is recommended to use semi-synthetic
penicillins. Remediation of the focus of primary infection. Due to the risk of hemorrhagic
complications

anticoagulants are contraindicated. The prognosis for life with adequate therapy is favorable, however
often a residual neurological defect persists.

Neurological complications of infective endocarditis

Infective endocarditis is a septic disease with localization of the main focus of infection
on the valves of the heart, less often on the parietal endocardium. By the rate of development of clinical manifestations
distinguish between acute and subacute
forms of the disease.

Etiology. Acute infective endocarditis that occurs in individuals with unaffected valves
is more often caused by Staphylococcus aureus, less often by streptococci, pneumococci, enterococci, in
In recent years, gram-negative bacteria are increasingly common as pathogens
(Escherichia, Pseudomonas aeruginosa, etc.) and Pseudomonas. Subacute infective endocarditis occurs, as a rule, in an already affected heart: against the background of rheumatic or congenital malformations
heart, especially in the presence of an open arterial (botallov) duct, defect
interventricular septum, tetralogy of Fallot, aortic and mitral stenosis, as well as after
cardiac surgery: suturing artificial valves, pacemaker installation,
imposition of systemic arterial shunts; less often the disease occurs against the background of prolapse
mitral or tricuspid valve. The causative agent of subacute infective endocarditis
more often Streptococcus viridens. In addition to these microorganisms, cause the development infective endocarditis can be rickettsia, Listeria, Salmonella, Haemophilus and fungi (Candida, Histoplasma, Aspergillus).
Various diseases, as well as therapeutic and
diagnostic manipulations accompanied by bacteremia: abscesses of various localization, osteomyelitis, pyelonephritis, pneumonia, otitis media; dental manipulations, urological, hy-noncological, proctological studies, etc. An important role in the development of the disease is decreased immune reactivity, which can occur with collagenoses,
immunosuppressive therapy for malignant tumors, diabetes mellitus. In recent years
due to the spread of immunodeficiency states and drug addiction, the spectrum of excitation bodies of the disease. It is increasingly caused by low pathogenic conditions under normal conditions.
microorganisms resistant to standard therapy.

Pathogenesis.

The clinical manifestations of infective endocarditis are due to
a combination of four main pathogenetic mechanisms: the formation of valvular: vegetation, embolism, leading to the development of ischemic lesions of various organs, dissemination with the formation of foci of metastatic infection, the formation and circulation of immune complexes. For lesions of the nervous system, in addition to the embolism mechanism, leading to the development
heart attacks and abscesses of the brain, the formation of mycotic aneurysms is important (the term "Mycotic" was first suggested by V. Osler in 1885 when describing endocarditis). Aneurysms are a consequence of vasa vasorum embolization, leading to a softening of the vascular wall. Usually aneurysms are located in the basin of the middle cerebral artery, more often on more distal vessels; they can quickly grow and burst both into the substance of the brain and into the subarachnoid space. It has been shown that after drug treatment, aneurysms can disappear. Brain abscesses, often feminine, usually resulting from infection of microinfarctions.

Clinic. Specific manifestations of infective endocarditis develop against the background general infectious symptoms: fever, often of the wrong type, accompanied by chills
and night sweats, weakness, weight loss, arthralgia. Possibly prolonged febrile
flow. Pallor of the skin and mucous membranes is characteristic.
Petechial hemorrhages are common, especially on the conjunctiva and under the nail

plates. On the fingers, palms, in the lower leg area, bright red or
purple, painful nodules (Osler's nodules). As a result of embolism of the kidneys, lungs, spleen
there is a defeat of these organs; splenomegaly is present in almost half of cases. At
auscultation of the heart marks the appearance of new or changes in existing murmurs.
Neurological manifestations are observed in 20-40% of cases of infective endocarditis.
Often it is with them that the disease begins; in some cases, neurological symptoms remain his
the only manifestations, and heart damage is found only at autopsy.
In a third of cases, neurological manifestations occur as a result of embolic stroke.
Generally, the more virulent the microbe, the earlier the stroke occurs. Embolism usually
develops in the basin of the middle cerebral artery, which leads to the occurrence of hemiparesis,
hemihypesthesia, aphasia (with damage to the dominant hemisphere), etc.
trunk and spinal cord. Small emboli can cause transient ischemic attacks.
In 5% of cases, as a result of rupture of mycotic aneurysms, intracerebral or
subarachnoid hemorrhages, often fatal. In 1-4% of cases
brain abscesses are formed. In 5% of cases, purulent meningitis occurs. Epilepsy is often noted.
tic seizures, both partial, resulting from a cerebral embolism, and generalized,
in the origin of which structural damage, as well as toxic
the effect of drugs, especially penicillin, especially in conditions of renal failure.
In 20% of cases, the so-called "toxic encephalopathy" or "embolic
encephalitis". This condition is characterized by impaired consciousness (usually confusion), mental
changes (confabulations, disorientation, personality changes), epileptic seizures. Wherein
there is no gross focal neurological defect. Pseudobulbar
manifestations. Various factors play a role in the pathogenesis of this complication, primarily
repeated
multiple microembolisms in small arteries and arterioles, which leads to diffuse lesions
brain.

Diagnostics. In the clinical analysis of blood, an increase in ESR, normochromic
anemia, leukocytosis (with acute endocarditis) or leukopenia (with subacute endocarditis). Often
proteinuria and hematuria meet. Repeated blood cultures are essential for
identifying the pathogen; their information content is higher if they are carried out before the onset of
antibacterial
therapy. To clarify the nature of heart damage, electrocardiography is used,
echocardiography, chest x-ray.
To detect neurological complications, CT or MRI is important to detect
foci of heart attacks, abscesses, intracerebral and subarachnoid hemorrhages. Cerebral
angiography reveals vascular occlusion by emboli, as well as the presence of mycotic aneurysms.
When examining cerebrospinal fluid, signs of both purulent and
serous meningitis.

Treatment. Antibiotic therapy forms the basis of treatment. Antibiotics are administered
intravenously in large doses. The choice of antibiotics is carried out taking into account the type of
pathogen and its
sensitivity. The most commonly used penicillin (12,000,000-24,000,000 U / day),
ampicillin, gentamicin, vancomycin, cephalosporins, etc. Duration of treatment
is determined by the patient's condition and is usually at least 6 weeks. Treatment of neurological
The incidence depends on the type of damage. Anticoagulants are generally not recommended due to
with the risk of hemorrhagic complications. For meningitis and abscesses, an appropriate
antibiotic therapy. The need for surgical intervention for abscesses arises
rarely, since they are usually multiple and small. In the presence of single surface
aneurysms, an increase in the size of aneurysms, which is verified angiographically, some authors
recommend surgical treatment.

Forecast. Depends on the age, the general condition of the patient, the timeliness of the start of
treatment,

the severity of cardiac pathology, the type of pathogen. High lethality is caused by staphylococci, enterococci, gram-negative bacteria and fungi. In the absence of neurological complications mortality is 20%, with their development increases to 50%, and in the presence of mycotic aneurysms reaches 80%.

Nocardiosis

Nocardiosis is an airborne infection that hematogenously gets into various organs, including the central nervous system. The causative agent is Nocardia, a microorganism close to actinomycetes. Complications from the central nervous system occur in 30% of cases, most often they are single or

multiple brain abscesses of various localization. Purulent meningitis is less common, osteomyelitis of the spine with the formation of an epidural abscess and compression of the spinal cord. V

In 50-80% of cases, there are predisposing factors: immunosuppressive therapy, collagenoses, malignant tumors. Diagnostics is based on the detection of nocardia in purulent foci, for which biopsy is sometimes necessary. Treatment is as follows: Biseptol (Bactrim) 15-20 mg / kg per day by mouth.

Mortality reaches 80-90%, it decreases with early diagnosis and adequate antibacterial therapy.

Actinomycosis

The causative agent of the disease is Actinomyces Israeli - special acid-resistant, gram-positive microorganisms that occupy an intermediate position between bacteria and mushrooms. A. Israeli is normally a saprophyte and lives in the oral cavity, multiplying in carious teeth. If the skin or mucous membrane is damaged, bacteria can invade tissues, causing depending on the place of introduction, the corresponding clinical manifestations, which fit into three main clinical forms: cervicofacial, thoracic, abdominal. Characteristic education subcutaneous dense infiltrates of bright red color with a cyanotic shade, followed by abscess formation, breakthrough of pus and formation of fistulas, purulent discharge from which is usually has a greenish tint interspersed with yellowish grains. Propagation process can occur contact through tissue cracks, as well as hematogenous. Neurological complications in actinomycosis are rare (no more than 3%). The most common are brain abscesses of various localizations, which can be single and multiple, osteomyelitis of bones occurs less often skulls, epidural abscesses, diffuse dural granulomas, purulent meningitis. Can develop osteomyelitis of the spine, epidural abscesses in the spinal canal with compression spinal cord, often combined with subcutaneous abscesses and paravertebral fistulas. Microscopic examination of pus is of diagnostic value, drusen are detected actinomycetes. Treatment is carried out with penicillin at a dose of 24,000,000 U / day for at least 8 weeks (sometimes it extended to 5 months). Single abscesses can be removed with surgery.

Epidural abscess

Spinal epidural abscess is a rare condition. Occurs with a frequency of 0.2-1.2 cases per 10,000 hospital admissions. The rarity of the disease, as well as the nonspecificity of its clinical manifestations often lead to delayed diagnosis.

Etiology and pathogenesis. The pathogenesis of spinal epidural abscess is somewhat different from the pathogenesis of intracranial abscess. First, there is a difference in pathogens (predominance staphylococci - 50-60% of all cases - and gram-negative bacteria with spinal abscess). Secondly, the dura mater of the spinal cord is not attached to bone structures. This allows infections spread intensively rostrally and caudally. Spinal epidural abscess can be acute and chronic. Most acute cases are the result of hematogenous skidding. Common sources of infection: boils, urogenital infections, dental diseases, chronic lung disease, septicopyemia. They account for 1/3 of all acute cases.

Contact abscesses in diseases of the tissues adjacent to the spine account for another 1/3 of acute and half of the chronic abscesses. Causes of chronic abscesses: osteomyelitis of the spine, pharyngeal infection, retroperitoneal abscess, abscess of the psoas muscle, bedsores, suppuration after surgery. Sources of abscess in children: skin infection in perineum, endocarditis, pharyngitis, urogenic infection. Often, the cause of the disease remains unexplained. Preceding back trauma is surprisingly common (20-30%).
An epidural abscess often develops in the midthoracic and lower lumbar regions, where the epidural space is best defined. As a rule, it forms in the rear space, since the anterior is relatively narrow and avascular. Less common anterior abscesses are usually develop with infection of the pharynx, peritonsillar spaces, mediastinum, with osteomyelitis spine. A spinal epidural abscess is generally uncommon to collapse hard shell and the development of purulent meningitis, although in very advanced cases with anterior an abscess such a course of events is possible.
Clinic. There are four stages of development of an acute abscess. The first stage is characterized by presence of back pain at the level of the lesion, high fever, chills and local rigidity

muscles. At the second stage, the pain takes on a radicular character and is accompanied by a change reflexes according to the level of damage. The third stage begins with the appearance of paresis, which paresthesias and pelvic dysfunctions are often associated. Then paralysis develops rapidly, which corresponds to the transition to the fourth stage. The rate of progression in these stages is different.
In acute cases, paralysis can develop after a few days, in chronic cases - after a few weeks. Shell symptoms and headache are common. In the area of the abscess, hyperemia and edema are possible.
In chronic epiduritis, as a rule, there is no fever and general phenomena, focal signs can build up for months.
In the blood, there is a high leukocytosis and increased ESR. Half of the cases are positive blood cultures. Cerebrospinal fluid is sterile and is usually high in protein, predominantly lymphocytic pleocytosis, 200 cells per 1 mm. The glucose content is normal.
Radiographs sometimes show signs of osteomyelitis. Highly informative CT and MRI. At myelography reveals a complete (80%) or partial block. Myelography helps differentiate chronic epidural abscess from arachnoiditis or epidural tumor.
Diagnostics. The rapid establishment of a diagnosis, despite all its need, is often due to subjective or objective reasons are not easy. This is due to the relative non-specificity symptoms. Acute epidural abscess is often mistaken for meningitis, perrenal abscess, rheumatoid spondylitis, common acute back pain, acute poliomyelitis, subcutaneous hematoma, hematomyelia, polyneuritis, spinal osteomyelitis, transverse myelitis. Differentiation with transverse myelitis is most difficult because of the similarity of symptoms. With transverse myelitis a neurological defect sometimes develops without back pain; about the possibility of developing an abscess should be remember for concomitant osteomyelitis, recent pyogenic infection, positive cultures blood. In all doubtful cases, at the slightest suspicion of epiduritis, it is indicated suboccipital myelography with water-soluble contrast. If possible, a reliable diagnosis Nose epiduritis will be provided by CT and MRI.
A chronic abscess is commonly mistaken for a spinal cord tumor, osteomyelitis spine, Pott's disease with compression myelitis. Other misdiagnoses: protrusion disc, pyelonephritis, hysteria, vertebral disc infection, shingles. Needed studies are usually not performed until the correct diagnosis can be suspected.
The first diagnostic tests were spondylogram and myelography. On the x-ray you can see signs of osteomyelitis, but the normal picture does not exclude the diagnosis of an abscess.
If you suspect epiduritis, a lumbar puncture deserves special attention. There is no other non-

urological situation, where this routine procedure would be such a real threat to development purulent meningitis. Puncture of the epidural space in order to verify the abscess went into past. Lumbar puncture is indicated only for epiduritis located above the lower thoracic level, ascending myelography is performed at the same time. Suspected lower thoracic epiduritis or lumbosacral localization - an absolute contraindication to a lumbar puncture! Risk introducing pus with a needle into the subarachnoid space with the development of purulent meningitis with this
localization is exceptionally great. Shown suboccipital puncture with injection water-soluble contrast. If possible, CT or MRI instead of lumbar puncture.
Blood culture is required.

Treatment. Urgent surgical intervention with drainage of the focus. During the operation, the focus takes on average 4-5 segments. Severe cases with the spread of the abscess are also possible hardly not along the entire length of the epidural space.

In half of the cases, the causative agent is Staphylococcus aureus. Second in frequency pathogens - gram-negative bacteria (escherichia, pseudomonas). The antibiotic must match the seeded pathogen. When the causative agent is unknown, therapy should be directed against penicillin-resistant staphylococci and other pathogens that can be suspected of being the cause of the disease. Antibiotics should be administered for at least 2-4 weeks. At
in the presence of osteomyelitis, treatment should be carried out for at least 6 weeks.

Forecast. Without surgery, the disease ends in death. Recovery rate after surgery depends on the duration and severity of the disease. Most patients after surgery completely recover. Others may have a residual defect. Often spinal cord destruction turns out to be more significant than could be expected in terms of the degree of compression, this is due to
with vascular occlusion in the area of abscess and spinal stroke.

ACUTE ENCEPHALITIS

Using the term "encephalitis", it is necessary to constantly remember that categorical the division of CNS infection on an anatomical basis is far from always possible. For any the infectious process, different structures are affected, albeit to varying degrees nervous system. In this regard, terms such as meningoencephalitis or encephalomyelitis, more accurately reflecting the anatomical distribution of the pathological process. At the same time, in each case it is necessary to clearly determine which of the parts of the nervous system:
the brain or spinal cord or their membranes - suffers the most. This is what is often gives a clue to the diagnosis.

There are primary and post- or parainfectious encephalitis. At the heart of the development of primary
encephalitis is brain damage caused by the direct penetration of an infectious agent across the blood-brain barrier. The so-called post-infectious or para-infectious acute encephalitis is characterized by perivenous infiltration and diffuse demyelination arising as a result of immune reactions. Approximately 50% of all cases encephalitis are parainfectious lesions. A very large number of microorganisms can become cause of acute encephalitis. However, in developed countries, the vast majority of cases primary encephalitis is caused by a viral infection.

With encephalitis, as a rule, meningeal signs are detected. In addition to such signs such as headache, fever and stiff neck muscles with encephalitis
signs of damage to the parenchyma of the brain are found: impaired consciousness from mild confusion to deep coma, changes in higher mental functions, disorientation. Frequent behavioral disorders accompanied by hallucinations. Focal neurological signs (pyramidal symptoms, coordination disorders and sensory disorders) can
meet in the most diverse combination.

In differential diagnosis, a thorough physical examination can
be useful. In particular, a variety of skin rashes are characteristic of enteroviral,
rickettsial, herpes and mycoplasma infections. Mumps is detected only in half
cases of meningoencephalitis caused by the mumps virus.
When conducting differential diagnostics, the primary task is to exclude
conditions clinically similar to acute encephalitis: tuberculous and fungal meningitis;
brain abscess, cerebral vasculitis, systemic lupus erythematosus. If an abscess is suspected
Brain CT should precede the lumbar puncture.
When examining cerebrospinal fluid, increased pressure is detected,
lymphocytic pleocytosis (from 10 to 2000 cells per mm); with extensive foci of necrosis,
erythrocytes. Inflammatory lesions can be found on CT and MRI.
Identification of an infectious agent is carried out using bacteriological and
serological methods. The etiology of acute encephalitis remains unclear in about 50%
cases. Attention is drawn to the scatter of statistical data from various sources. Total number
patients in WHO statistics 42 651, USA - 1662, Institute of Poliomyelitis, Russian Academy of
Medical Sciences - 270, M. Koskiniemi
- 412.
Treatment is carried out according to general principles of intensive care, including
measures to combat cerebral edema and anticonvulsant therapy. Application question
corticosteroids for encephalitis is still unresolved, since no convincing
data in favor of their effectiveness. Specific treatment is possible only in rare cases.

Herpetic encephalitis

Herpetic encephalitis is the most common and severe form
sporadic acute encephalitis. It occurs evenly throughout the year in all countries.
of the world, affecting people of all ages, but more often (in 1/3 of cases) persons over 40 years old
get sick. V
the vast majority of cases of encephalitis is herpes simplex virus type 1
(HSV-1), which also causes herpetic lesions of the oral mucosa. Virus
herpes simplex type 2 (HSV-2) causes a rash in the genital area and is sexually transmitted
by; he is the causative agent of encephalitis in newborns, the infection of which occurs from
mothers with active genital herpes during passage through the genital tract.
Herpes simplex virus is widespread: in about 70-90% of adults
detect antibodies to it. Apparently, the virus is able to persist for a long time in the body.

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a person, being in a latent state mainly in the neurons of sensory nodes and
becoming more active in certain conditions. It is still unclear, however, whether encephalitis occurs
in
as a result of primary infection with a virus or due to its reactivation.

Pathomorphology and pathogenesis. Pathomorphologically, herpetic encephalitis is
acute necrotizing asymmetric hemorrhagic process with lymphocytic and
plasmacytic infiltration. In the acute stage of the disease, neurons and glial cells are
fire intranuclear eosinophilic inclusions. Typical for herpes encephalitis
is the predominant localization of the lesion in the medial part of the temporal lobes (especially in
areas of the hippocampus) and in the lower (orbital) part of the frontal lobes. In the residual stage
diseases at the site of necrosis cystic cavities are formed. Such a unique localization
lesions, possibly due to the characteristics of the spread of the virus. It is believed
infection of the central nervous system can occur through the olfactory bulbs or as a result of
activation
viruses persisting in the trigeminal nodes, from which they spread along the fibers,
innervating the vessels of the meninges.

Clinic. The clinic of herpetic encephalitis as a whole does not differ from the clinic of any
other acute viral encephalitis: usually there is an increase in body temperature, headaches
pain, vomiting, stiff neck muscles. At the same time, relatively typical for this disease

are generalized epileptic seizures already at an early stage of the disease and especially
focal symptoms indicating damage to the temporal and frontal lobes: olfactory and gustatory
hallucinations, anosmia, partial complex epileptic seizures, behavioral disorders,
memory disorders, aphasia, hemiparesis. The disease is quite fast (within a few days)
progresses; stupor develops, then coma. In the absence of treatment, in 50-70% of cases fatal
Exodus. Sometimes, with a rapidly growing cerebral edema, a transtentorial insertion of one
or both temporal lobes, leading to deep coma and death from respiratory arrest in
during the first 24-72 hours of the disease.

Diagnostics. When examining cerebrospinal fluid in the case of herpetic
encephalitis there is an increase in pressure, lymphocytic or mixed lymphocytic
neutrophilic pleocytosis (usually 50-100, sometimes up to 500 in 1 mm), a slight increase in
concentration
protein, normal glucose content (it is occasionally possible to decrease it, which creates difficulties in
differential diagnosis with tuberculous or fungal meningitis). In some cases, in
cerebrospinal fluid show erythrocytes and xanthochromia, which reflects
hemorrhagic nature of the lesion. EEG reveals periodic high-amplitude
fast waves in the temporal areas, as well as slow-wave activity 2-3 / s.

Herpetic encephalitis should be differentiated from other viral encephalitis,
Wernicke-Korsakov encephalopathy, subdural empyema, brain abscess, Reye's syndrome, tumors,
parasitic diseases, thrombosis of the superior sagittal sinus. Early on, before
development of pronounced neurological manifestations, the diagnosis can be made using CT and
especially MRI, revealing inflammatory changes, edema, and sometimes minor hemorrhages in
temporal and frontal areas. However, these changes are not found in all cases.

CT and MRI also rule out other diseases. Serological results
studies consisting in increasing the titer of specific antiviral antibodies in repeated
blood and cerebrospinal fluid samples become known too late (on the 10-12th day),
to have a real impact on the diagnosis and treatment process. Based on data from the clinic and
of the above research methods is possible only with varying degrees of probability
suggest the presence of herpetic encephalitis. Reliable intravital diagnosis is possible
only with a brain biopsy. It should be emphasized that the presence of herpes labialis is not
diagnostic sign of herpetic encephalitis.

Treatment. Herpetic encephalitis is one of the few encephalitis in which there is
effective specific therapy. The drug of choice is acyclovir (virolex, zovirax).

The drug selectively suppresses the synthesis of viral DNA, with little or no effect on
replication of host cell DNA. It is administered 3 times a day at a single dose of 10 mg / kg every 8
hours.

Each dose of the drug should be administered for at least an hour to prevent it
precipitation in the renal tubules, therefore, it is recommended to inject the drug by drip,
after diluting it in 50-100 ml of a special solvent or isotonic solution
sodium chloride. An important condition for the effectiveness of acyclovir is its early administration
(as
at least until the development of a coma). The duration of treatment is usually 10-14
days. Acyclovir has low toxicity and is generally well tolerated; among the possible complications

dermatitis or phlebitis at the injection site, nausea, headache are noted. Occasionally observed
bone marrow suppression and liver dysfunction. In 1% of patients receiving acyclovir,
encephalopathy develops, which is difficult to recognize against the background of encephalitis
clinic. A drug
excreted by the kidneys, therefore, in case of renal failure, its dose should be reduced.

Since accurate diagnosis of herpetic encephalitis based on clinical data
almost impossible, and, the introduction of acyclovir, as a rule, is not accompanied by serious
adverse reactions, the appointment of the drug is indicated for any suspicion of herpetic
the nature of encephalitis, that is, in almost every case, severely current acute encephalitis is unclear

etiology. Also important is pathogenetic and symptomatic therapy, directed
to maintain water-mineral balance, fight against cerebral edema, and in the presence of epileptic seizures, including the use of anticonvulsants.
Forecast. Depends on the age of the patient and the degree of impairment of consciousness: the chances of recovery
worse in patients over 30 years old, as well as in patients who are in a coma. Application acyclovir reduces the mortality rate from 70 to 28%, and among surviving patients to increase from 5 to 38% the proportion of those who recover completely or with minor neurological violations. Korsakov syndrome, dementia,
epileptic seizures, aphasia, etc.
Tick-borne spring-summer encephalitis
Tick-borne spring-summer (taiga) encephalitis is an acute primary viral
a disease characterized by a sudden onset, fever, severe damage to the central nervous system. Refers to natural focal human diseases.
Epidemic outbreaks of tick-borne encephalitis were first recorded in 1933-1934.
in the Far East near Khabarovsk. Later it was shown that this disease
found not only in the Far East, but also in Siberia, the Urals, in many regions of Europe
Peysk part of Russia. Natural foci of tick-borne encephalitis were also found in the Czech Republic, Slovakia,
Hungary and Poland.
Etiology. Tick-borne encephalitis virus belongs to the group of arboviruses transmitted
ticks. In the Far East, Siberia and the Urals, these are mainly ticks Ixodes persulcatus, in European regions of Russia and Central Europe - ticks Ixodes ricinus.
The tick-borne encephalitis virus is sensitive to high temperatures and dies when boiled in within 2-3 minutes. At low temperatures and even when frozen, it remains viable.
The virus is sensitive to disinfectants. Is neurotropic and consistently excreted
from the substance of the brain. The disease causes persistent immunity that lasts throughout life recovered from the disease.
The virus enters the human body through a tick bite. Directly get infected from a sick person tick-borne encephalitis is impossible. The virus is found in all organs of the tick, but especially a lot of it
excreted through the salivary glands.
Epidemiology. The favorite habitats of ticks are old forests with high
grass and shrubs that make up a well-defined undergrowth. In nature, the reservoir of the virus are small animals living in the taiga (forest mice, chipmunks, hedgehogs) and birds (goldfinches, blackbirds,
finches). Of domestic animals, goats are the most susceptible to disease. Attacking the wild animals and birds that are carriers of the tick-borne encephalitis virus, ticks absorb together with a virus that enters the salivary glands of the insect. As a result, such a mite becomes
a carrier of the virus, capable of transmitting it to other warm-blooded animals and to their offspring. The virus does not die in the tick's body even in winter. In tick-borne encephalitis endemic foci in local residents who do not have a history of episodes of acute diseases of the nervous system, high titers of antibodies specific for tick-borne encephalitis are found. It is possible that tick-borne encephalitis disease can be very mild, which either goes away
unnoticed, or is regarded as a common cold.
The disease is of a pronounced seasonal nature, which is directly related to the period activity of ticks. For different regions of Russia, this period is different, but falls on the first warm months of the year - April, May, June, July. At other times, the disease is much less common. How as a rule, tick-borne encephalitis affects people who, due to the nature of work, are forced to stay in spring-summer period in the taiga: geologists, lumberjacks, surveyors, hunters. Ticks are especially dangerous for
newly arrived people.
The tick bite is painless. The mite digs deeply into the skin, can drink blood for

several days, increases in size, after which it comes off on its own and falls to the ground.
But usually a person begins to feel itching at the site of a tick bite after it has sucked, therefore the tick is removed earlier. As a rule, patients remember well whether there was a tick bite. On rare occasions
bites can go unnoticed, and when interviewed, patients deny them. When bitten by a tick, the virus falls into the blood of the bitten. There is a definite relationship between the massiveness of infection (i.e., the number of viruses that have entered the bloodstream), the severity of the local reaction at the site of the bite and
the severity of the disease, as well as the duration of the incubation period. Severe local the reaction is more often combined with a severe course of the disease.
There is another way of penetration of infection into the human body - alimentary - with eating raw goat milk. Infection is possible if the virus enters the mucous membrane from contaminated hands, such as by crushing a tick. Naturally disease encephalitis is not observed after any tick bite. It has been proven that even in particularly active endemic foci, only 0.5-5% of all ticks are carriers of the virus.
Pathogenesis. After the virus enters the body through the skin during a bite, it begins multiply in the skin and subcutaneous fatty tissue in the immediate vicinity of the bite site. With alimentary infection, the virus multiplies in the tissue of the gastrointestinal tract, then it enters the bloodstream and hematogenously spreads throughout the body (stage viremia). In the brain tissue, the virus can be detected 2-3 days after the bite, its concentration reaches a maximum by the 4th day. In the future, it gradually decreases.
The incubation period lasts 8-20 days. With the alimentary method of infection, the incubation the period is shorter than 4-7 days. The disease is possible at any age, but more often in 30-40 years. The severity of clinical symptoms, the severity of the course in the eastern regions and in Siberia in to some extent more significant than in the western regions of Russia and other countries. That's why there is an idea of two variants of tick-borne encephalitis - eastern with a more severe current and western - with a benign course. This concept has received its confirmation. in virological studies that detected two different strains of the tick-borne virus encephalitis.
Pathomorphology. There is swelling of the membranes, substances of the brain, numerous hemorrhages, mainly in the area of the brain stem, cervical thickening of the spinal cord. At histological examination reveals the phenomena of acute non-suppurative inflammation with a pronounced
vascular reaction, proliferation of glia and severe degenerative changes in ganglion cells. The most pronounced inflammatory reaction in the anterior horns at the level of the cervical thickening
spinal cord and motor nuclei of the brain stem. Inflammatory changes are also noted in the cerebral cortex, subcortical formations, cerebellum.
Clinic. The disease, as a rule, begins acutely with a sharp rise in temperature to 39-40 ° C, chills, severe headache, nausea and vomiting. Consciousness is preserved, but in more severe cases
stunnedness, delirious state is possible. On the 2-4th day of the disease, they join pronounced meningeal phenomena - stiffness of the neck muscles, symptoms of Kernig, Brudzinsky, then
paralysis of the peripheral type occurs in the muscles of the neck, shoulder girdle and proximal regions
hands. Their occurrence is due to the predominant lesion of the anterior horns of the spinal cord. at the level of the cervical thickening. As a result, the patient cannot raise his arms up, spread apart, bend and straighten at the elbow joints. Due to the weakness of the neck muscles, a "drooping" head develops.
This is one of the most characteristic symptoms of the paralytic form of tick-borne encephalitis. Limitation of movement in the legs is possible, but knee and Achilles reflexes are increased, abnormal foot reflexes can often be elicited.

Bulbar syndrome with dysarthria and tongue atrophy is quite common. Quite typical the patient's appearance: hyperemia of the face, neck, injections of the sclera, conjunctiva. Moderate leukocytosis with neurophilia and left shift, increased ESR are noted in the blood. In the cerebrospinal fluid, moderate lymphocytic pleocytosis is detected, insignificant increased protein content.

The temperature decreases on the 5-7th day of the disease. Headache gradually decreases myalgia, meningeal symptoms. At the end of the 2nd week, a period of convalescence begins, which may vary in duration. In some cases, restoration of motor functions may be complete, in others - a gross disabling defect remains: more often weakness remains and atrophy in the muscles of the neck, shoulder girdle, and proximal arms. The hanging head is one of the typical symptoms not only of the acute stage, but also of the residual period.

In addition to the described classic poliomyelitis form of tick-borne encephalitis, there are

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also meningeal, meningoencephalitic, febrile, polyradiculoneuritic forms.

The meningeal form is serous meningitis caused by the tick-borne virus encephalitis. Meningeal symptoms in this form occur early - from the 1st day of the disease on the background fever, intense headache, vomiting. In the cerebrospinal fluid, it is found pleocytosis of a predominantly lymphocytic nature, reaching several tens or hundreds cells. Fluid debridement is slower than clinical improvement. Full recovery comes in 2-3 weeks. In some cases, long-term asthenic syndrome persists. V in recent decades, the proportion of the meningeal form has increased, while paralytic forms were less common.

The meningoencephalitic form, which is more severe than the meningeal form, is characterized by a combination of cerebral and focal neurological symptoms: paresis, cranial lesions nerves, hyperkinesis. When the lesion is localized in the medulla oblongata, a threat may develop. life-giving respiratory and hemodynamic disturbances. Kozhevnikovskaya may develop later. epilepsy (Epilepsia partialis continua). It is characterized by constant myoclonic twitching. in certain muscle groups, against the background of which generalized convulsive seizures. The course of Kozhevnikovskaya epilepsy can be stable, remitting or progreduated.

The febrile form of the disease is characterized by a favorable course with a rapid recovery. The duration of the fever does not exceed 3-5 days. Dominate general infectious manifestations. Cerebrospinal fluid readings remain normal.

The polyradiculoneuritic form is less common than the previous ones, it is characterized by damage to the roots and peripheral nerves. It is manifested by pain along the nerve trunks, symptoms of tension, sensitive and motor loss on polyneuropathic type.

A special variant is encephalitis with a two-wave course. First wave fever usually lasts 3-7 days and is characterized by a mild course, meningeal symptoms are mild, and focal symptoms are absent. The first wave is followed by the period of apyrexia, lasting 7-14 days. Then a second, more difficult flowing develops. febrile wave with pronounced meningeal, and sometimes focal neurological symptoms and lymphocytic pleocytosis up to 100-400 in 1 mm^3.

The course of the disease is acute. However, in a number of cases, a progressive course of tick-borne encephalitis, when after a certain time (from several months to several years) after an acute phase, the severity of focal symptoms increases. The essence of such violations remains unclear.

Diagnostics. In the diagnosis of tick-borne encephalitis, elucidation of epidemiological data, information about the patient's stay in the epidemic focus in the spring summer period. It is extremely important to have indications of a tick bite. Acute stage of the disease should be differentiated from various forms of serous meningitis, influenza, acute poliomyelitis (in

children), Japanese encephalitis (in the Far East). Clarification of the diagnosis is made by carrying out serological reactions: complement binding reactions, neutralization reactions, hemagglutination inhibition reactions. The complement binding reaction gives a positive the result from the 2nd week of the disease, the neutralization reaction - from the 8-9th week. The most prompt method for diagnosing a disease is the method of fluorescent antibodies.

Treatment. Homologous gamma globulin is used as etiotropic treatment, titrated against tick-borne encephalitis virus (injected in b ml intramuscularly daily in within 3 days); serum immunoglobulin obtained from the plasma of donors living in natural foci (on the first day, 3 ml 2 times, in the next 2 days, 3 ml intramuscularly); inactivated culture vaccine against tick-borne encephalitis (1 ml is injected 3 times with at intervals of 10 days); ribonuclease (30 mg every 4 hours intramuscularly in isotonic sodium chloride solution for 4-6 days). In the acute period, sym- automatic therapy, maintenance of water and electrolyte balance, detoxification therapy, with the presence of severe intracranial hypertension - dehydration. In the residual stage, complex rehabilitation therapy.

Forecast. A relatively favorable course of tick-borne encephalitis is observed in the West and in the European part of the country; in the Far East, there are more severe forms with a high (up to 30%)

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lethality.

Prevention. Vaccination with tissue inactivated vaccine plays a leading role.

Active immunization is carried out according to epidemiological indications, the vaccine is administered in 1 ml subcutaneously 3 times in the autumn, then once in the spring, followed by an annual revaccination.

Anti-mite gamma globulin is administered to persons who have undergone bites (adults 3 ml, children 10 15 years - 2 ml) intramuscularly; a week later, the dose can be re-administered.

Mosquito encephalitis

Mosquito (Japanese) encephalitis was isolated as an independent disease after epidemic of 1924, although cases of that form of encephalitis occurred in Japan every summer and have been described back in 1871, the cause of the disease is a mosquito-transmitted flavivirus. For Japanese encephalitis is characterized by a seasonality of occurrence with the largest number of cases in dry hot the weather. It is most commonly found in Japan and Southeast Asian countries. On the territory of our of the country, it is common in the Far East and in the Primorsky Territory. Degeneration is characteristic ganglion cells of the cerebellum, cerebral cortex, basal nuclei, substantia nigra, medulla oblongata and spinal cord. The pia mater is infiltrated with lymphocytes, vessels the parenchyma of the brain is surrounded by monocytes and macrophages.

The onset of neurological symptoms may be sudden or follow a prodromal period lasting 3-4 days and characterized by headache, myalgia, fever, pharyngitis, gastrointestinal manifestations. The headache gradually builds up the stiffness of the muscles of the neck develops. Revitalization or decrease in tendon reflexes, tremors of fingers, lips and tongue, ataxia, pelvic dysfunction and rarely lesion cranial nerves. Severe cases are characterized by delirium, stupor, or coma.

The fever that occurs from the first days resolves after 10-14 days. Heart rate increases in proportion to temperature. Bradycardia is rare.

In the analysis of blood, leukocytosis is noted; in cerebrospinal fluid - lymphocytic pleocytosis, on average up to 100 cells per 1 mm, there were reports of pleocytosis reaching 500 or more

cells. The glucose content is normal. In 1 / 3-1 / 4 cases due to inadequate secretion antidiuretic hormone hyponatremia develops.
The disease is more common in children. Mortality in some epidemics reached 60%. However, this figure is clearly overestimated, since patients with a mild form of the disease do not apply to
hospitals and are not included in statistics.
Possible severe residual neurological defect and mental retardation in children.
The diagnosis can be verified by isolating the virus from blood, cerebrospinal fluid, brain tissue, as well as with adequate immunological tests. The specific treatment is pet.
For prophylaxis, an inactivated vaccine is used.

Epidemic encephalitis Economo

The disease was epidemic at the end of the First World War. Currently is extremely rare. It is apparently caused by a filtering virus. During epidemic encephalitis is divided into two stages - acute and chronic.
The disease can occur acutely, but often begins gradually. The most characteristic symptoms are increased drowsiness and oculomotor disturbances. The patient can sleep in any, sometimes completely inappropriate conditions. It is this feature of encephalitis and served as the basis for designating it as “lethargic encephalitis”.
Oculomotor disorders are manifested most often by diplopia, less often nuclear or supranuclear ophthalmoplegia. Often absent or decreased pupil response to convergence and accommodation.
Extrapyramidal symptoms typical of the chronic stage of epidemic encephalitis, also occur in the acute stage. They can manifest as moderate hypokinesia, tremor, rigidity.
The acute stage of the disease can result in complete recovery. But in significant the number of cases following the acute stage over a period of time, which can be measured from several months to tens of years, a gradually progressive chronic stage occurs. V in some cases, symptoms of the chronic stage may develop without a previous acute stages. It is assumed that in these cases there is a chronic course of the infectious process, characteristic of the so-called slow infections.

A typical picture of the chronic stage of encephalitis is Parkinson's syndrome. Specific Oculogyric crises were considered a symptom of encephalitic parkinsonism in the past. Wherein the eyeballs are retracted upward, and the patient is not able to return them to their normal position. The duration of crises ranges from several minutes to many hours. Currently shown that oculogyric crises often occur in patients with drug-induced parkinsonism.
There are no specific treatments for the acute stage of epidemic encephalitis.
Treatment for post-encephalitic parkinsonism is similar to that for Parkinson's disease.

POLIOMYELITIS AND POLIOMYELITO-LIKE DISEASES

Polio

Poliomyelitis (syn. Epidemic infantile paralysis, Heine-Medina disease) - acute an infectious disease of viral etiology, the causative agent of which, entering the nervous system, selectively affects certain neuronal populations. As a result, the disease develops. treatment with a specific clinical picture characterized by the onset of flaccid paralysis and muscle atrophy.

Epidemiology and etiology. The poliomyelitis virus belongs to picornaviruses (group enteroviruses) and is an RNA-containing virioi. The virus is resistant to low pH levels, therefore it spreads easily by water (including through rivers and sewers). By antigenic characteristics distinguish three types of virus (types 1, 2 and 3); there is no cross-immunity. From of the three strains, type 1 is the most virulent.
In rare cases, the causative agent of poliomyelitis-like syndrome can be ECHO viruses, Coxsackie virus and the causative agent of mumps.
The virus is ubiquitous. In mid-latitudes, poliomyelitis is characterized by

seasonality: most cases of polio infection are asymptomatic. In an unfavorable
the hygienic environment is most often affected by children aged 2-4 years.
The age of patients at the time of the onset of the disease affects its severity. Among small children, 1 in 1000 infections lead to paralysis, compared to 1:75 in adults. Bulbar
the form of poliomyelitis is more common in adults. Over the past 30 years, due to widespread
the introduction of preventive vaccinations, the number of cases of the disease has dropped sharply. Should have in
the existence of other enteroviruses that can cause poliomyelitis-like
syndromes in the vaccinated population.
Pathophysiology and pathomorphology. In typical cases, infection occurs
alimentary way. Initially, unimmunized individuals develop gastrointestinal
intestinal tract. Secondary replication of the virus occurs in the tonsils, lymph nodes and Peyer's patches, which leads to viremia. In this phase of the disease (which lasts 1-5 days) nonspecific signs of the disease appear: slight gastrointestinal
upset, slight malaise, fever. In more than 95% of cases, the disease stops
at this stage. However, occasionally the pathogen due to viremia enters the central nervous system. The virus has a special
tropism for motor neurons. The anterior horns of the lumbar spine are most severely affected.
the spinal cord, and also, to a lesser extent, the cervical and thoracic spinal cord. V
the pathological process also involves the nuclei of the brain stem (polioencephalitis), cerebellum, thalamus, hypothalamus, precentral parts of the cerebral cortex.
The reasons for the predominant involvement in the pathological process of these particular departments
nervous system are unknown. It has been established that the virus needs special receptors for penetration into the cell. It is believed that the virus infects cells with the highest concentration traction of these receptors on its membrane.
Clinic. The incubation period is 3-35 days, with an average of 17 days.
Preparalytic or non-paralytic forms. In the preparative stage, you can
distinguish two phases. Initial symptoms are fever, malaise, headache,
drowsiness or insomnia, sweating, flushing of the skin, hyperemia of the pharynx, often gastrointestinal
intestinal disorders (anorexia, vomiting, diarrhea). This phase lasts 1-2 days, then it is possible temporary improvement in the condition with remission of fever for 48 hours and transition to the next phase, when
the headache grows and pain in the back, extremities, sometimes soreness in the muscles joins. Symptoms resemble some forms of viral meningitis.
The stiffness of the neck muscles and Kernig's symptom are determined, and delirium may develop. At

in non-paralytic forms, the patient recovers; transferring in one form or another one or both phases preparative stage.
In the second phase, changes are found in the cerebrospinal fluid that are characteristic of meningitis. The pressure is increased, there is cytosis, usually 50-250 cells per mm. At the beginning there are
both neutrophils and lymphocytes, after the 1st week of the disease, only lymphocytes are determined.
Protein and globulin fraction are moderately increased, glucose content is normal. During the 2nd week
protein can be as high as 1 g / l.
Paralytic forms.
The development of paralysis, often preceded by muscle
fasciculations, usually observed immediately after the preparative stage and is accompanied by severe pain in the limbs and muscle soreness when compressed. In some cases, the duration

the preparative phase can be 1-2 weeks. Paralysis can be common or
localized. In severe cases, the muscles of the neck, trunk, and both arms and legs are affected. At
the smaller the volume of the lesion, the asymmetric “spotted” character comes to the fore
paralysis. Paralysis usually reaches its maximum after 24 hours. In the ascending form,
consistent involvement of the legs and further overlying departments; life threatening
damage to the respiratory muscles. There is also a descending form.
The disease leads to the temporary loss of function of many cells of the anterior horns, which
then they are restored. Improvement usually occurs by the end of the 1st week after the start
paralytic stage. As with other diseases that cause peripheral paralysis
motoneurons, with poliomyelitis, weight loss of the affected muscles and loss of skin and
tendon reflexes. Complete paralysis of the muscles surrounding the joint can lead to subluxation.
Asymmetric paralysis of the vertebral muscles causes scoliosis. The affected limbs are bluish and
cold, often with symptoms of edema. Fasciculations can be observed for a long time in
partially paralyzed muscles.
In the affected limbs, bone growth slows down; when radiography in the bones are determined
signs of osteoporosis.
In some cases, paralysis of the muscles of the face, pharynx, larynx, tongue, oculomotor muscles
develop
muscles. Dizziness and nystagmus may occur. The threat of damage to the respiratory and
cardiovascular center.
Post-polio may develop 20-50 years after paralytic poliomyelitis.
syndrome. It is characterized by late onset muscle weakness and amyotrophy.
The disease has a slowly progressive or gradual course. EMG and muscle biopsy
indicate active denervation. So far, no reliable data has been received on
persistence of the virus. It is believed that after the acute stage of poliomyelitis, intact neurons
provide innervation of fibers denervated as a result of the disease due to the appearance
new terminals. Years later, however, the stability of the new extended motor
units.
Diagnostics. The diagnosis is rarely possible with an abortive course or in
preparative stage, except in cases of epidemics. Polio should be suspected if flaccid
asymmetric paresis is combined with pleocytosis in the cerebrospinal fluid. Isolation of the virus
from
pharynx, feces, or from the spinal cord (at autopsy) confirms the diagnosis. Sometimes it is possible
to isolate a virus
from cerebrospinal fluid. A characteristic is a 4-fold or more increase in the level of antibodies
in the reaction of neutralization or binding of complement between the acute stage and the stage
convalescence. In non-paralytic cases, it should be distinguished from meningitis.
other etiology. In acute pyogenic forms, the glucose content in the cerebrospinal fluid
reduced and cells are represented exclusively by neutrophils. Meningitis with mumps
(lymphocytosis is also found in the cerebral fluid) usually does not cause difficulties in
diagnosis if there is an increase in the parotid glands. Harder to differentiate
tuberculous meningitis, however, its onset is usually more gradual. The diagnosis is still
based on changes in the cerebrospinal fluid: in both diseases, it can
appear cytosis, neutrophilic and lymphocytic and increased protein content. At
polio glucose concentration is normal; with tuberculous meningitis, it is invariably
reduced.
The spinal form of the paralytic stage is usually easy to diagnose. If the pain and
muscle soreness is significantly pronounced, it must be distinguished from acute rheumatism and
acute osteomyelitis. In recent diseases, however, soreness is usually more localized
Vana, felt in or near the joint; tendon reflexes are not lost, as in poliomyelitis.

The strange and yet unexplained polio-like syndrome that develops after
asthmatic attack, described in children. This "post-asthmatic pseudopoliomyelitis"

the syndrome is diagnosed on the basis of its combination with asthma and the absence of an increase in antibody titer
to poliovirus.
In adults, poliomyelitis should be differentiated from transverse myelitis and the syndrome Guinea Barre. In the latter case, a sharp increase in
protein content.
Treatment. General recommendations. If poliomyelitis is suspected, even with minor manifestations, it is necessary to ensure complete rest, since physical activity in
the preparative stage increases the risk of developing severe paralysis.
From the point of view of approaches to the treatment of paralytic poliomyelitis, three categories of patients: 1) patients without respiratory disorders and bulbar disorders; 2) patients with respiratory disorders in combination with bulbar or without them; 3) patients with bulbar paralysis.
Treatment of patients without respiratory and bulbar disorders. To relieve pain and anxiety use acetylsalicylic acid or other analgesics and sedatives (diazepam). WITH
for the same purpose, hot compresses are applied to the area of painful muscles. Light, passive movement is the only form of physical activity allowed at this stage of the disease. Introduction antibiotics are not indicated, except for the prevention of pneumonia in patients with respiratory paralysis.
The introduction of immunoglobulin is also impractical.
The course of the disease after the development of paralysis can be divided into several stages: 1) acute
the stage when pain and soreness in the muscles persists; usually lasts 2-3 weeks; 2) stage convalescence, during which muscle strength is restored; can go on
from 6 months to 2 years; 3) a chronic stage, which is said if, after the maximum possible recovery remains permanent paralysis.
The principal task in the acute stage is to prevent the tension of the paralyzed
muscles and the development of contractures in the corresponding antagonist muscles. The patient should be
on a firm bed, the limbs are placed in a position in which the paralyzed muscles
relaxed (but not taut), sandbags and pillows are used for this purpose.
During the recovery period, prolonged bed rest is necessary only in severe cases,
appropriate tires can be used to prevent contractures.
In addition to cases of severe paralysis of the muscles of the trunk, patients are allowed, if possible, get up several times a day, with paralysis of the vertebral muscles to prevent the development deformations, you can use supporting corsets. Active
exercises. They can be performed with the support of unauthorized persons, on simulators and in the water. V
of the affected muscle groups the full range of possible movements should be carried out at least 1-2 times a day. Physiotherapy is also important. In the later stages of development contractures and deformities produce tenotomy and other operations.
Treatment of respiratory disorders. Patients with respiratory paralysis need
carrying out mechanical ventilation, the duration of which varies depending on the severity of the condition. Decrease
breathing volume by 30-50%, the appearance of irregular breathing or obvious difficulty in breathing are an indication for transferring the patient to mechanical ventilation.
Infectious complications require appropriate antibiotic therapy; at
the development of atelectasis, it may be necessary to conduct bronchoscopy with suction content; acute enlargement of the stomach is now a rare complication.
Treatment of bulbar paralysis. In the absence of respiratory disorders in isolated bulbar paralysis there is a risk of secretions or vomit flowing into the lungs. Dysphagia leads to feeding difficulties. The patient should be given a position intermediate between lying on his stomach and lying on his side, and after a few hours turn it over to the other side; the foot end of the bed is raised 15 ° above the horizontal level. When
the need for procedures, the toilet and when an unauthorized person is in

in the immediate vicinity, the patient can be in a natural, relaxed
position. Tracheostomy in such patients, as a rule, is not needed, except in cases of bilateral paralysis of the vocal cords. The secret that accumulates in the pharynx is removed by mechanical suction. At
In significant difficulties, feeding is carried out through a nasogastric tube.
Forecast. Mortality in different epidemic outbreaks varies from 5% to 25%.
Immediate cause of death is respiratory paralysis due to direct injury

the respiratory center with a bulbar form or involvement of the intercostal muscles in the process and diaphragm with ascending paralysis; mortality decreased significantly with the introduction of mechanical ventilation.
If the progression of paralysis is stopped, it is safe to speak about significant
in the future recovery. Favorable signs are the presence of voluntary
movements, reflexes, and muscle contraction in response to nerve stimulation 3 weeks after development of paralysis. Once started, the improvement process can continue for a year or even longer.
Late respiratory failure is a rare consequence of poliomyelitis when severe
kyphoscoliosis, it is sometimes preceded by a long period of alveolar hypoventilation. Repeated polio attacks are very rare and can be caused by a different strain of the virus.
Prevention. For the prevention of poliomyelitis, a live vaccine containing
an attenuated virus strain (Sebin oral vaccine). Effective immunity against everyone
the three types of virus persist throughout life. The risk of contracting polio due to
vaccination with live vaccine is extremely small (1: 3,000,000). Live vaccine of any type, including including against poliomyelitis, should not be used by persons who are suspected of immunodeficiency, since
how the risk of contracting polio due to vaccination is increased in them.
Poliomyelitis-like diseases
Against the background of the practical eradication of poliomyelitis, the proportion of acute infectious diseases has increased.
diseases that are clinically similar to various forms of poliomyelitis. As
agents that can cause poliomyelitis-like syndromes, Coxsackie viruses can act
(types A and B), ECHO, enteroviruses (types 70, 71, 72), mumps virus.
The clinical picture of infection caused by ECHO viruses sometimes resembles
non-paralytic form of poliomyelitis. Children are more often affected than adults. The main symptoms include fever, coryza, pharyngitis, vomiting, and diarrhea. Often available
rubella-like rash. Involvement of the nervous system is characterized by the appearance of a head pain, stiff neck, the development of drowsiness, muscle weakness. Disease
proceeds benignly, recovery occurs in 1-2 weeks.
Enterovirus 70 causes epidemic outbreaks of acute hemorrhagic conjunctivitis.
Neurological complications occur in 1 in 10,000-15,000 cases of conjunctivitis,
mainly in adults. The clinical picture is poliomyelitis-like syndrome with
flaccid, asymmetric proximal paralysis in the legs accompanied by severe
radicular pain. In half of the cases, the paralysis does not regress. In addition, there are isolated lesion of the cranial nerves (more often the facial), pyramidal signs, dizziness, sensory disorders, dysfunction of the bladder. Since the neurological symptoms
tomato develops almost 2 weeks after the onset of acute hemorrhagic conjunctivitis,
it is practically impossible to isolate the virus at this moment. The diagnosis is made retrospectively at
the basis of serological reactions. Acute hemorrhagic conjunctivitis in the overwhelming
in most cases, it precedes the development of lesions of the nervous system.
Enterovirus 71 affects children and adolescents. Neurological disorders
occur in 25% of cases and include aseptic meningitis, cerebellar ataxia, and flaccid

paresis and polioencephalitis. The diagnosis is made when the virus is isolated from the pharynx, feces,
herpes-like vesicles or in the reaction of neutralizing antibodies.

NEUROSYPHILIS

Neurosyphilis is the general name for lesions of the nervous system of a syphilitic nature. Though the fact of Tg invasion. pallidum in the nervous system is beyond doubt, it is still difficult to determine
how much damage is due to the direct impact of the microorganism, on the one hand,
and immune and other mechanisms - on the other.

Until the 40s, almost the majority of cases were associated with this sexually transmitted disease. referrals to neurologists and psychiatrists. The situation changed dramatically with the introduction of
medical practice of penicillin after World War II. Treatment of the disease began at
early stages before the development of damage to the nervous system. Another factor also mattered: development of laboratory methods for early diagnosis of the disease.

The widespread use of penicillin has resulted in the prevalence of atypical, difficult

diagnosed forms. There is also a change in the frequency of forms of neurosyphilis. So, according to Wolters, among 518 patients observed in the pre-antibiotic period (1930-1940), the ratio
forms of neurosyphilis were as follows: tabes dorsal 45%, progressive paralysis 8%, taboparalysis 9%, vascular syphilis 9%, meningitis 19%, syphilitic myelitis 10%. The same author reports that in the group of patients (121 people) who underwent treatment in 1970-1984, the palette of neurosyphilis
turned out to be different: tabes dorsal 15%, progressive paralysis 12%, taboparalysis 23%, vascular syphilis 19%, meningitis 23%, myelitis 8%.

The problem of the relationship between syphilis and
AIDS. The possibility of simultaneous infection indicates the exclusive role of sexual
contact in the spread of these diseases. Another issue that is now being intensively studied is
is an increase in the incidence of syphilis and a change in its clinical picture in conditions of immunodeficiency
deficiency caused by HIV.

The dramatic decrease in the incidence of neurosyphilis in the antibiotic era allowed predetermine that in the future he will disappear altogether. However, after stabilization at a sufficiently low
level in recent years, the number of cases began to increase again.

Pathomorphology. In early neurosyphilis, there is an infiltration of the meninges
lymphoid and other mononuclear cells. The defeat of the cranial nerves is of the type
axonal degeneration against the background of the inflammatory process. Proliferation of endothelium in small
meningeal vessels can cause their occlusion with subsequent ischemic necrosis
brain tissue. Along with this, demyelination, destruction in the spinal cord,
transverse myelitis.

Paralytic dementia is characterized by the slow development of pathological changes. Inflammation from the meninges passes to the small vessels of the cortex and the cortex itself, where it is observed
significant lymphoid and plasma cell infiltration. This process is accompanied by the loss
cortical neurons and glial proliferation. Unlike other forms of neurosyphilis in the cortex with paralytic dementia spirochetes are found.

With tabes dorsalis, following inflammatory changes in the meninges and
meningeal vessels degeneration of the posterior roots, posterior cords of the spinal cord occurs, sometimes - cranial nerves.

Clinical forms of neurosyphilis. Asymptomatic neurosyphilis. With asymptomatic
variant, there are changes in serum and cerebrospinal fluid, indicating the presence of

syphilis. The incidence of asymptomatic forms before antibiotic use and at present approximately the same and accounts for 30% of all cases of syphilis. Diagnosis of asymptomatic neurosyphlis
established only on the basis of changes in cerebrospinal fluid, and accurate statistical data on this form can only be obtained by examining the fluid in all patients with syphilis.
The pathological changes that characterize asymptomatic neurosyphilis have not been found.
The prevalence of asymptomatic forms in the general structure of the incidence of syphilis depends, in particular
on the duration of the process. At the same time, the increase in the number of cases in the first 2 years of the disease is replaced by their
decrease occurring for two reasons: recovery with the normalization of cerebrospinal fluids or transformation into a manifest form of neurosyphilis.
Syphilitic meningitis. With damage to the meninges, a clinic of acute or subacute meningitis, often involving the cranial nerves. In some cases, there is focal symptoms due to syphilitic arteritis. The period from the moment of infection to the onset of meningeal symptoms is several months or years, on average 1 year. AT 10% cases of meningitis is combined with cutaneous manifestations of secondary syphilis.
In the pathogenesis of the disease, three mechanisms are important: 1) increased ICP and hydrocephalus in
as a result of a violation of the outflow of cerebrospinal fluid; 2) direct damage cranial nerves; 3) the formation of heart attacks in the brain substance caused by thrombosis of small vessels. Meningeal symptoms usually go away without treatment, symptoms of the same lesion the cranial nerves persist for a long time. The need for therapy during this period is due to the desire to prevent the development of progressive paralysis or dorsal tabes.
Meningovascular syphilis. Syphilitic endarteritis can cause heart attacks in the brain and spinal cord. There is a gradual increase over several days focal symptoms. It may be preceded by a period (weeks, months) characterized by personality changes, the presence of prolonged headache. Men are affected more often than women shins. Symptoms usually appear 5-30 years after infection, but this
the period may be shorter if vascular neurosyphilis is combined with acquired syndrome immunodeficiency.

The defeat of the spinal cord in meningovascular syphilis is in the nature of myelopathy. The course is usually progressive. Development of transverse myelitis is possible. Study cerebrospinal fluid reveals changes specific to syphilis.
Specific anti-syphilitic treatment causes symptoms to reverse meningomyelitis. The changes caused by heart attacks in the spinal cord persist and lead to disability.
Gumma. It is a syphilitic avascular granuloma that forms a well-demarcated from the surrounding brain tissue, the focus of damage. Associated with the dura mater a granuloma is essentially a localized form of meningitis. The clinical picture repeats brain tumor. The widespread use of CT has shown that gums are much more common than possible. suggest from clinical data.
Dorsal tabes (tabes dorsalis). The clinical picture consists of piercing pains, progressive ataxia, reduction or complete loss of deep types of sensitivity, loss tendon reflexes, dysfunction of the pelvic organs.
Inflammatory changes in the pia mater are localized mainly in the posterior the surface of the spinal cord and extends to the intraspinal portion of the posterior roots. The degeneration process involves the posterior roots, mainly in the lumbar and sacral affairs, and the posterior cords of the spinal cord (thin bundle - Gaul's bundle). Involvement of the posterior roots in
the cervical spine is rarely observed. Possible damage to visual, auditory and other cranial

nerves.
The cardinal signs of tabes dorsalis are loss of knee and Achilles reflexes,
disorder of vibration and muscle-articular sensitivity in the legs with positive
Romberg's symptom. 94% of patients have anisocoria, changes in the shape of the pupils, lethargy
pupillary reactions to light. In 48% of cases, Argyll-Robertson symptom with loss of reactions is
detected
pupils to light and preservation for accommodation and convergence.
Violations of superficial sensitivity are also possible, trophic changes in the form
Charcot joint. Currently, as a rule, only the abortive form of tabes is found.
Progressive paralysis (generalized paralysis of the mentally ill, syphilitic meningoencephalitis). The disease is chronic. The pia mater is cloudy,
thickened, adhered to the surface of the brain. The convolutions are atrophic, the grooves are
widened and
filled with cerebrospinal fluid. The ventricles are dilated, their walls are covered with granulations
(granular ependymatitis).
The manifestations of progressive paralysis are manifold. The clinic is based on dementia with
growing violation of criticism and emotional lability. In the finale - growing weakness
arms and legs (hence the "progressive paralysis of the mentally ill" - paralysis progressiva
allienonun),
epileptic seizures are possible. Untreated progressive paralysis leads to the death of patients in
for 3-5 years. The effectiveness of penicillin depends on the nature and volume of available to the
beginning
treatment of changes in the nervous tissue.
Congenital syphilis. Spirochetes enter the fetus during the period from IV to VII months
pregnancy. The more time has passed from the moment of infection of the mother to pregnancy, the
more
less chance of intrauterine infection. Currently, due to early detection
and the treatment of syphilis in adults, congenital forms of damage to the nervous system are very
rarely.
Congenital forms of syphilis are similar to the clinical forms described in adults for
with the exception of tabes dorsal, which is not observed in children.
Additional signs of congenital neurosyphilis are hydrocephalus and triad
Hutchinson's (interstitial keratitis, tooth deformity, and deafness), but a complete triad
is rare. Penicillin therapy is carried out in the same way as in adults. Treatment leads to
stopping the infectious process, and the pathological changes and
neurological symptoms persist.
Serological diagnostics. Diagnosis of active neurosyphilis, manifest or
asymptomatic, based on the results of the study of cerebrospinal fluid. With active
an infectious process, lymphocytic pleocytosis (200-300 cells in 1
mm) with a small number of plasma cells, an increase in the content of protein and β-globulins.
A reflection of the body's immune response is the appearance in the cerebrospinal fluid
oligoclonal antibodies and increased IgG content. The fading Lange reaction with
colloidal gold reflects, as it turned out, hypergammaglobulinrachia.
Serological tests for syphilis are divided into two groups. The first is the reaction
Wasserman and a number of others, demonstrating the presence of antibodies to lipid antigen. To the
second group

are highly specific reactions outcome. Encephalitis is subacute or chronic
flow. With CMVI, the development of polyneuropathies is possible.
Diagnostics. The diagnosis is based on the isolation of cytomegalovirus from the cerebrospinal
fluid, urine, blood, liver tissue biopsies or autopsy material, as well as to identify
antibodies against cytomegalovirus of the IgM class at birth (in umbilical blood). Pre-
a positive diagnosis is possible when typical cytomegalic cells are found in stained

preparations of urinary sediment or saliva. Congenital CMVI should be suspected in children with microcephaly, in which, on craniography and CT of the skull, periventricular calcifications, however, the last symptom is not specific and can be observed with toxoplasmosis. Prenatal diagnosis is possible when cytomegalovirus is isolated from amniotic fluid.

Treatment. Acyclovir is ineffective in treating congenital infections in newborns. V in a number of cases, its therapeutic effect was noted in CMVI in adults. If defeated fetus, the issue of termination of pregnancy should be discussed.

HEREDITARY DISEASES OF THE NERVOUS SYSTEM

NERVO-MUSCULAR DISEASES.

Degenerative diseases with a predominant lesion of the neuromuscular apparatus constitute the most significant group among all hereditary diseases.

Extremely important, and often decisive in the diagnosis of neuromuscular diseases are the results of electrophysiological and biochemical studies. The same the importance of pathomorphological findings is great. Examination of muscle biopsy in light microscope helps to differentiate myogenic from neurogenic atrophy. Histochemical the study is necessary to identify metabolic muscle lesions, and electronic microscopy discovered a whole large class of diseases - non-progressive myopathies.

Progressive muscular dystrophies. The term muscular dystrophy is called a group of genetically determined disorders characterized by progressive degenerative changes in muscle fibers without primary pathology of peripheral (lower) motor neuron.

The various forms differ from each other in the types of inheritance, the timing of the start of the process,
the nature and speed of its course, the originality of the topography of muscle atrophy, the presence or
the absence of pseudohypertrophies and tendon retractions and other signs.

Most muscular dystrophies are well studied clinically, their detailed description made at the end of the last century. But, despite the almost century history of studying myodystrophies,
questions of their pathogenesis and treatment remain unresolved until now. Great expectations are entrusted to molecular genetics, with the help of which the location of genes is already many nosological forms.

Diagnosis of muscular dystrophies is often very difficult. There is high variability of clinical manifestations, and the small number of family members makes it difficult
determining the type of inheritance.

A characteristic motor defect in patients with muscular dystrophies is "duck" gait: the patient walks waddling to side to side. It is mainly associated with weakness. gluteal muscles, especially the middle and small, which fix the pelvis relative to the femoral bones. As a result, with the disease, the pelvis tilts towards the unsupported leg (the phenomenon Trendelenburg) and compensatory inclination of the trunk in the opposite direction (phenomenon Duchenne). When walking, the side of the slope is constantly changing. The specified changes can be checked
and in the Trendelenburg test, asking the patient to raise one leg, bending it at a right angle in knee and hip joints: the pelvis on the side of the raised leg drops (and does not rise as in normal) due to weakness of the gluteus medius muscle of the supporting leg.

Rising from a horizontal position, a patient with severe muscle weakness of the proximal muscles with difficulty turns over on the stomach, then, resting his hands on the floor, becomes

on all fours and after that, resting his hands on the shins, then on the hips, gradually straightens. This phenomenon of “typing by oneself” is called the Govers technique. It is often associated with weakness.
gluteus maximus muscles.
Duchenne myodystrophy. Duchenne pseudohypertrophic muscular dystrophy
occurs more often than all other diseases of the muscular system (30 per 100,000 live births). It is characterized by early onset and malignant course. The classic picture emerges
a change in gait in a child aged 2-5 years, by the age of 8-10, children can already walk with difficulty, by the age of 14-15
they are usually completely immobilized. In younger children, initial symptoms
are manifested by a lag in motor development: they begin to walk later, do not know how to run and
jump. Patients die in the 2-3rd decade of life.
One of the first signs of the disease is the compaction of the calf muscles and
a gradual increase in volume due to pseudohypertrophies. Atrophy of the muscles of the thigh, pelvic girdle
often masked by well-developed subcutaneous adipose tissue. Gradually, the process took the ascending direction and spreads beyond the shoulder girdle, back muscles, and then to proximal arms.
In the terminal stage, muscle weakness can spread to the muscles of the face, pharynx,

respiratory muscles.
In the advanced stage of the disease, there are characteristic symptoms such as "duck gait"; pronounced lumbar lordosis, pterygoid scapula, symptom of "free shoulder girdle". Typical early muscle contractures and tendon retractions, especially of the Achilles tendons. Early knee reflexes fall out, and then reflexes from the upper limbs.
Pseudohypertrophy can develop not only in the gastrocnemius, but also in the gluteal, deltoid muscles, abdominal muscles, tongue. The heart muscle is very often affected by the type cardiomyopathy. Disorders of the rhythm of cardiac activity, expansion of the boundaries of the heart,
deafness of tones, ECG changes. Acute heart failure is the most common cause
deaths in Duchenne muscular dystrophy. An autopsy reveals fibrosis and fatty
infiltration of the heart muscle.
Often there is a violation of the motility of the gastrointestinal tract.
Decreased intelligence is a common symptom. Of interest is the fact that in some
in families, oligophrenia is sharply expressed, in others it is comparatively moderate. Change of higher
mental function usually does not progress and does not correlate with the severity of the muscle defect.
It cannot be explained only by the pedagogical neglect of sick children who early
are turned off from children's groups, do not attend kindergarten and school due to motor defects. CT and MRI often reveal cerebral atrophy, possibly associated with
violation of prenatal brain development.
Often, children develop adiposogenital syndrome, sometimes other signs
endocrine insufficiency. Changes in the skeletal system are often found: deformity of the feet, chest, spine, diffuse osteoporosis.
A distinctive feature of the Duchenne form is a high degree of hyperenzymemia
already in the early stages of the development of the process. So, the level specific to muscle tissue
enzyme - creatinine phosphokinase - in blood serum can exceed tens and even hundreds of times

normal performance. A sharp (10-100 times) increase in creatinine phosphokinase (CPK) with nervous
muscular pathology should prompt the discussion of the following diseases first of all:
illness
Duchenne,
illness
Becker,
poliomyositis
and
dermatomyositis,
paroxysmal uymioglobulinuria, distal myodystrophy. Only in advanced stages
disease, the degree of hyperenzymemia gradually decreases. There are reports of an increase in CPK by
stages of intrauterine development.
Duchenne myodystrophy is transmitted by a recessive type linked to the X chromosome. Gene localized in the short arm of the X chromosome. The frequency of gene mutations is quite high (30%), which
explains a large number of sporadic cases.
A mutation (most often a deletion) leads to a sexual or almost complete absence of the product gene - a structural protein of dystrophy. The physiological role of dystrophy is not fully established.
It is found in high concentrations in the area of the sarcolemma, playing, apparently, a certain role in maintaining the integrity of this membrane. The absence of dystrophy causes structural changes in the sarcolemma, which in turn leads to the loss of intracellular components and increased calcium intake, which ultimately leads to the death of myofibrils. It is believed that deficiency of dystrophy in the synaptic zones of cortical neurons is the reason for the delay mental development.
For medical genetic counseling, it is very important to establish a heterozygous
carrier. With Duchenne muscular dystrophy in heterozygotes, approximately in 70% of cases, subclinical, and sometimes clear signs of muscle pathology - some compaction and even
an increase in the calf muscles, rapid muscle fatigue during exercise, changes in
EMG and pathomorphological examination of muscle biopsies. Most often in
heterozygous carriers show an increase in creatinine phosphokinase activity.
In the presence of a clinical picture of Duchenne muscular dystrophy in females, one should first of all, to exclude the possibility of an anomaly on the X chromosome - Shereshevsky-Turner syndrome
(XO), Morris syndrome (XY) or mosaicism for these syndromes.
Duchenne muscular dystrophy, which begins to develop in the prenatal period,
is essentially a congenital myopathy and can be diagnosed shortly after birth by
conducting muscle biopsy and determining the activity of CPK.

Becker's myodystrophy. Along with the severe, malignant X-linked
Duchenne muscular dystrophy, there is a benign form - Becker's disease. By clinical symptoms, it is very similar to the Duchenne form, however, it usually begins later - at 10-15 years, flows gently, patients maintain their working capacity for a long time, at the age of 20-30 years and later still
can walk. Fertility is not reduced, so the disease is sometimes traced in several
generations of the family: a sick man passes the disease on to his grandson through his daughter (“the grandfather effect”).
The initial symptoms, as in Duchenne disease, are manifested by weakness in the muscles of the pelvic girdle,

then in the proximal parts of the lower extremities. Patients change their gait, they have difficulty climbing stairs, getting up from a low seat. Are characteristic pseudohypertrophy of the gastrocnemius muscles. Retraction of the calcaneal (Achilles) tendons is less pronounced sharply than with Duchenne disease.

In this form, there are no intellectual impairments, cardiomyopathy is absent or expressed slightly.

As with other X-linked myodystrophies, with the Becker form, the CPK activity, although to a lesser extent than in Duchenne disease, not exceeding 5000 units. Gene Becker's disease, like Duchenne's disease, is localized in the short arm of the X chromosome; probably both locus are closely related or allelic. Unlike Duchenne disease, with which dystrophy is practically absent, with Becker's disease, an abnormal dystrophy. Differences are also found on muscle biopsy. With Becker's muscular dystrophy muscular fiber usually non-circular, hyaline fibers, characteristic for Duchenne muscular dystrophies are extremely rare.

Myodystrophy Landouzy-Dejerine (face-humeral myodystrophy). Disease transmitted in an autosomal dominant manner with high penetrance, but somewhat variable expressiveness. It is much less common than Duchenne muscular dystrophy (0.4 per 100 thousand population).

It is believed that the gene for this disease is located on the 4th chromosome. Women get sick more often men (3: 1), Physical overload, intense sports, as well as irrational conducted physical therapy can contribute to a more severe course of the disease.

Myodystrophy Landouzy-Dejerine is a relatively favorably current form of muscle pathology. It begins at the age of about 20, sometimes later. However, in family cases diseases, when it is possible to trace the younger family members in dynamics, it is possible to identify some muscle weakness, such as the muscles of the face, and at an earlier age.

Muscle weakness and atrophy first appear in the muscles of the face or shoulder girdle. Gradually, these disorders spread to the muscles of the proximal arms, and then to lower limbs. In most cases, the muscles of the anterior surface are affected first. legs (with the development of a drooping foot), then the muscles of the proximal legs. On high diseases are grossly affected by the circular muscles of the eye and mouth, pectoralis major, anterior dentate and lower trapezius muscle, latissimus dorsi, biceps, triceps shoulder. The appearance of patients is characteristic: a typical face of a myopath with a “transverse smile” (“smile La Gioconda "), protrusion of the upper lip (" tapir lips "), pronounced pterygoid scapula, a kind of deformation of the chest with its flattening in the anteroposterior direction and rotation inside the shoulder joints. Often there is an asymmetry of the lesion, even within the same muscle (for example, the circular muscle of the mouth). There may be pseudohypertrophy of the gastrocnemius,

deltoid muscles, sometimes facial muscles. Contractures and retractions are moderately expressed.
Tendon reflexes are preserved for a long time, but sometimes they decrease already at an early stages.
Signs of damage to the heart muscle are rarely detected. Serum activity
enzymes are increased slightly and may be normal. Intellect does not suffer.
Life expectancy in most cases does not decrease. Of interest is the fact that
EMG for myodystrophy Landouzy-Dejerine is often not quite typical of the muscle level.
defeat. In some patients (members of the same family), there may be a decrease in amplitude biopotentials, interference type of the curve, in others, on the contrary, a decrease in frequency and
hypersynchronous activity, sometimes with a typical stockade rhythm. It should be remembered about the spinal
a variant mimicking Landouzy-Dejerine disease.

Myodystrophy Erba-Rota (limb-girdle myodystrophy). Transmitted by autosomal recessive type, both sexes suffer the same. The onset of the disease in most cases refers to the middle of the 2nd decade of life (14-16 years), but is described as early, pseudo-Duchenne form, when the first symptoms appear before the age of 10 years and the disease
is difficult, and the late version with the onset after 30 years.
The course of the disease can be fast or slower, on average complete
disability occurs 15-20 years after the onset of the first symptoms. Myodystrophy
begins either with damage to the muscles of the pelvic girdle and proximal legs (Leiden form - Moebius), or from the shoulder girdle (Erb form). In some cases, the shoulder and pelvic girdles are affected at the same time. The muscles of the back and abdomen are affected quite significantly. In sick
there is a characteristic “duck” gait, it is difficult to get up from a lying and sitting position, lumbar lordosis. The muscles of the face in most cases are not affected. For this form, thorns of contracture and pseudohypertrophy. Terminal atrophies and tendon lesions may occur. retraction. Intelligence is usually preserved. The heart muscle is mostly unaffected. Level serum enzymes, as a rule, increased, but not as sharply as with X-linked
myodystrophy. There are indications that in male patients the level of CPK is higher than in patients
women. There is a significant difference in the expressivity of the mutant gene among different family members -
along with a severe clinical picture, there may be relatively mild and even blurred clinical symptoms. Death usually occurs from pulmonary complications.
Since the clinic of limb-girdle myodystrophy is especially willing to imitate nervous
muscle diseases of a different nature, it is necessary, especially in sporadic cases and with late onset of the disease, conduct a thorough clinical examination to exclude
spinal amyotrophy, polymyositis, metabolic, endocrine, toxic, medicinal,
carcinomatous myopathies. In the past, there has been a clear overdiagnosis of this form of muscle
dystrophy.

Charcot-Marie-Tooth neural amyotrophy

It occurs with a frequency of 1 per 50,000 population. Autosomal dominant inheritance, recessive type linked to the X chromosome.
Pathomorphology. Demyelinating type changes are found in peripheral
nerves. There may be changes in the posterior and lateral columns of the spinal cord, atrophy and degeneration in

anterior and posterior columns of the spinal cord. Atrophy of the muscle bundles is found in the muscles
fibers.
Clinic. The onset of the disease occurs at the age of 15-30 years. The first signs of the disease there may be increased muscle fatigue during exercise, when walking for long
distance. Often, to reduce fatigue, patients mark time. Then appears
atrophy of the peroneal muscle group - the muscles of the legs and feet. There is a sagging of the foot,
gait is disturbed. With significant atrophy, a dangling foot develops. Legs and feet
takes on a deformed appearance. The legs are like "inverted bottles" or "stork legs". Foot takes the form of Friedreich's foot: high arch, sunken interdigital spaces, fingers Z-figurative type. The gait changes, becomes "stepage" type, in order not to scoop up with the toe the ground, the sick raise their legs high.
Atrophies of the muscles of the hands join later, 5-10 years after the development of muscle atrophy
legs. The hand often takes the form of a "clawed paw" or "monkey paw".
Achilles reflexes are lost in the early stages of the disease, while knee
reflexes from biceps and triceps remain intact for a long time.
Sensory disorders appear in the later stages of the disease. There may be pains paresthesia, hypesthesia in the distal extremities of the "gloves" and "socks" type. Revealed pain on palpation of the neurovascular trunks.
There are vegetative-trophic disorders in the form of hyperhidrosis of the hands and feet, hyperemia of the hands and feet.
The course of the disease is slowly progressive. Forecast in most cases
favorable.
Treatment. Therapy is aimed at improving muscle trophism and impulse conduction along nervous tables. Prescribe ATP, cocarboxylase, riboxin, cerebrolysin, methionine. Are effective

vitamins E, A, groups B and C. To improve microcirculation, nicotinic acid is prescribed, pentoxifylline. Proserin is used to improve neuromuscular conduction.
Among non-drug remedies, exercise therapy, massage, physiotherapy are used.

Myotonia

Myotonia is a disease characterized by a violation of muscle tone in the form
slowing down muscle relaxation after active contraction.
Described by Leiden in 1874. In 1876, Thomsen drew attention to the example of his family on the hereditary nature of this disease.
The frequency of occurrence is 0.3-0.7 per 100,000 population. Inheritance type - autosomal dominant.
The first signs of the disease appear at the age of 8-15 years. Myotonic spasms
localized in various muscle groups, more often in the muscles of the hand, legs, chewing muscles, circular
muscles of the eye. Strong clenching of the hand into a fist, or clenching of the jaws, or tightly closing the eyes,
or prolonged standing causes tonic spasms. The muscle relaxation phase is delayed by
long time. Patients cannot quickly unclench their hand, jaw or open their eyes.
Repetitive movements reduce myotonic spasms.
There are several typical techniques:

- Tapping the thenar muscles with a hammer induces adduction of the thumb;
- When the tongue is struck, a dimple appears in the tongue;

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When you hit a large muscle (biceps), a roller appears.
The appearance of patients resembles athletes. Muscles are dense, firm, their strength at the same time
reduced. Tendon reflexes are normal, in some cases reduced.
The course of the disease is slowly progressive.
Treatment. Diphenin is prescribed 0.1-0.2 3 times a day for 2-3 weeks. Diakarb at 0.125 2 once a day for 2-3 weeks. It is assumed that diphenin has an inhibitory effect on polysynaptic conduction in the central nervous system, and diacarb changes membrane permeability.
Treatment of muscular dystrophies. Therapeutic options for muscular dystrophies very limited. There is practically no etiological and pathogenetic treatment.
Symptomatic treatment is aimed primarily at preventing the development of contractures, maintaining the existing muscle strength and possibly a slight decrease in the rate of development
atrophy. The main task is to maximize the period during which
the patient is able to move independently, as in the supine position they rapidly increase contractures, scoliosis, respiratory disorders. The medical complex should include remedial gymnastics, massage, orthopedic measures, drug therapy.
Therapeutic gymnastics consists of passive and active movements performed in all joints in various positions: standing, sitting, lying, with different positions of the limbs.
It is preferable to perform active movements in isometric mode. Gymnastics is not should be carried out regularly, several times a day. At the same time, caution should be exercised against
excessive exercise, especially when accompanied by muscle overstretching. The importance of (especially after immobilization of the patient) have breathing exercises.
Orthopedic measures of a conservative (special splints) and operational nature (achillotomy, transection of the gastrocnemius muscle) aimed at correcting contractures and forming pathological attitudes of the limbs, also aim to preserve the possibility independent movement. In this case, in each case, it is necessary to individually weigh the alleged benefits and possible harm from surgery. It should be borne in mind that often (in particular, with severe hyperlordosis and weakness of the quadriceps femoris muscle) equinovarus installation of the feet has a compensatory value and after carrying out, for example, achillotomy the patient may be completely immobilized. With developing
contractures, it is recommended to carefully stretch the muscles up to 20-30 times a day with the subsequent imposition of a splint during sleep.
Drug therapy involves the appointment of metabolic drugs,
aimed at replenishing energy and protein deficiencies, but their effectiveness
highly questionable. Calcium antagonists are used (in connection with the revealed in Duchenne disease

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a defect in cell membranes, leading to an increased flow of calcium into the cell), immunomodulators, phosphorus-containing compounds (ATP, phosphaden), vitamin E (100mg orally 3
times a day). It has been shown that in Duchenne disease, the use of prednisolone (0.75 mg / kg per day)
can dramatically increase muscle strength, but this effect lasts for less than a year and in general does not affect the outcome of the disease. Due to the serious side effects that occur during long-term use of the drug, its use is impractical. Effect estimates
anabolic steroids are controversial and their appointment is often associated with unjustified

risk. When evaluating the effect of certain drugs in Duchenne disease, it should be borne in mind that when
moderate severity of the disease in patients aged 3-6 years, there may be a relative
stabilization of the state associated with age-related development of the muscular system, the acquisition
motor skills, which can temporarily compensate for the continuously flowing
dystrophic process.
Correction of the patient's nutrition is of certain importance, a diet with a high
protein and low fat and reduced calories with optimal
the content of vitamins and minerals. Psychological support of the patient plays an important role,
continuation of training, correct vocational guidance.

NZNS with EPS lesion

Huntington's disease

Huntington's disease occurs with a frequency of 4-6 per 100,000 population and is inherited by autosomal dominant type. One of the features of this disease is the phenomenon
anticipation, i.e. an increase in the severity of the disease and its appearance at a young age in subsequent
generations.
The pathological and anatomical picture of the disease is characterized by diffuse changes in the cerebral cortex, which predominate in the anterior central and occipital gyri. At
this *item lenticularis is* sharply wrinkled, cells in the *putamen* disappear . In the pallidum, a secondary is found
rebirth.
The study of *neurotransmitter systems of the* brain in the basal ganglia revealed
a decrease in the content of acetylcholine, serotonin, GABA and nitropeptides (methenkephalin, B-
endorphin, substance P, angiotensin).
The Huntington's disease gene is thought to act by damaging
glutamatergic corticosteroid pathway. In turn, this leads to overstimulation of neurons.
striatum and their damage by excess of peroxide radicals.
Among the hereditary causes of choreic hyperkinesis, the main significance is
imparts a functional predominance of dopaminergic activity in the subcortical nuclei and
a decrease in the inhibitory effect of GABA on dopaminergic neurons of the substantia nigra.
Due to severe damage to the dopamine receptors of the striatum in the late stage of the disease (as in the juvenile akinetic-rigid variant) insufficiency develops
dopaminergic processes in the basal ganglia. Moreover, in the clinical picture of the disease Parkinson's symptoms begin to prevail.
Clinically **,** hyperkinetic, akinetic-rigid and mental forms **are distinguished.**
Huntington's disease.
The classic hyperkinetic form begins after 30-40 years. Onset of the disease
gradual, its main manifestations are reduced to choreic involuntary movements
muscles and a gradually increasing decrease in intelligence. Choreic movements abruptly aggravated with excitement, they are not accompanied by weakness, go against the background of a low or
normal muscle tone and disappear only in sleep. When walking, patients scatter their hands, dance, swing the body, but keep balance. Characteristic is
grimacing, unnecessary sounds during conversation, inability to keep the tongue out at rest. With over time, hyperkinesis builds up, patients lose the ability to move around and take care of themselves.
In a small percentage of cases, the clinical picture of Huntington's disease is dominated by mental disorders. The mental disorder increases gradually (apathy, memory impairment,

decrease in criticism). As the disease progresses, dementia develops. Sometimes observed auditory, visual hallucinations, as well as a state of arousal and delusional ideas.
In 5-10% of all cases of Huntington's disease, an *akinetic-rigid* variant of the disease is observed

with the onset of symptoms in the 1st-2nd decade of life and rapid progression (Westpha-
la). The clinical picture is characterized by muscle rigidity, contractures, impairment
mental development. Choreic hyperkinesis may be absent. Often epileptic
seizures, myoclonus, athetosis, dystonia, ataxia, pyramidal symptoms and oculomotor
violations.
Patients die from associated diseases, sometimes there are cases
suicide.
CT scan of the brain in patients reveals different degrees of external and internal
hydrocephalus. On MRI, enlargements of the lateral ventricles of the brain, atrophy of the head are clearly visible
caudate nucleus. Positron emission tomography reveals a decrease in metabolism
glucose in the subcortical formations of the brain. EEG shows depression and even complete absence of α-
rhythm.
The gene for Huntington's disease was mapped in 1983 on the short arm of the 4th chromosome (4p16.3). V
1993, the gene for the disease IT-15 was identified, which contains in the 5th region an unstable sequence of trinucleotide repeats of cytosine-adenine-guanine (CAG). Normally, the number of these
triplets no more than 25-30, while in mutant chromosomes their number was more than 37. And the more
CAG repeats were observed, the earlier the disease occurred and at the same time it had a faster rate
progression. The malignant juvenile type of the disease (akinetic-rigid) is characterized by
the largest number of copies of CAG repeats. Mutant alleles are genetically unstable and this leads to
an increase in the degree of expansion of CAG repeats in subsequent generations. Instability is higher at
paternal transmission of the disease. All this explains both the effect of anticipation and the effect of paternal
transmission.
Establishing the nature of the mutation in Huntington's chorea made it possible to develop methods of direct
DNA diagnostics of the disease based on determining the degree of expansion of trinucleotide CAG replays.
There is no effective *treatment for* Huntington's chorea. Antidopaminergic drugs are used funds
from
groups
antipsychotics *(haloperidol,*
stelazine,
stage of the operation,
tiapride and
etc.). *Haloperidol is* used at a dose of 1.5-3 mg / day. With akinetic-rigid form, use drugs that increase the activity of the dopaminergic system *(L-dopa, pakom, cinemet, madopar).* Also used are agents of nonspecific metabolic action: *group vitamins B, nootropics, cerebrolysin.*

Currently of paramount importance is the medical and genetic
counseling and identification of carriers of the Huntington's chorea gene (taking into account the ethical issue).

SPINAL AMYOTROPHIES

The many-sided group of hereditary diseases, which are based on the defeat of the anterior horns of the spinal cord. It is generally accepted that spinal amyotrophies are one of the variants of the disease.
motor neuron.
Clinically, the lesion of the anterior horns is manifested by flaccid paralysis and muscle atrophy. In addition, resting fasciculations may occur, mainly in adults, but they are more characteristic of rapidly progressing diseases. At the same time, when slowly
progressive lesions, which occur in most cases of spinal amyotrophy,
fasciculations in the affected muscles are rare; only at arbitrary voltage,
carried out against resistance, irregular contractions of large
muscle bundles. In addition, in these patients, resting fasciculations can be provoked
the use of anticholinesterase drugs.
Different types of spinal amyotrophy differ mainly in the age of onset,
the rate of progression of the disease and the type of inheritance. In most cases, there is symmetrical weakness of the proximal muscles, and only rare variants are characterized by distal muscle damage, asymmetric damage, bulbar muscle involvement.
There are no sensitive disorders. Most forms are inherited in an autosomal recessive manner.
type, however, sometimes, mainly in adults, an autosomal dominant or
X-linked recessive inheritance.

Treatment of spinal amyotrophy is symptomatic. The principles of rehabilitation are the same as treatment of myodystrophies.

Werdnig-Hoffmann disease (acute malignant infantile spinal amyotrophy). Autosomal recessive disease with a frequency of 1: 25,000
newborns. During pregnancy, in 30% of cases, sluggish fetal movement is noted. Initial manifestations of the disease occur from birth and in the first *5* months. The course of the process is malignant,
rapidly progressing. The average age of death of children is 7 months; 95% of children die before 1.5 years of age.
Respiratory infections are the leading cause of death.
Already in the first days after the birth of a child, obvious muscle paresis with
a decrease in muscle tone and a decrease in tendon reflexes. Sometimes complete areflexia. Characterized by bulbar syndrome with fasciculations in the tongue. Mimic muscles are affected at a late stage. The diaphragm is usually not involved. Fasciculation in the skeletal muscles are often absent. As a rule, tachycardia is noted. In 10% of cases, there are
joint deformities or contractures, which may resemble congenital arthrogryposis.
Spinal amyotrophy Vernig-Hoffmaia should be differentiated from others
diseases that cause flaccid child syndrome. The syndrome is manifested by pronounced muscle hypotension, accompanied by excessive mobility in the joints, is characteristic of the "frog" posture with
abducted hips and feet rotated outward. Werdnig-Hoffmann disease is diagnosed
in more than 60% of cases of flaccid child syndrome. Less commonly, it is caused by atonic form of cerebral palsy, congenital myopathies, neonatal or congenital
myasthenia gravis, muscular dystrophies, botulism, Down syndrome, intrauterine poliomyelitis, congenital polyneuropathy, metabolic disorders, Marfan syndrome.

Chronic infantile spinal amyotrophy. Autosomal recessive disease
differs from Werdnig-Hoffmann disease in a later onset (between the 3rd and 24th months) and

relatively slow progression of symptoms. If the disease occurs in the first year life, then the child quickly loses previously acquired motor skills, stops sitting, standing and walk. Often, the first symptoms are detected after an infection or food intoxication. Flaccid paresis first occurs in the legs, mainly in the proximal regions, then in the muscles of the trunk and arms; followed by weakness in the muscles of the neck, bulbar
musculature. Fasciculations in the tongue and skeletal muscles occur in more than 50% of patients.
Flaccid paresis is accompanied by the occurrence of contractures, scoliosis. Common hyperhidrosis. By 4-5 years of age, pneumonia usually develops as a result of respiratory failure and
there is a lethal outcome.
With a late onset of the disease, it flows more benignly, affecting mainly muscles of the trunk and pelvic girdle. Diagnosis of chronic infantile spinal amyotrophy, as well as the diagnosis of Werdnig-Hoffmann disease, it is based (in addition to the early onset disease and characteristic clinical picture) on the results of EMG. Almost always spontaneous resting bioelectrical activity with the presence of fasciculation potentials. At voluntary contractions recorded reduced electrical activity with a “rhythm stockade ”. Serum enzymes are usually normal.
Kugelberg-Welander disease (juvenile form of spinal amyotrophy). In 1955 g. G. Wohlfart et al. described a disease manifested by proximal muscle atrophy and resembling muscular dystrophy, but with widespread fasciculations. In 1956 E. Kugelberg and L. Welander emphasized that the disease is relatively benign; a thorough electromyographic examination established the spinal character of the muscular atrophy.
The disease occurs between 2 and 15 years of age, in most cases before the age of 5 and very slowly progressing. Patients retain the ability to self-care for a long time and even sometimes working capacity. In terms of clinical symptoms, the disease resembles Erba myodystrophy.
Muscle weakness and atrophy develop initially in the proximal lower extremities and the pelvic girdle, then extend to the shoulder girdle. Similarity to myodystrophy is supported by the presence of pseudohypertrophy of the gastrocnemius muscles in 1/4 of patients. Bone deformities

and tendon retractions are usually absent. The process can extend to bulbar Department.
Paraclinical studies in Kugelberg-Welander disease reveal a fairly peculiar changes: if the EMG indicates clear signs of anterior nerve lesion, then the pathomorphological picture with a muscle biopsy reveals a mixed picture - along with neurogenic amyotrophy, there are signs of muscle damage. Enzyme activity, including the number of CPK is often increased, although to a lesser extent than in true myodystrophy.
Congenital myopathies
Congenital myopathies are a group of hereditary, usually non-progressive or slowly progressive diseases, usually detected by mediocre after birth or shortly after. For their designation, the term was previously used “Benign congenital hypotension”. Currently, their names are being determined pathomorphological changes detected by electron microscopy of muscle biopsy. According to the clinical picture, most congenital myopathies differ little from each friend. Signs that the fetus is developing myopathy can be recorded even during prenatal period, in this case, the expectant mother usually notices that fetal movements do not are active enough. After birth, the child usually has a pronounced

generalized hypotension; congenital myopathies are one of the causes of the flaccid child ". Muscle weakness usually extends to the pelvic girdle and proximal regions legs, while the muscles of the shoulder girdle and arms are less affected. In some cases generalized muscle weakness is observed. Muscle underdevelopment is often noted. At some congenital myopathies may be weakness of the facial and / or extraocular muscles, in in the latter case, patients never complain of double vision. Congenital hip dislocation, dolichocephalic head shape, gothic palate, equine foot, kyphoscoliosis. Delayed motor development is characteristic: children begin to sit late, get up, walk, often fall while walking and have little ability to run. Later, they are usually unable to perform the simplest gymnastic exercises, participate in outdoor games. As a result the child cannot fully participate in the life of the children's collective, and since the intellect is almost always preserved, then the motor defect serves as a source of constant emotional voltage.

Reflexes may be normal, decreased, or absent. CPK level is usually normal or slightly elevated. EMG usually reveals short-term low-amplitude polyphasic motor potentials. The biopsy reveals predominant lesion of tonic "red" fibers (type I) or phasic "white" fibers (type II). Conduction speed along motor and sensory fibers is normal.

An extremely important point is the absence of progression or very slow progression of muscle weakness. Since congenital myopathy is often combined with congenital dislocation of the hip, it must be excluded in each case of diagnosis of dislocation.

Central core disease. Inherited in an autosomal dominant manner, although there are and sporadic forms. Congenital dislocation of the hip is common. Muscle weakness is usually not

is progressing. There is an increased risk of developing malignant hyperthermia during operations with general anesthesia. This is of great practical importance, since such patients often undergo surgery for dislocation of the hip or equine foot.

In the study of muscle biopsies, in addition to the predominance of type I muscle fibers, in the fibers themselves reveal abnormal myofibrils (the so-called rods), which are located in the center of the fiber, although they can also be noted on the periphery. Most fibers have

only one rod, although in some of them the number reaches 5. With electron microscopy it was revealed that mitochondria are absent in these rods and, accordingly, with histochemical studies in rods stretching along the entire fiber, no oxidation activity is detected of the mitochondrional enzymes.

Nonmaline myopathy. The disease is inherited in an autosomal recessive or

autosomal dominant type, there are also sporadic cases. In addition to dolichocephalic the shape of the skull, many patients have an elongated face, gothic palate, underdevelopment musculature. Along with the weakness of the muscles of the limbs, weakness of the muscles of the face is often observed.

Muscle weakness progresses in about half of patients. In some patients a severe condition associated with weakness may occur immediately after birth respiratory muscles, tongue, chewing and swallowing muscles, manifested by cyanosis, aspiration, respiratory tract infection, difficulty feeding; deaths are possible.

Muscle biopsy reveals a predominance of type I muscle fibers. Under sarcolemma often reveals clusters of filamentous structures (Greek peta - thread). At electron microscopy shows that filamentous structures originate from Z-disks. Sometimes threads are found in all fibers, and sometimes only in some of them. There is no correlation between

the number of filaments and the severity of the disease. It should be noted that similar filamentous structures can be detected in other congenital myopathies, as well as in phenotypically healthy heterozygous carriers of the recessive gene.
Cases are described when the disease manifests itself at a later age by a decrease in strength muscles of the proximal regions, more often on the legs, or scapular-peroneal syndrome with Dangling foot.
Central nuclear (myotubular) myopathy. Inherited autosomal dominant or autosomal recessive, sporadic cases have also been reported. Facial muscles and extraocular muscles are affected in about half of patients. With a muscle biopsy, often fibers with centrally located nuclei are detected, in the absence of fibers with normal the location of the nuclei under the sarcolemma. Sometimes the fibers lack central myofibrils, in connection with which the fibers resemble embryonic muscle cells or myotubules (when following with a light microscope); hence the name "myotubular myopathy". V
In some cases, a decrease in the diameter of type I fibers is also observed.
X-linked myotubular myopathy (type I fiber hypotrophy with central nuclei). It is a separate form of congenital myopathy. The severity of clinical manifestations are very variable: sometimes the defeat is so severe that the child; dies 1-2nd day after birth. During pregnancy, there is little fetal movement. Children usually are born with cyanosis due to severe respiratory failure. If they survive then in the future they have a non-progressive generalized muscle weakness, grasping facial and extraocular muscles. In the study of biopsies, we are revealed cervical fibers resembling myotubules, as well as reduced in size (hypotrophic) type I fibers. Similar changes can be found in healthy carriers.
pathological gene.
Congenital myopathy with an imbalance in muscle fiber types. In children immediately after birth, hypotension and generalized muscle weakness are noted. Frequent contractures and multiple deformities of the skeleton, such as dislocation of the hip, kyphoscoliosis,
viral or hallux valgus. In the early years, muscle weakness can progress, in the future the condition stabilizes. Sometimes weakness can even decrease. Children often look disproportionately small for their age. With a biopsy reveals a relative decrease in the number of type I fibers in comparison with the number of type II fibers.
In half of the cases, however, the opposite relationship is observed with the prevalence of type I fibers.
Multi-core disease. In muscle fibers, there are multiple small areas (5-10 microns in diameter) of the disappearance of the transverse striation. It was found that in these
areas, there is not only disintegration of sarcomeres, but also a decrease in the number or absence mitochondria. If the size of such areas in diameter does not exceed 3 microns, then they speak of a disease
small rods (minicoredisease). This disease is especially rare. If the sizes of the zones lesions are larger (35-75 microns) and if they also contain vesicular nuclei, they speak of local disappearance of cross striation. With this disease, external

muscles of the eye and progression is noted. Sometimes in a biopsy or even in one fiber can all three described types of changes can be detected, which makes histological diagnosis unusually difficult.
Sometimes clinical manifestations of multi-rod disease immediately after birth are absent and appear only in childhood or even in adults. Transmitted autosomal in a recessive or autosomal dominant way.

MYASTENIA AND MYASTENIC SYNDROMES

Myasthenia gravis (miasteniagravispseudoparalitica) is characterized by a pronounced phenomenon
pathological muscle fatigue and muscle weakness. The etiology of the disease is not
installed. There are descriptions of familial cases, although the hereditary nature of the disease has not been proven.

In most cases, patients find a tumor or hyperplasia of the thymus gland. but
along with the high therapeutic effect of thymectomy or X-ray irradiation of the thymus
glands in some patients, myasthenia gravis is first detected after surgery for thymoma.

Pathogenesis. The pathogenesis of myasthenia gravis is complex. The fact of a violation of the neuromuscular
transmission, which is confirmed by a whole range of pharmacological effects and results
modern morphological studies using electron microscopy.

Postsynaptic block is associated with a decrease in the number of terminal cholinergic receptors
plates and / or their lack of sensitivity to acetylcholine, as well as impaired synthesis
acetylcholia as a result of a defect in enzyme activity. A determining role in the development of this
conditions play, obviously, autoimmune disorders.

Circulating antibodies to the protein of acetylcholine receptors were found in 90% of patients with
a generalized form of myasthenia gravis and in 70% of patients with an ocular form. There is a correlation
between the antibody titer and the severity of myasthenia gravis, however, it is not detected in all cases, which
indicates the heterogeneous nature of the disease. Treatment methods leading to a decrease
antibody titer, such as thymectomy, use of immunosuppressive drugs, plasmapheresis,
often lead to clinical improvement. Antibodies were originally thought to block
receptors, but then it turned out that they react not only with the active centers of the receptors,
but also with their protein component, causing accelerated degradation of receptors. An important role in
the formation of antibodies is played by the thymus, in which lymphocytes are found that produce
antibodies to receptors.

Thymoma is found in 10% of patients with myasthenia gravis, in general, the pathology of the thymus (more often in the form
hyperplasia) are detected in 60% of patients. In this case, myasthenia gravis develops in 30% of patients with thymoma,
60% of them are men.

Clinic. Myasthenia gravis is characterized by pathological muscle fatigue -
a unique and specific symptom of this disease. The muscle that develops at the same time
weakness differs from ordinary paresis in that with repetition of movements (especially in frequent
rhythm), it increases sharply and can reach the degree of complete paralysis. When working in slow motion
tempo, especially after sleep and rest, muscle strength remains relatively long.

The disease in most cases occurs between the ages of 20-30, although cases are not uncommon
the appearance of the first symptoms of myasthenia gravis in childhood or in puberty. More often (in 2
times or more) female persons get sick. Myasthenia gravis can also develop in old age,
then, as a rule, men are ill, and often they have thymoma. Disease development
more often subacute or chronic, although possibly acute, associated with provoking
the effects of infections, intoxication, as well as endocrine changes (pregnancy, menopause

period). By the nature of the course, a progressive form is distinguished (gradual
an increase in the severity and prevalence of myasteic manifestations), a stationary form, or myasthenic state (stability of myasthenic type defects for a significant
period) and myasthenic episodes (short periods of myasthenic disorders and long spontaneous remissions).
According to clinical symptoms, localized myasthenia gravis is distinguished - with lesion oculomotor muscles (ocular form), muscles of the tongue, larynx, pharynx (bulbar form) and the most common generalized myasthenia gravis.
In typical cases, oculomotor disorders appear first. Patients complain about

double vision of objects, especially with prolonged reading, drooping of the eyelids. Asymmetry is characteristic
lesions and dynamism of symptoms: in the morning hours the condition is better, in the evening ptosis and double vision
grow significantly. As a rule, in the initial stages there is only a defeat of certain
of the oculomotor muscles, however, as the process develops, a complete external ophthalmoplegia. Failure to maintain gaze for long periods of time while lateral abduction can create
erroneous opinion about the presence of nystagmus and, accordingly, internuclear ophthalmoplegia, which is
early stages can lead to a diagnostic error. The inner muscles of the eye are very affected rarely, pupillary reactions are preserved, although anisocoria or depletion of the reaction is sometimes observed
pupils to light upon repeated examinations. Weakness and fatigue join later.
mimic muscles and chewing muscles. The defeat of the bulbar muscles leads to dysfunction of the soft palate and epiglottis: patients complain of difficulty swallowing food, liquid entering the nose (these symptoms are aggravated by eating), to a "nasal" shade voices, its "fading", fatigue during a conversation. The complaint of fatigue is also typical.
chewing muscles when chewing solid food. In severe cases, patients are forced to take a break while eating. Sometimes they have difficulty even swallowing saliva.
Due to impaired swallowing, aspiration pneumonia or alimentary exhaustion may develop.
Often in patients, especially in the later stages, the tongue has 3 longitudinal grooves, which can have a certain diagnostic value.
When the weakness spreads to the muscles of the limbs, the muscles tend to suffer more.
proximal sections, at first in the hands. The traditional complaint of women about fatigue with combing. Neck muscles, especially the extensor muscles, are often affected; at the same time arises
characteristic writing off of the head. With generalized forms of myasthenia gravis, one of the most severe
the symptom is weakness of the respiratory muscles. Tendon reflexes can be preserved or depleted on repeated studies. Muscle atrophy is occasionally noted, especially in muscles of the tongue, neck, proximal arms; they are usually mild, with improvement conditions can decrease and disappear. No sensitivity disorders are observed,
movement disorders of the central type, as well as pelvic disorders.
Myasthenia gravis in childhood occurs in four clinical forms: neonatal,
congenital, early childhood and juvenile. Neonatal myasthenia gravis occurs in children born in mothers with myasthenia gravis, and develops as a result of the passage of antibodies across the placenta. Have
children have pronounced muscle hypotonia, weaker cry, weak sucking, frequent shallow breathing. The clinical picture may correspond to the "flaccid child" syndrome.
There is a clear therapeutic effect with the introduction of anticholinesterase agents. Usually all

the phenomena disappear after 4-6 weeks.
Congenital myasthenia gravis occurs in children whose mothers, as a rule, do not get sick myasthenia gravis. The disease can be manifested by weak fetal movement, and in the postnatal period -
weak cry, difficulty in sucking, swallowing, ptosis, limited mobility of the eyeballs, sluggish movements in the limbs. The course of the disease is long. Anticholinesterase drugs, plasmapheresis, and surgery are ineffective. Antibodies to acetylcholine receptors are not detected. Currently, several variants of congenital myasthenia gravis have been described.
In some cases, there is a congenital postsynaptic defect, in others - a presynaptic one. Type of inheritance is different. A small proportion of patients may experience spontaneous remission.
Early childhood myasthenia gravis develops, as a rule, in the 1st-2nd year of life, proceeds relatively mild, characterized in most cases by oculomotor disorders. V
the process may also involve mimic and chewing muscles.
The juvenile form is more common than others, the first symptoms appear at the age of 11-16 years, mostly girls are ill. There are, as a rule, generalized disorders. Can
join muscle wasting.
In old age, myasthenia gravis usually begins with oculomotor disorders, the process generalizes quickly, respiratory disorders appear early.
With myasthenia gravis, there may be an acute worsening of the condition due to the influence exogenous to endogenous causes, it is called myasthenic crisis. In patients with an average, and sometimes even mild forms of myasthenia gravis develop acutely generalized muscle weakness, severe bulbar disorders (aphonia, dysarthria, dysphagia), respiratory

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violations. The effectiveness of anticholinesterase drugs decreases. Muscle weakness sometimes reaching the state of tetraplegia, accompanied by pronounced vegetative disorders in the form of tachycardia, weak pulse, mydriasis and intestinal paresis. Sometimes during
death can occur in several tens of minutes.
Diagnostics and differential diagnostics. For the diagnosis of myasthenia gravis, an important it is important to identify characteristic complaints of transient disorders of motor activity, clearly dependent on the degree of physical activity, as well as on fluctuations in the intensity of all violations
niy during the day. Consideration should be given to the possibility of spontaneous remissions and exacerbations due to
intercurrent infections and intoxications. Particular difficulties are usually caused by recognition of myasthenia gravis that begins in old age. As a rule, in these cases it is preferable violation of cerebral circulation lays.
There are special tests to identify the patient's pathological
muscle fatigue. First of all, it is necessary to examine the muscles most affected
with myasthenia gravis: external muscles of the eye, involved in 90% of cases, bulbar muscles, muscles
proximal extremities and extensors of the neck. To provoke the appearance or
an increase in ptosis or diplopia, it is necessary to ask the patient for at least 30 seconds without looking up
look up or to the side (or at a light source). Dysarthria may appear in the process conversations, it can be provoked by asking the patient to count or read any text
aloud. Weakness of the masticatory muscles can be detected by asking the patient to quickly open and

close your mouth (a healthy person can make about 100 such movements for 30 seconds). Weakness
flexors of the neck can be identified by asking the patient lying on their back to raise their head and look
within 1 min on your navel. To identify weakness in the muscles of the shoulder girdle, it is necessary that
the patient stretched his arms forward or to the sides and maintaining this position for 3 minutes. For determining
weakness of the leg muscles, you can ask the patient to do deep squats, walk on socks, heels. In some patients, the phenomenon of M. Walker can be detected: repeated contractions and
unclenching of the hands causes not only weakness of the muscles of the forearms, but also an increase in ptosis. If at
When performing the test, a compression cuff is applied to the shoulders, then ptosis develops during
seconds after removing the cuff.
Proserin test occupies a very important place in diagnostics. It should be remembered that the dose of proserin should be sufficient from 1.5 to 3 ml (depending on body weight) 0.05% solution,
which is injected subcutaneously. To eliminate the side effects of proserin, atropine is prescribed (0.5
ml of 0.1% solution). In typical cases, a dramatic effect is noted - 20-40 minutes after introduction, regression of almost all symptoms occurs. It is natural to return them to their original
level after 2-2.5 hours. There are forms of myasthenia gravis, relatively insensitive to anticholinesterase drugs, in particular the ophthalmic form.
An especially important method for verifying the diagnosis of myasthenia gravis is electromyographic
study. Supramaximal stimulation of the motor nerve is performed with a frequency of 1-3 v give me a sec. The surface electrode registers the total potential of muscle action. First
the action potential is usually normal, the amplitude of the subsequent ones progressively decreases. For mi-
Asthenia typically decreases the amplitude of the action potential by at least 10%. There may be other electrophysiological phenomena: post-tetanic potentiation, post-tetanic
exhaustion. The degree of block of neuromuscular transmission on EMG examination usually corresponds to
the severity of clinical signs of impaired motor function.
A muscle biopsy is not of significant diagnostic value. Can be detected
type II muscle fiber atrophy, signs of denervation, lymphocytic infiltration, but how
usually, the biopsy is normal: More reliable changes are found in a biopsy taken from
areas of end plasty, followed by a study by the method of intravital staining
methylene blue or using electron microscopy (expansion of the synaptic cleft).
Differential diagnosis is carried out with multiple sclerosis, intrastem tumor, polioencephalitis, basal meningitis, and with the skeletal form of the disease - with myopathy, poliomyositis, McArdle syndrome.
Treatment. The main objectives of therapy are to improve neuromuscular transmission and prevention of further destruction of acetylcholine receptors by affecting the autoimmune
a process directed against acetylcholine receptors.
Anticholyesterase drugs. The most commonly used are proserin and kalymin (mestinon).

These drugs inhibit cholinesterase, an enzyme that hydrolyzes acetylhodine to choline and

acetylcoenzyme A in the synaptic cleft.
Improvement in neuromuscular transmission occurs as a result of increased concentration acetylcholine in the synaptic cleft, however, it is possible that the mechanism of action of these drugs is more complex.
Proserin is a short-acting drug: it leads to a quick effect,
the maximum concentration of the drug in the blood plasma is observed in less than an hour, discontinuation
actions - after 2-3 hours. Patented tablets contain 15 mg of proserin. If swallowing is impaired Proserin (the ampoule contains 1 ml of a 0.05% solution) is administered either subcutaneously in 3 ml (1.5 mg) doses, or
intravenously, 1 ml (0.5 mg). The effect of taking Kalimin comes slower and lasts for within 3-5 hours after taking the drug. Proserin is taken periodically, mainly for short-term increase in muscle strength before the upcoming physical activity or before food. The daily dose of Kalimin, divided into several doses, provides long-term effect all day long. The dose of drugs is selected individually. With moderate the severity of the disease may be sufficient to take proserin immediately before meals or 60 mg of Kalimin 3-4 times a day. In severe cases, frequent intake of Kalimin is required, sometimes
every 3 hours during the day and possibly at night.
In severe myasthenia gravis, increased muscle weakness during treatment anticholinesterase drugs may be due to an exacerbation of the disease or the development cholinergic crisis as a result of competitive depolarization blockade of acetylcholine receptors. With a cholinergic crisis, fasciculations, narrow pupils, hypersalivation are observed, increased sweating, pallor, bradycardia, diarrhea, intestinal colic. However, if the patient simultaneously receives atropine (to prevent the undesirable effects of anticholinesterase funds), the clinic of the crisis may be less clear. It should be borne in mind that the most resistant to
anticholinesterase therapy of the respiratory muscles, therefore, in this situation, translation is necessary
the patient in the intensive care unit, the cancellation of all drugs, carrying out according to the indications of mechanical ventilation. Selection
a new dose of drugs is started in 2-3 days.
Sudden increase in muscle weakness with the onset of respiratory failure
may be due to myasthenic crisis. Distinguish myasthenic crisis from much less often occurring cholinergic is not easy; in addition, it should be borne in mind that there may be a combination of both types of crisis. If in doubt about the diagnosis, you should refuse to use anticholinesterase agents and carry out symptomatic therapy, especially mechanical ventilation. Conducting a proserin test in unclear cases may (in the presence of a cholinergic crisis) sharply worsen the condition, therefore, if it should be carried out, then with extreme caution. For
treatment of myasthenic crisis parenterally use proserin, in addition, use prednisolone at a dose of 100 mg for 3 weeks.
Long-term treatment with anticholinesterase agents in high doses of skin leads to violation of neuromuscular transmission and morphological changes in the terminal motor plate. Electromyography detects neurogenic atrophy in these patients.
muscles.
Thymectomy. On average, stable remission or improvement is noted according to various statisticians in 70% of those operated. The best results were observed in people with a short history.
diseases (from 2 to 5 years) regardless of gender and age.
Prospect during surgery is worse in elderly patients, in the presence of

thymoma and in the case of its invasive growth (in comparison with the localized form). Detection in the thymus gland of a large number of germinal centers is an unfavorable sign. Postoperative radiation can cause a transient deterioration in the patient's condition.
Thymectomy is indicated in the presence of thymoma, as well as in all patients with generalized myasthenia gravis, as well as with severe course of other forms of myasthenia gravis (including ocular) with lack of effect from therapy with anticholine-esterase drugs. It is not recommended to conduct operations for patients over 70 years old, as well as with a slight severity of symptoms. Most a reliable method of imaging an enlarged thymus without evaluating benignity process is CT of the anterior mediastinum. Conventional lateral tomography results less reliable.
A low level of surgical mortality has been achieved in specialized centers. Maintaining postoperative patients are carried out in intensive care units; in the first days a dose

anticholinesterase drugs are reduced to about 50% of the preoperative. In this period Proserin is usually used parenterally. Patients operated on for thymoma are carried out irradiation of the anterior mediastinum.
In terms of preparation for the operation, plasmapheresis is performed in order to achieve a temporary remission. This procedure is carried out periodically for several months before the operation, and also after her.
Immunosuppressive therapy. Steroids are often very effective for myasthenia gravis and allow you to achieve long-term remission. However, in severe cases, according to some researchers, before starting the use of steroids, it is advisable to perform a thymectomy, bearing in mind the possibility of side effects and less favorable conditions for the operation and healing in the postoperative period against the background of steroid therapy. Preferred appointment the drug once a day; Prednisolone is used more often. Prescribing prednisone often increases muscle weakness. Therefore, it is recommended to start treatment with a small dose - 10 mg every other day, then it is increased by 10 mg every week, so the optimal dose is 50-100 mg every other day achieved after about 10 weeks; upon reaching remission, the dose is gradually reduced by 5 mg per a month to a maintenance dose of 20-40 mg every other day. For prophylactic purposes, reception is shown potassium chloride, H-receptor blockers. Within 1 month, improvement in the condition occurs in 80% of patients, however, after 3 months, positive dynamics persists in less than 50% of patients.
The results of steroid therapy are independent of previous thymectomy or existing pathology of the thymus gland. During treatment with steroids, the level of antibodies to receptors acetylcholine is usually reduced. However, the possibility of long-term use of these drugs limited due to the side effects caused.
Azathioprine has an effective immunosuppressive effect. He is prescribed daily at increasing doses - from 50 to 150-200 mg / day. While taking azathioprine, you can significantly reduce the dose of steroids faster. The clinical effect appears after 6-12 weeks and reaches maximum after a year or more from the start of treatment. Against the background of improvement in the condition, gradually

reduce the dose of anticholinesterase drugs. Thymus pathology does not affect the observed clinical effect of azathioprine; during treatment, the level of antibodies against receptors are progressively reduced.
Azathioprine therapy can be continued for several years, and only in some cases cancellation the drug is possible without subsequent relapse of the disease. Against the background of the reception azathioprine in the blood, macrocytosis is detected, which may indicate the use a sufficient dose of the drug and an adequate response to treatment.
Cyclosporin is also used among immunosuppressants for the treatment of myasthenia gravis. Before implementation in the practice of plasmapheresis, drainage of the thoracic duct was performed, which, as a rule, short-term effect. We also used anti-lymphocytic and anti-monocytic globulins, irradiation of the spleen and the whole body.
Plasmapheresis. The essence of the procedure is the exchange using a cell separator 2-4 liters the patient's blood plasma on a heated composition containing purified fractions of human protein (50%), calcium and potassium in physiological concentrations, dextran (25%), Ringer's solution (25%).
The procedure can be repeated as needed. Positive dynamics is noted in a week after the start of the course of plasmapheresis, in severe cases, daily plasma exchange is required. The maximum possible improvement is observed after 2 weeks and can persist for 4-6 weeks. Longer remission can be achieved with simultaneous use immunosuppressants.
The main indications for plasmapheresis are the acute course of myasthenia gravis, myasthenic crisis, the initial stages of steroid therapy in connection with a possible clinical worsening and rarely long-term treatment of severe forms of myasthenia gravis in case of ineffectiveness of others methods. Possible complications of plasmapheresis include septicemia, air embolism, hypotension, convulsions, thrombophlebitis; most of them can be avoided with proper technical performance of the procedure.
Management of patients with myasthenia gravis.
1. Establishing a diagnosis based on clinical presentation and results electrophysiological research. The most accurate diagnostic method is

determination of the level of antibodies against acetylcholine receptors.
2. The use of cytostatics (azathioprine or faster, although causing more pronounced side effects, cyclophosphamide) can usually reduce the required dose of steroids. They can also be used in isolation. Unlike steroids Azathioprine is less toxic and can be taken for a long time. During treatment, it is necessary periodically conduct a CBC and liver function tests.
3.
Sick women should avoid pregnancy, as some patients the condition can worsen significantly, especially in the first trimester and during childbirth. In patients with myasthenia gravis during the first trimester, miscarriages often occur. Carry out an abortion according to medical indications are not recommended as they may aggravate. During childbirth, avoid general anesthesia, replacing it if possible with local or regional. With myasthenia gravis newborns, special measures are taken to ensure adequate breathing, the introduction nutrients, preventing aspiration. Anticholinesterase agents are administered.

4.
Patients with myasthenia gravis should avoid taking the following drugs that can increase muscle weakness: relanium, diphenin, trimethine, aminoglycosides, streptomycin, erythromycin, lincomycin, ampicillia, diacarb, quinine, novocainamide, penicillamine, β-adrenergic blockers.
5.
It should be borne in mind that the course of the disease is difficult to predict and highly variable.
It is influenced by intercurrent infections, endocrine factors (menstruation, menopause), emotional stress. As a rule, the disease reaches its peak after 3-5 years, after where there are only small changes in the severity of symptoms, and new symptoms appear rarely.
If all of the above measures are ineffective as a last resort in in severe cases, total irradiation is used at a dose of 15 glad 2 times a week for 5 weeks.
Myasthenic syndromes. Lambert-Eaton syndrome. Paraneoplastic syndrome which is characterized by weakness and fatigue of the proximal muscles of the extremities, more often the legs, with the relative safety of the extraocular and bulbar muscles. Weakness often diminishes with physical exercise, but with continued load it reappears. Tendon reflexes may be reduced or absent. Frequent complaints of dry mouth and muscle pain. Sometimes there are paresthesias, impotence, ptosis. Unlike myasthenia gravis among patients with the syndrome Lambert-Eaton is dominated by men, usually over 40 years old. The syndrome is especially common with lung cancer, often its development is ahead of the main symptoms by several months or even years. In addition to lung cancer, the syndrome can occur with malignant tumors of other organs - prostate, stomach, rectum. There are reports of idiopathic forms. The syndrome has also been described in many autoimmune diseases, including diseases of the thyroid gland, while taking certain medications (for example, neomycin). Based on long-term material from the laboratory of B.M. Hekht (about 5000 patients with myasthenia gravis) Lambert-Eaton syndrome was found only in 50 patients.
The prognosis depends on the etiology. It has been experimentally established that the disease is due to the interaction of antibodies with the antigenic determinant of the presynaptic membrane, which determines release of acetylcholine at the neuromuscular synapse. As a result, the presynaptic release of acetylcholine. An increase in the size of the end motor plates in the postsynaptic region, which is associated with an increase in the synthesis postsynaptic acetylcholine receptors. At the same time, the number of presynaptic active zones are reduced (the latter, possibly, represent calcium channels). It is these zones, like are believed to be the target of an immune attack. It has been shown that the disease develops in sick with certain HLA status. Oatmeal cancer (emerging from neuroectodermal cells) and cholinergic neurons are thought to share antigens in common. There are no specific laboratory changes. Research should focus on

search for a tumor. On EMG, the amplitude of the initial evoked potentials at supramaximal less stimulation than normal. With stimulation at a frequency of 2 Hz, a slight increase is observed
amplitudes of potentials, as is the case with myasthenia gravis. With an increase in the frequency of stimulation (more
10 Hz), the amplitude of muscle potentials sharply increases the phenomenon of training. Increase
the amplitude of muscle potentials is also observed after exercise.

Electromyography using concentric needle electrodes reveals
only minimal changes.
In some cases, there is also peripheral carcinomatous neuropathy with a decrease speed of conduction along the nerves.
Muscle biopsy shows no specific changes.
Lambert-Eaton syndrome is sometimes difficult to recognize, especially in patients with cachexia.
Differential
diagnostics
spend
with
myasthenia gravis,
disease
motor neuron,
carcinomatous polyneuropathy and polymyositis.
Removal of the tumor only occasionally reduces muscle weakness, while at the same time after surgery
the degree of neuromuscular transmission defect may increase due to the use of muscle relaxants and anesthetics. An increase in the release of acetylcholine is caused by the infusion of calcium gluconate, more
long-term improvement is possible with guanidine ingestion. Effect of application
the drug is usually observed after a few days. However, due to the pronounced side phenomena: chronic interstitial nephritis, bone marrow suppression - drug
rarely used.
4-aminopyridine also has a similar mechanism of action. Its action is potentiated anticholinesterase drugs.
There is experience in the successful use of plasmapheresis and immunosuppressive therapy. After
After a plasmapheresis session, improvement occurs in a few days.

DISEASES OF THE PERIPHERAL NERVOUS SYSTEM
POLYNEUROPATHY
This large group of neuropathies includes symmetrical, diffuse lesions of the PNS. Polyneuropathies are divided into axonopathies and myelinopathies, which are based on anatomical
physiological concepts of the functioning of the PNS.L. Cavanagh proposed classification acquired axonal polyneuropathies. The first group includes volatile
neuropathies with a presumptive defect in oxidative metabolic processes - limited distal axonopathy with thiamine, riboflavin deficiency, arsenic poisoning, thallium.

The second group consists of axonal neuropathies - distal, mainly
sensory, with more proximal nerve damage than in diseases in the first group:
isoniazid polyneuropathy.
The third group included polyneuropathies involving the long conductors of the spinal cord
and peripheral nerves: in case of poisoning with acrylamide and organophosphorus compounds.
Segmental demyelination usually means primary destruction
myelin sheath with an intact axon (as opposed to secondary demyelination in
axon degeneration). In this case, Schwann cells are selectively affected and myelin is destroyed,
the process often begins in the area of Ranvier's interceptions. After repeated episodes of
demyelination and
remyelination, “bulbous heads” appear. This term refers to circular leaves.
Schwann cells surrounding the axonal rod. The result of segmental demyelination
is the blockade of the impulse or its pronounced slowdown. The muscle is not
denervates, but its atrophy may develop from prolonged inactivity. With active
remyelination recovery can occur quickly, complete recovery is possible, for example
with acute diphtheria demyelinating polyneuropathy.
In most cases, the disease manifests itself in symmetrical sensory or motor
disorders or more often a combination. Distal tendon reflexes, especially Achilles,
usually absent. Sensitive disorders are of the “socks” and “gloves” type. Isolated
motor or sensory polyneuropathy is rare. Equally vegetative
neuropathy is usually part of generalized polyneuropathy and as an isolated syndrome
is very rare. In case of involvement in the process, in addition to peripheral nerves,
spinal roots more adequate term "polyradiculoneuropathy". In these cases
usually dominant proximal muscle involvement, often cranial
neuropathy, and an increased protein content is found in the cerebrospinal fluid.
As a rule, with polyneuropathy, the lesion of the legs dominates. The onset of the disease from
the hands, and their
predominant damage is sometimes observed with lead and porphyric neuropathy, with
B12-deficiency neuropathy and Guillain-Barré syndrome. Damage to the autonomic nervous
system
leads to orthostatic hypotension, abnormal heart rhythm, impaired sweating,
dysfunction of the pelvic organs (in total - about 30 syndromes). Progressive vegetative
failure is observed in diabetes, amyloidosis, porphyria, alcoholism, carcinomatous
sensory neuropathy, acute inflammatory demyelinating polyneuropathy, some
hereditary forms, etc.
Thickening of the peripheral nerves, detected with palpation, occurs with leprosy,
amyloidosis,
illness
Refsum,
chronic
inflammatory
demyelinating polyradiculoneuropathy (CIDP), a hypertrophic form of Charcot's disease -
Marie - Tooth.
There are four types of polyneuropathy: acute (symptoms develop faster than
per week), subacute (no more than 1 month), chronic (more than a month) and recurrent, when
repeated exacerbations occur over the years.
Below is a description of the most relevant forms of neuropathy for practice.
It seems appropriate, in addition to highlighting the characteristics of each polyneuropathic
syndrome, indicate other forms of neuropathy accompanying this form of polyneuropathy, and
also lesions of the central nervous system. An attempt at rigid fixation only in the picture of
polyneuropathy
impractical.

Functional data can be used to analyze the clinical symptoms of polyneuropathy.
thin and thick fibers that make up the peripheral nerve. All motor fibers are
thick myelinated fibers. Proprioceptive and vibration sensitivity
are also carried through thick myelinated fibers. Fibers that transmit pain and

temperature sensitivity, refer to unmyelinated and thin myelinated;
vegetative fibers - to thin unmyelinated ones, while in the transmission of tactile
sensitivity involves thick and thin fibers. Damage to fine fibers can lead to
selective loss of pain and temperature sensitivity, burning pain and dysesthesia with
absence of paresis and with normal reflexes. Thick fiber neuropathy causes muscle
weakness, areflexia, sensitive ataxia and mild impairment of superficial sensitivity.
The involvement of all fibers leads to mixed (sensorimotor and autonomic) polyneuropathy.
It should be borne in mind that this relationship between the nature of the lesion and the clinical picture
are not absolute.
Pain in polyneuropathies depends mainly on the severity of the process, as well as on the type and
the size of the affected fibers. Chronic idiopathic sensory neuropathy is accompanied by
ataxia due to the defeat of thick myelin fibers. Patients are worried about paresthesia,
violation of proprioceptive sensitivity, but pain is completely absent. Very difficult and
the genesis of pain in some forms of diabetic neuropathy, also occurring with
defeat of thin nerve fibers. Hyperglycemia is known to reduce pain threshold and
reduce the antinociceptive effect of analgesics, therefore, in diabetic neuropathies
normalization of blood glucose can lead to a significant reduction in pain. Hyperpathy
dysesthesia and allodynia in neuropathies are usually associated with the regeneration of axonal processes in the
the damaged segment of the nerve. The outgrowths appearing in the process of regenerative sprouting can
be a source of ectopic spontaneous impulse activity. They also have the conditions
for epaptic transmission of electrical activity (transmission of a nerve impulse during direct
contact of axons without the participation of a mediator), leading to spontaneous pain sensations.
Movement disorders in polyneuropathies are often localized distally. However, with
some types of polyneuropathies (porphyria, Guillain-Barré syndrome) proximal groups
muscles may suffer more than distal ones. Fasciculations may appear with a lesion
roots.
The study of cerebrospinal fluid is important in diagnosis. With acute and
chronic inflammatory demyelinating polyradiculoneuropathy
protein is one of the diagnostic criteria. Moderate increase in protein content
also typical for other demyelinating polyneuropathies, including diabetic.
Cell-protein dissociation is characteristic of Bannwart's meningopolyneuritis in Lyme borreliosis and with AIDS.
Along with a clinical examination to assess neuromuscular disorders in
for polyneuropathies, electrophysiological research is of great importance.
A biopsy of the cutaneous nerve provides essential assistance in diagnosis. Usually for study
take the sural nerve or the superficial branch of the radial. Biopsy helps diagnose
amyloidosis, leprosy, metachromatic leukodystrophy, metabolic diseases, some
cases of CIDP. A biopsy is indicated only in cases where a diagnosis cannot be made using
non-invasive methods.
It should be emphasized that, despite an exhaustive examination, in about 1/3 of patients
with polyneuropathy syndrome, the etiological diagnosis cannot be established.

Trigeminal neuralgia - Fosergil's disease

Pathogenesis. The primary link in the pathogenesis of this disease is, as a rule, lesion
a peripheral segment of a nerve - a branch or root. The latter is confirmed by the well-known fact
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turning off the affected branch, for example, alcoholization, leads to the cessation of pain attacks
before
until the nerve regenerates.
Under the influence of the compression factor and prolonged subthreshold irritation with
peripheries in the brain, it is believed, an algogenic system arises with stability, high
excitability and responding to any afferent messages with paroxysmal excitation.
Clinic and diagnostics. The disease usually develops after the age of 50. Amazed
more often the maxillary or mandibular nerve (the second or third branch of the trigeminal
nerve).
Often, at the beginning, pains are local in nature, projecting into the area of a particular tooth,
gums,
in connection with which patients turn to dentists. Soon the area of pain increases, capturing,
as a rule, the zone of innervation of the corresponding branch. A feature of pain is
short duration of paroxysm, usually lasting for several seconds. Despite this,
the intolerable nature of the pain makes it possible to classify trigeminal neuralgia as extremely
severe suffering. In addition, it should be added that in the period of exacerbation, pain attacks

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provoked by any stimuli - talking, chewing, eating, facial movements.
Patients cannot wash, shave, eat only liquid food with difficulty, often explain
exactly. The pains are compared with the passage of an electric current, excruciating twitching,
lumbago
("As if a red-hot rod was stuck in the face"). It is characteristic that during painful paroxysms
the patients do not scream, do not rush, and those stunned by the terrible pain freeze, fearing to
move. V
in more rare cases, patients rub their cheeks or press on the temple in order to relieve an attack
(antagonist gesture).
It has long been noticed that with trigeminal neuralgia, pain attacks are often
accompanied by a spasm of the muscles of the face - pain tic (ticdouloureux). Muscle spasms can
be
partial, capturing the circular muscle of the eye, buccal muscle, or take hemifacial
character.
Pain attacks are usually accompanied by autonomic disturbances - nasal congestion
or the separation of fluid secretion, lacrimation, flushing of the face. Paroxysms rarely occur in
night time. If the first cardinal sign of trigeminal neuralgia is
pain attacks characteristic of her, then the so-called trigger
zones - areas of the skin or mucous membranes of various sizes that have superexcitability.
Irritation of these areas - touch, cold wind, skin displacement - usually
cause a painful attack. Kurkovye zones appear during the period of exacerbation of the disease
and disappear
in the period of remission. There is a tendency towards the location of the trigger zones in the
medial regions.
faces. Kurkovye zones are also located on the mucous membrane of the oral cavity, and in some
patients
- only in this area. Often the trigger zone is a tooth.
In an objective examination of patients, in addition to the trigger zones, it is often noted
soreness when pressed at the exit point of the corresponding branch of the trigeminal nerve on
the face,

sometimes areas of hyperesthesia in the innervation zone of the affected branch. As the disease progresses,
the clinical picture often reveals certain changes: in the pauses between attacks can
a feeling of dull pain persists or a burning sensation occurs, hyperesthesia is replaced
hypesthesia and even anesthesia. However, the cardinal signs of neuralgia are characteristic of it.
painful paroxysms and trigger zones - persist. It should be assumed that there is a single
a disease that has different causes, but the same pathogenesis.
The diagnosis is based on the identification of the paroxysmal nature of the pain, the characteristics of pain
seizures, the presence of trigger zones on the skin of the face or in the mouth, provoking seizures with food,
washing, shaving, etc. In some cases, a false impression of an unusual
the duration of pain attacks for several hours. Meanwhile, statusneuralgicus is
a series of painful attacks following each other. However, even in this situation, a thorough survey
allows you to establish the true nature of the attacks.
In rare cases, neuralgia of individual branches of the trigeminal nerve - the lingual nerve,
upper alveolar nerves, lower alveolar nerve. They are all kind
partial form of trigeminal neuralgia, are manifested by painful
seizures and trigger zones with limited localization. So, with neuralgia of the lingual nerve
attacks of pain occupy the anterior 1/3 of the corresponding half of the tongue and are also located here
trigger zones. With neuralgia of the upper alveolar nerves, pain attacks are localized in the area
teeth and part of the upper jaw, and with neuralgia of the lower alveolar nerve - in the area of the teeth
the lower jaw, trigger zones are usually found in the area of the corresponding gums, one or another tooth.
Lingual nerve neuralgia must be distinguished from glossopharyngeal neuralgia, in which
pain and trigger zones are localized mainly in the region of the root of the tongue, tonsils, palatine
arches, as well as glossalgia. In the latter case, a surprisingly large number of patients complain of
pain, burning sensation, sensation of hair on the tongue, dry mouth. Pain attacks and trigger zones
no. All sensations disappear while eating. The disease is a classic form of psychogenic
algium. The diagnosis of idiopathic trigeminal neuralgia requires exclusion of all others.
causes of nerve compression and pain in the face. Essentially toothache, sinusitis, glaucoma are secondary
forms of trigeminal neuralgia. Occasionally, the latter can be observed in multiple sclerosis.
and tumors of the brain stem, which must be taken into account, especially in young patients and in the presence of additional focal symptoms.
Treatment. In 1962, carbamazepine entered the practice of treating trigeminal neuralgia, which
opened up real prospects for successful treatment, at least 70% of cases of this difficult
suffering. Other anticonvulsants are also widely used: diphenin, suxilep,
clonazepam, sodium valproate. The dose of the drug is selected individually. Daily dose

carbamazepine (finlepsin) is usually 400-1200 mg / day. After reaching the therapeutic
effect (cessation of painful attacks), the dosage of the medication is reduced to the minimum when
which the therapeutic effect persists, and this dose of the drug is used by the patient for
long time (supportive therapy). When carrying out maintenance therapy with

over time, in a number of patients, the effect decreases, the drugs can be used in isolation, and (with insufficient efficiency) in combination.
In some cases, baclofen is effective, which enhances presynaptic inhibition in neurons of the nucleus of the spinal tract of the trigeminal nerve, as well as pimozide (orap). As with other pain syndromes, the use of antidepressants, as well as percutaneous electrical stimulation of the affected branch. Low efficiency conservative therapy of trigeminal neuralgia in the era before the use of antiepileptic drugs was the reason for the development of various methods of surgical treatment, many of which currently have only historical interest. The operation is applied as on peripheral branches of the trigeminal nerve, and intracranial structures.
"Chemical cutting" of the nerve by introducing 80% alcohol into the place of its exit on the face with novocaine is currently almost abandoned, as it brings only a temporary effect. WITH by regeneration of the nerve, pains are restored, however, a productive process arises in the nerve, which leads to a decrease in the effectiveness of both repeated blockades and the use of antiepileptic funds.
Discovered the possibility of compression of the trigeminal nerve root in the posterior cranial fossa an abnormally located vessel or aneurysm has opened a new page in neurosurgical treatment. Similar neurovascular conflicts were noted in facial hemispasm, vestibular ataxia, spastic torticollis, glossopharyngeal neuralgia.

Facial nerve neuropathy

For the first time, the clinic of defeat of the facial nerve was described by S. Bell in 1836. Currently, the term Bell's palsy is used to refer to an idiopathic lesion of the facial nerve, while while the term "facial nerve neuropathy" (ILN) also includes forms with known etiology.
NLN is the most common cranial neuropathy, affecting 13-24 people per 100,000 population, equally often in men and women. This prevalence of ILN is due to probably by its course in a narrow bony facial canal (fallopian canal) of the pyramid of the temporal bone and features of vascularization, which creates the preconditions for its ischemia, edema and compression.
Etiology. Most cases of NFN are associated with damage to a nerve in the facial canal. The leading pathogenetic factor of its defeat is ischemia, edema and compression in a narrow bone canal. NLN can occur against the background of arterial hypertension, atherosclerosis, sugar diabetes, viral diseases, diseases of the middle ear and parotid glands, multiple sclerosis, acute and chronic inflammatory demyelinating polyradiculoneuropathy, with trauma temporal bone, tumor of the cerebellopontine angle, metastatic or leukemic infiltration. Hypothermia before the onset of the disease, which is often indicated by patients, can be a starting point. Family-inherited IPN is a special case.
facial nerve damage in Ramsay Hunt syndrome, Melkersson-Rosenthal syndrome, neurosarcoidosis, Moebius syndrome, AIDS, Lyme disease.
Clinic. The main syndrome of NFN, regardless of the level of damage, is weakness facial muscles of half of the face (prosoparesis, prosoplegia). The patient's face is asymmetric, skin folds on the affected side are smoothed, the corner of the mouth is lowered. The patient cannot wrinkle his forehead, frown, close your eyes, show your teeth, whistle, puff out your cheeks. On the affected side of the eye

the slit is wider, blinking is reduced, when the eyes are closed it does not close (lagophthalmos is the “hare” eye).
When the eyes are closed, the eyeball moves up and outward, while a white becomes visible a strip of sclera (Bell's symptom). Due to paralysis of the eyelids, the circulation of the tear fluid is impaired, and
a tear trickles down my cheek. Decreased superciliary and corneal reflexes. With mild paresis, circular
the muscles of the eye with a strong screwing up of the eyelashes do not completely go into the palpebral fissure (symptom
eyelashes). Food gets stuck between the cheek and gum, while chewing, the patient often bites cheek, liquid food pours out from the corner of the mouth. Weakness of the subcutaneous muscle of the neck can be detected if
counteract the tilt of the head forward with the mouth wide open.
The clinical picture of NFN depends on the level of damage to the facial nerve.
1. When the nucleus is damaged, prosoplegia is observed on the side of the lesion, but isolated damage to the nucleus is rare. This is usually combined with the involvement of the intracerebral part.

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the root of the facial nerve, the nucleus of the abducens nerve and the pathways of the bridge (alternating
Miyard-Gubler and Fauville syndromes).
2. With the defeat of the root in the cerebellopontine angle at the entrance to the internal auditory the opening of the temporal bone, prosoplegia, hearing loss and vestibular excitability are observed,
violation of taste in the anterior 1/3 of the tongue and dryness of the eye.
3. In case of damage to the trunk of the nerve in the facial canal to the knee (that is, before the large
stony nerve) - prosoplegia, dry eyes, hyperacusis, disorders of taste and salivation.
4. In case of damage to the nerve in the bone canal below the branch of the large stony nerve - prosoplegia, increased lacrimation, hyperacusis, taste and salivation disorders.
5. In case of damage to the nerve in the bone canal below the discharge of the stapedial nerve and above
drum string - prosoplegia, lacrimation, taste and salivation disorders.
6. In case of damage to the nerve in the bone canal below the discharge of the tympanic string or after
exit from the styloid opening - prosoplegia, lacrimation.
It should be borne in mind that lacrimation (levels 4, 5, 6) is not due to damage, but
the safety of lacrimal fibers and is a consequence of a violation of the tear flow due to paralysis of the circular muscle of the eye. Impaired salivation (dry mouth) by patients like as a rule, it is not noticed due to the preservation of the function of the salivary glands of the healthy side.
Some patients with NFN at the onset of the disease or during its development report pain or numbness in
the parotid region (mastoid process, auricle), which is due to the involvement of the posterior the ear nerve and, possibly, the connections of the facial nerve with the trigeminal nerve system.
It is assumed that unilateral involvement of the facial nerve in Bell's palsy is due to individual morphofunctional relationships of the nerve, bone canal and
blood supply. Bilateral HJIH is more likely to indicate the presence of a systemic disease. NLN usually develops acutely or subacutely. The muscles of the lower face tend to be more affected. than the top. The restoration of the function of facial muscles begins from the upper part of the face,

gradually spreading to the bottom. It usually takes no more than 2-4 months, but in some
In favorable cases, it can be delayed.
Diagnostics. The diagnosis of Bell's palsy in typical cases is straightforward. However, always
it is necessary to exclude the possibility of secondary forms of neuropathy. It should be borne in
mind that despite
the traditional scheme, according to which central nerve palsy is manifested as a defect only
of the facial muscles, damage to the corticomuscular pathway actually causes
very mild insufficiency and circular muscle of the eye (symptom of eyelashes). Occasionally due
to
individual structural features of the central motor neuron of the facial nerve with hemispheric
lesions can experience such a significant weakness of the upper facial muscles that there is
a picture almost indistinguishable from neuropathy. The correct diagnosis in such cases helps
the presence of a pyramidal defect and central paresis of the hypoglossal nerve on the side of the
paresis
mimic muscles, as well as the preservation of the brow reflex (closing of the eyelids with
percussion
superciliary arch), which always falls out when the trunk of the nerve is damaged. About the
degree of defeat
facial nerve (VII) can be judged by ENMG and blink reflex data.
Treatment. Bell's palsy treatment aims to relieve swelling and repair
microcirculation in the nerve trunk. For this, glucocorticoids are used, which dehydrate and
vasoactive agents. Prednisolo is taken orally starting from 60-80 mg daily in the morning for
7-10 days, followed by rapid withdrawal of the drug. The effect of hormone therapy is higher if
it
begins in the first 4 days of the disease. The effectiveness of corticosteroid therapy in later
terms, as well as commonly used diuretics, vasodilators, vitamins
group B is doubtful. From the first days of the disease, gymnastics of facial muscles is
recommended,
stickers made of adhesive plaster to prevent overstretching of the affected muscles, paraffin
applications; from 7-10th day - massage, acupuncture. With dry eyes and lagophthalmos
a protective eye patch and moisturizing with eye drops are recommended. Appointment of
proserin
contraindicated. Surgical decompression of the facial nerve is ineffective.
Complications. A frequent and persistent complication of NFN is contracture of facial muscles,
pathological synkinesis. For treatment, massage is used with kneading local muscle
nodules, post-isometric relaxation of the affected muscles, carbamazepine, clonazepam,
diphenin,
baclofen. However, a lasting effect is usually not achieved.
Forecast. Unfavorable prognostic signs: elderly patients, hyperacusis,
deep paralysis with a gross loss of muscle tone, leading to pronounced asymmetry of the face.
However, a categorical judgment about the outcome of the disease is always problematic. Even
in the lightest cases
contracture of facial muscles may develop. Electrophysiological data make it possible to make

the prognosis of the disease after 5-7 days from its onset ..
About 80% of patients recover completely after a few weeks or months. V
in other cases, recovery is incomplete or does not occur at all. As a result
the weakness of the facial muscles remains of varying severity, eversion of the eyelids
(ectropion),
contractures of the affected muscles, synkinesis, and sometimes facial hemispasm
disappear. Subjectively

a painful complication - crocodile tears syndrome: due to aberrant regeneration
salivary fibers grow into lacrimal fibers, which leads to abundant
discharge while eating (Bogorad's syndrome). Another rare complication is geniculatory
neuralgia (geniculate neuralgia) - excruciating pain in the affected half of the face,
radiating into the external auditory canal.

NEUROLOGICAL COMPLICATIONS OF OSTEOCHONDROSIS OF THE SPINE

In connection with the acquisition of a vertical position in a person, the lower lumbar and
the lower cervical spine is subjected to significant overload. So early, starting
from the 3-4th decade of life, wear of the indicated vertebral segments begins. Spine
a ny motor segment is a pair of adjacent vertebrae, an intervertebral disc and the connecting
fibrous tissue and intervertebral muscles. Arising under the influence of static-dynamic
loads, deformation of the tissues of the spinal motor segment are the cause of constant
lingering irritation of its receptors, especially pain receptors. These deformed tissues (primarily
the affected discs) can also have a mechanical effect - compression - on the dorsal
brain and nerve roots. Vertebrogenic syndromes are the most common
chronic human diseases - every second person during his life suffers from pain in
back or neck.

Among the lesions of the spine, accompanied by neurological disorders,
the most common degenerative-dystrophic processes: osteochondrosis and
spondyloarthrosis. These forms should not be confused with spondylosis. Spondylosis on
radiographs
represented by vertically directed growths of the vertebral bodies, mainly due to
calcification of the fibrous rings of the discs and the anterior longitudinal ligament, which
promotes fixation
spine in the elderly. Spinal osteochondrosis is a degenerative lesion
cartilage of the intervertebral disc and reactive changes from the adjacent vertebral bodies. He
occurs with a primary lesion of the nucleus pulposus. Under the influence of unfavorable
static-dynamic loads an elastic gelatinous core, which plays a shock-absorbing role and provides
increasing flexibility of the spine begins to lose its physiological properties, primarily due to
account of depolymerization of polysaccharides. It dries up and sequesters over time. Under the
influence
mechanical loads, the fibrous ring of a disc that has lost its elasticity protrudes, and in
subsequently, fragments of the gelatinous nucleus fall out through its cracks: the protrusion is
replaced
prolapse - disc herniation. In conditions of altered, increased mobility of the spinal segment
(instability) there are reactive changes in the adjacent vertebral bodies and in the joints
(spondyloarthrosis accompanying osteochondrosis).

X-ray signs of osteochondrosis: a change in the configuration of this segment,
usually local kyphosis instead of lordosis (judged by the line of the posterior edges of the
vertebral bodies); shift adjacent
vertebral bodies, especially when unbending - the overlying vertebra is shifted posteriorly
(pseudospondylolisthesis); deformation of the end plates of the opposite vertebral bodies -
their thickening, unevenness, horizontally directed marginal growths (osteophytes); flattening
disc - a decrease in the height of the intervertebral fissure. The vertebral segment includes not
only
disc, fibrous tissue and adjacent vertebrae, but also the muscles connecting them - transverse,
interspinous and rotator cuff muscles. They are influenced by impulses from the receptors of the
affected
segment, especially from the posterior longitudinal ligament, reflexory tense. It is asymmetrical
stress causes the often occurring local scoliosis, which is indicated
radiologists as a “spreader symptom”. Reflex tension of deep (segmental) as well as
of the superficial long muscles of the spine creates a natural immobilization, often protective:

muscle corset, or collar. Over the years, the same immobilization is created due to disc fibrosis. Early fibrosis in the area of one spinal segment, functional shutdown of this link the kinematic chain of the spine leads to an overload of neighboring ones, which contributes to the development of degenerative processes.

Oblique or vertically directed growths ("beaks") - clinically insignificant

signs that characterize not osteochondrosis, but spondylosis. On the contrary, posterior osteophytes, directed into the spinal canal is a highly clinically relevant sign on the cervical level and less often - at the lumbar. In the later stages of the degenerative process, it is possible development of spondylosis and associated osteochondrosis in adjacent segments. Sometimes hernia discs are introduced not into the spinal canal, but through the hyaline plate into the vertebral body. These cartilaginous hernias of the vertebral bodies - intraspongy Schmorl's hernias, clinically asymptomatic. They are not should be mixed with lateral and median hernias of the spinal canal. Is important yellow ligament located between the arches of the adjacent vertebrae. Undergoing traumatization, she thickens, protrudes into the epidural space, putting pressure on the root.

The early development of osteochondrosis and its clinical manifestations are facilitated by some spinal anomalies: the presence of transitional lumbosacral vertebrae, lumbarization, i.e. the presence of the VI lumbar vertebra due to the upper sacral segment, or, conversely, sacralization, i.e., fusion of the distal lumbar vertebra with the sacrum; asymmetrical arrangement joint spaces of the facet (facet) joints (violation of articular tropism); splitting arcs. The congenital narrowness of the spinal canal is very important: in these Loves and small hernia, or osteophyte, puts pressure on the nerve elements.

Depending on which nerve formations have a pathological effect the affected structures of the spine, there are compression and reflex syndromes. TO compression syndrome refers to syndromes in which over the specified vertebral structures the root, vessel or spinal cord are stretched, squeezed and deformed. Kreflektorny include symptom complexes due to the influence of these structures, which innervate them receptors, mainly the endings of the recurrent spinal nerves (sinuvertebral nerve Lushki). Impulses propagating along this nerve from the affected vertebra arrive behind the root into the posterior horn of the spinal cord. Switching to the anterior horns, they cause reflex tension of the innervated muscles - reflex muscle tonic disorders.

Pathogenesis of pain caused by degenerative diseases of the spine.

All the elements of the spinal column have powerful sensory innervation; spongy part only the vertebral bodies and epidural vessels, apparently, can not be the source of pain. hypertonic saline solution to healthy volunteers in the ligaments, muscles and in the area of facet the joints of the spine leads to the appearance of intense pain in the corresponding part of the back.

Recall that facet joint syndrome as a source of pain in any part of the spine, especially in the lumbar, was clinically described in 1937. Joint blockage with an anesthetic completely on time eliminates pain and, according to some reports, sometimes leads to the restoration of lost depth side reflexes. In the experiments described, irritation of the annulus fibrosus, ligaments, facet joints, paravertebral muscles, in addition to local pain, accompanied by their

irradiation to the gluteal region, thighs, groin and along the entire length of the leg. Rigid fixation
there are no such pains to the level of the affected vertebral segment.
The mechanism of the described somatic spondylogenic pain is unclear. It is assumed that in the central nervous system
the zone receiving impulses from the vertebral segments coincides with the zone of reception from the legs (arms).
In conditions of hyperexcitability of neurons caused by painful stimulation of the spine, normal sensory flow from the limbs is perceived as pain.
The introduction into everyday life of the concept of "reflected spondylogenic pain" is in no way deactualizes the usual concept of reflected pain in diseases of the visceral organs and vessels (stomach ulcer, pancreatitis, myocardial infarction, dissecting aortic aneurysm).
It is with the exclusion of a possible connection between lumbodynia, thoracalgia and cervicalgia with the disease
organs of the abdominal and thoracic cavity, the diagnosis of acute and chronic pain in the back and
neck.
Within the framework of spondylogenic somatic pain, muscle pain should be distinguished. At them, both pains of a reflected nature and those caused by muscle contracture are possible, spasmodic impulses from the spine. Blockade of the gluteal or piriformis muscles relieves temporarily lumboischialgia, just like the blockade of the scalene muscle removes the often cruel pain in the arm. The concept of myofascial pain is increasingly used in the analysis of lumboischialgia.
R. Melzak admits the role of osteochondrosis of the spine in the occurrence of myofascial pain.
J. Hubert et al., Analyzing a group of patients with chronic refractory
“Benign” pain in the neck and back, detection of
in patients in almost all cases of several trigger points.
The relative frequency of the somatic and radicular components in the total mass lumboischialgia and cervicobrachialgia have not yet been established, since, following the Lasegue symptom,

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who have lost their specificity of the radicular sign, there is a doubt whether all the defects of the vigor and deep reflexes are always associated with the interest of the roots
(“Non-dermatomal sensory disorders”).
The value of radicular compression in the genesis of vertebral pain and other sensitive disorders are often overestimated. Typical acute radicular pain associated with compression, judging by
according to experiments during laminectomy, it has a shooting short-term character. But development of more persistent pain is possible, due in these cases to ischemia, edema, causing stimulating the nociceptors of the spinal roots.
Lumbar syndromes. Lumbar compression syndromes. Hernia compression
any lumbar root can undergo a disc. However, the early
wear of disks L IV - V and Lv-Si. Therefore, the L5 and S1 roots are most often compressed. V epidural space due to the Liv-v paramedian hernia, the L5 root is affected, and due to hernia Lv-Si - root S1. These are the most common variants of radicular compression syndromes.
If the hernia spreads laterally, it compresses the root in
the intervertebral foramen at the Liv-v level - the L4 root, at the Lv-Si level - the L5 root . Big a hernia can squeeze two roots at once, pulling on the dural sac, and with it the neighboring ones dural radicular cuffs. So, one hernia can manifest itself clinically bi- and
polyradicular syndromes. A similar hernia Liv-v exerts direct compression

on roots L5 and L4, Lv-Si hernia - on roots S1 and L5. In a stretched and squeezed root, edema, venous stasis, and subsequently due to trauma and autoimmune processes (tissue a prolapsed disc - autoantigen) - and aseptic inflammation. Since these processes are deployed in epidural space, aseptic adhesive epiduritis develops here.
Clinical manifestations of root compression: shooting pains, dermatomal hypalgesia, peripheral paresis, weakening or loss of a deep reflex.
Only the detection of one of these signs or their combination allows you to diagnose participation of the radicular component in the picture of lumbar ischialgia and cervicobrachialgia along with
spondylogenous and muscle-fascial pain. Symptom is missing from the list
Lasega, which for many years was considered a classic indicator of radicular lesions. This point view turned out to be erroneous. Lasegue's symptom can undoubtedly arise as a direct consequence
defeat
roots,
for example
at
acute
and
chronic
inflammatory
demyelinating polyradiculoneuropathy. However, within the framework of lumboischialgia, the Lasegue symptom
allows you to differentiate this symptom complex from other leg pains (thrombophlebitis, coxarthrosis), but is not a sign of complicity in the painful complex of radicular compression.
Radicular pain increases with coughing, sneezing due to reflex muscle tension
the lower back and due to the resulting cerebrospinal fluid impulse with an impact on the root, with
reception of Kwekenstedt. The pain is aggravated in the lower back during movement, especially when the body is tilted.
vista, that is, when the anterior parts of the vertebral bodies come together, which aggravates the displacement of the disc.
Upper lumbar roots L1, L2, L3 (discs L I -L P , Lp-Lsh and Lsh-Liv). Relatively rare localization. Herniated disc Li-Lp also affects the cerebral cone. Onset of radicular syndrome is manifested by pain and loss of sensitivity in the corresponding dermatomes, and more often - by
the skin of the inner and front thighs. Symptoms appear early with median hernias lesions of the cauda equina. As a rule, symptoms of the lower lumbar
radicular damage as a result of tension of the hard shell of the spinal cord of the upper lumbar hernia. In old age, cruralgia with paresthesias occurs in a wide area above and below knee due to compression of the upper lumbar roots. Determine weakness, hypotrophy and hypotonia of the quadriceps femoris muscle, lowering or loss of the knee reflex and disorders sensitivity. Compression of the L1 and L3 roots can cause lateral symptoms.
cutaneous nerve of the thigh, however, the discogenic genesis of Roth's disease is very rare.
L4 spine (Lп-Liv disc). Infrequent localization; there is a sharp pain that radiates along the inner anterior thighs, sometimes up to the knee and slightly below.
In the same area, there are paresthesias; movement disorders are manifested almost only in quadriceps muscle; unsharp weakness and hypotrophy with a decrease or absence of the knee reflex.
L5 spine (Liv-Lv disc). Frequent localization. L5 root is compressed by hernias disc Liv-Lv usually after a prolonged period of lumbar lumbago, and the picture radicular lesion is very severe. During this long time, the gelatinous nucleus

manages to break through the annulus fibrosus, and often the posterior longitudinal ligament. The pain radiates from

lower back to the buttock, along the outer edge of the thigh, along the antero-outer surface of the lower leg to
the inner edge of the foot and thumbs, often up to only one toe; the patient is experiencing tingling sensation, chilliness. Pain from the "hernial point" can also be given here, when causing the phenomenon of the intervertebral foramen, with coughing and sneezing. In the same area, especially in the distal
parts of the dermatome, hypalgesia is revealed. Determined the decrease in the strength of the extensor of the first finger
(muscle innervated only by the L5 root) , hypotension and hypotrophy of the anterior tibial muscles. The patient has difficulty standing on the heel with the foot extended.
S1 spine (Lv-Si disc). Frequent localization. Since a herniated disc does not last long
a narrow and thin posterior longitudinal ligament at this level, the disease often begins immediately with
radicular pathology. The period of lumbago and lumbodynia, if it precedes radicular pain, sometimes short. Pain radiates from the buttocks or from the lower back and buttocks along the outer-posterior edge
thigh, along the outer edge of the lower leg to the outer edge of the foot and the last toes, sometimes only up to V
finger. Often the pain extends only to the heel, more to its outer edge. In the same zones
only occasionally does the patient experience a tingling sensation and other paresthesias. This can also
to give pain from the “hernial point”, when causing the phenomenon of the intervertebral foramen (when coughing
and sneezing). In the same area, especially in the distal dermatome, hypalgesia is determined.
Determined by a decrease in the strength of the triceps muscle of the leg and flexors of the toes (especially
flexor of the V finger), hypotension and hypotrophy of the gastrocnemius muscle. The patient has difficulty
when standing on toes, there is a decrease or absence of the Achilles reflex.
When the S1 root is compressed, scoliosis is observed, more often heterolateral - trunk tilt to the sore side (which reduces the tension of the relatively short root over the hernia). At compression of the L5 root scoliosis is often homolateral (which increases the height of the corresponding
intervertebral foramen). The direction of scoliosis is also determined by the localization of the hernia:
lateral hernias, as a rule, homolateral scoliosis is noted, with medial -
heterolateral.
The conventionality of the topical significance of the directionality of antalgic scoliosis is demonstrative.
with the so-called alternating scoliosis, when during the day the scoliosis changes several times your "sign".
An extremely unfavorable variant of lumbar vertebral compression
radicular symptom complex is compression of the cauda equina. It occurs with median hernias that exert pressure not in the lateral sections of the epidural zone, where in each segment runs along one radicular nerve, and medially, where it is compact in the dural sac
the roots of the cauda equina are located. The pain is usually severe, extending to both legs, and loss of sensitivity like "rider pants" captures the anogenital zone. How
usually pelvic disorders occur.

The clinical manifestations of compression at the lower lumbar level include syndromes ischemia of the spinal cord. With an acute development of the process, they talk about spinal strokes, with
subacute and chronic - about myelopathy due to (pressure of the radicular arteries. Compression radicular-spinal artery L5 and S1 is reduced to the following. In a patient experiencing
pain in the leg and lower back, weakness in the foot appears. At the same time, sensitivity disorders are not
arises. These disorders appear to be associated with ischemia of the anterior horns of the spinal cord.
and is defined as paralyzing (paralyzing) sciatica. More extensive areas may be affected spinal cord - the cerebral cone, epicone and thoracic spinal cord, causing the development severe paralysis and sensory disorders in the legs and lower parts of the trunk, and
pelvic disorders.
The course of radiculomyeloischemia is usually two-stage. Initially due to irritation disc herniation of the posterior longitudinal ligament receptors causes lumbar pain. A bout of these pains
can be repeated in the future, and then (in one of the exacerbations) develop stroke-like paresis of other spinal disorders.
Lumbar reflex syndromes. Irritation of the annulus fibrosus receptors
the affected disc or the posterior longitudinal, interspinous and other ligaments, as well as joint capsules,
as already mentioned, it becomes a source of not only pain, but also reflex reactions. It's in first of all, tonic tension of the lumbar muscles. Lumbar vertebral syndrome
pain is defined as lumbago in acute development of the disease and as lumbodynia in subacute or chronic development.
Lumbago. Lumbago often occurs during physical exertion or awkward
movement, and sometimes for no apparent reason. Suddenly or within minutes or hours

there is a sharp pain, often shooting ("lumbago"). Often the pain is burning, bursting (“As if a stake had been stuck in the lower back”). The patient freezes in an uncomfortable position, cannot
straighten if the attack occurred at the time of lifting the weight. Trying to get out of bed turn, cough, sneeze, bend the leg are accompanied by a sharp increase in lower back pain or in the sacrum. If the patient is asked to stand up, a sharp immobility of the entire lumbar region, while there is a flattening of the lumbar lordosis or kyphosis, often with scoliosis. The lumbar spine remains fixed (natural immobilization)
and when trying to passively shift the leg in the hip joint, therefore, carefully performed flexion of the leg extended at the knee joint, with lumbago, is not always accompanied by pain in lower back: the affected disc is well protected in the immobilized vertebral segment.
Lumbodynia. Lumbodynia can also occur due to awkward movement, prolonged
tension, cooling, but not acutely, but for several days. Aching pain, intensifying
when moving, in the position of the patient standing or sitting, and especially when moving from one position to
other. Intense palpation reveals tenderness of spinous processes or interspinous
ligaments at the level of the affected vertebral segment. When the patient is in a prone position with good
relaxed psoas muscles; strong palpation of the facet joint area (at a distance
2-2.5 cm from the interspinous space) often reveals the soreness of the corresponding articular capsules. The lumbar spine can be deformed, as with lumbago, but to a lesser extent.
Movements in this part of the spine are possible, which provides the conditions for a detailed assessment

the condition of the lumbar muscles, especially the multifidus muscles. Turning off and becoming
soft when the body is tilted back, they sharply tense, keeping the body from falling,
leaning forward. With such an inclination in the range of 15-20 °, a sharp tension is normally noted
multiparticulate muscles. They are visually defined as two paravertebral shafts with a thickness of
finger, and palpation - like cords of stony density. With a further tilt of the body forward
(more than 15-20 °) the superficial lumbar muscles of a healthy person are turned off. With lumbodynia,
as with other vertebrogenic syndromes, the shutdown of this muscle tension is delayed
on one or both sides.

Sciatica. Lumbonschialgia - pain and reflex manifestations caused by
osteochondrosis, spreading from the lumbar to the gluteal region and leg. Source
pain impulses are receptors of the annulus fibrosus, posterior longitudinal ligament, facet
joint and other ligaments and muscle formations. Irradiation of pain sensations does not occur according to
dermatomes, and sclerotomes. Pain is felt in the buttock, in the posterior-outer parts of the leg, not
reaching your fingers! As with lumbodynia, they increase with a change in body, arrival and
prolonged sitting, coughing, sneezing. When feeling
the areas of the lumbar spine and leg tissues mentioned above, painful areas are found. They
localized at such bony prominences as the superior posterior iliac spine, inner edge
greater trochanter, head of the fibula. Often painful areas of the triceps muscle of the lower leg in
popliteal fossa. Along with this, painful nodules in the muscles themselves are found. These nodules
often cause reflected pain, acting as trigger points. Thus,
a typical picture of myofascial pain develops.

A sign of stretching of the back tissues of the leg (Lasegue symptom), as already mentioned, for a long time
mistakenly associated with nerve stretching. If you raise the straightened leg of the patient lying on
back (or sitting on a chair), then at a certain angle of ascent there is pain in the lumbar
the sacral region or in the tissues of the back of the leg: in the lower leg, popliteal fossa, in the area
ischiocrural (muscles that attach to the ischial tuberosity and lower leg) or gluteal muscles.
When this symptom is caused, the leg and pelvis begin to act as one and the lumbar lordosis
straightens or turns into kyphosis. At the same time, the anterior lumbar regions come closer
vertebrae and in the affected vertebral segment, the tissues behind the displaced disc are injured. So
explains that variant of the symptom when the patient indicates the appearance of pain in the lumbar
sacral area. However, the fixation of the leg and pelvis as a whole is by no means passive.
closure of the joint. Lifting the leg stretches the ischiocural muscles behind the thigh
bones. They are thrown over the hip and knee joints, so they turn out
"Short", when stretching is required until full extension in the knee joint and flexion in
hip. This movement includes the lumbar spine - it bends and due to
the tonic reaction of his flexor - the iliopsoas muscle. Simultaneously tense up and
the gluteal muscles (the pelvis rises), as well as the rectus abdominis muscles.
By the same mechanisms, pain appears in the popliteal fossa with forced pressure on
knee-patient, lying on his back, with passive extension of the foot.

A sign of stretching the iliopsoas muscle (Wasserman symptom, “reverse
Lasegue symptom ") was mistakenly associated with a stretching of the femoral nerve: the appearance of pain below
groin area with passive lifting of the patient's leg, lying on his stomach. The same pain appears with passive flexion of the leg at the knee joint (Matskevich's technique); while the pelvis
rises.
No matter how informative with lumboischialgia symptoms of tissue stretching and their soreness,
when establishing a diagnosis, especially in export difficult cases, it is necessary to exclude aggravation of tension symptoms. Incomparably more valuable for these purposes are muscle-tonic symptoms, for example, persistent tension of the multifidus muscles after tilt of the body forward by 20 ° or more. Particularly important is the symptom of homolateral tension of the multifidus muscle. Normally, this muscle relaxes when standing on one leg. on the homolateral and sharply strained on the heterolateral side. With lumboischialgia, relaxation
There is no lingering on the homolateral side - the muscle always remains tense.
Reflex manifestations of lumboischialgia extend not only to muscle and
fibrous, but also on vascular tissues. Vasomotor disorders can cause subjective (feeling of chilliness, fever) and objective symptoms (impaired blood circulation, change color and temperature of the skin of the legs, etc.).
Cervical syndromes. The clinical assessment should consider some significant anatomical features of the cervical spine in comparison with the lumbar.
First, Ci and Cn differ significantly from the rest of the vertebrae. They connect without through the disk, rotational movements prevail here. Significant clinical significance have anomalies of the craniovertebral junction. For example, when the C wave is high (axis, axial vertebra), its apex protrudes into the foramen magnum above the plane of this holes, due to which the brain stem here bends over the tooth, stretches. At unfavorable circumstances (hypermobility, ischemia), the occurrence of stem, spinal, radicular disorders.
Secondly, the transverse processes of the cervical vertebrae have transverse holes, through which runs the vertebral artery. The anterior and posterior tubercles of these processes are well are defined in lateral projection and, therefore, it is easy to determine the course of the vertebral arteries.
Thirdly, the adjacent bodies of the SS and the vertebrae located below are not completely separated from each other.
disk from a friend. In the posterolateral parts of the vertebral bodies, they are extended upward in the form of crescents -
lunate or hook-shaped processes (processusuncinatus). They touch the bodies of the lying above the vertebrae, forming the so-called uncovertebral joints. Sideways to these joints the vertebral artery is adjacent, and in front they limit the intervertebral foramen.
On the radiograph in direct projection with uncovertebral arthrosis, it is well defined structures that can act on the vertebral artery. On the radiograph in oblique projection you can see the extent to which uncovertebral growths, located in front of the root, su-
live the intervertebral foramen. Axes of the intervertebral foramen (canals) at the cervical level are located not frontally, as on the lumbar, but obliquely. Therefore, the indicated holes in the pictures in
lateral projection cannot be detected. In the picture in the lateral projection, the con-
figuration of the entire cervical spine; straightening of lordosis or kyphosis at the affected level; the change

disc heights and reactive changes in the bodies of adjacent vertebrae; changes in articular processes
with arthrosis, subluxation; sagittal canal diameter, which should normally be at least 14 mm. Thus, on plain radiographs (direct and lateral), as well as on the image in oblique projections can assess the state of almost all parts of the spine.

Radicular compression syndromes. Since early wear and tear are often lower cervical discs, in the corresponding vertebral segments concomitant spondyloarthrosis and, which is especially important, uncovertebral arthrosis. Corresponding bone
growths narrow the intervertebral foramen, therefore, at the cervical level, the roots more often are not compressed due to a herniated disc in the epidural space, as is the case in the lumbar department, but in the intervertebral foramen itself. With movements in the neck, uncovertebral growths
injure the root and its membranes, and the developing edema in them turns relative narrowness the intervertebral foramen (canal) into the absolute. There is swelling of the squeezed root, in it reactive aseptic inflammation develops.

The clinical picture of damage to the cervical roots is as follows.

C3 root (disc and intervertebral foramen Cp-Ssh). Rare localization. Pain in accordance the next half of the neck, a sensation of swelling of the tongue [connection with the hypoglossal nerve through
hypoglossal loop], hypalgesia in the dermatome corresponding to the C3 root.

C4 root (disc and intervertebral foramen Csh-Civ). Rare localization. Pain in the area shoulder girdle, clavicle, atrophy of the back muscles of the neck (trapezius, belt, lifting the scapula,
the longest muscles of the head and neck). A decrease in the tone of these muscles and, as a result, an increase
air cushion at the apex of the lung. With symptoms of irritation of the roots C3-C4 an increase in the tone of the diaphragm leads to a downward shift of the liver; possible pain that mimics
angina pectoris. With the phenomena of prolapse, the diaphragm relaxes.

C5 root (disc and intervertebral foramen Civ-Cv). Relatively infrequent localization. The pain radiates from the neck to the shoulder girdle and to the outer surface of the shoulder; weakness and malnutrition
deltoid muscle.

The most common damage to the roots of C6 and C7. C6 back (disk and
intervertebral foramen Cv-Cvi). Pain extending from the neck and scapula to the shoulder girdle, along
the outer surface of the shoulder, to the radial edge of the forearm and to the 1st finger, paresthesia in the distal
departments of this zone. All these subjective phenomena are amplified or provoked by the induction of fe-
nomen of the intervertebral foramen or during voluntary head movements, hypalgesia is noted in the dermatome corresponding to the C6 root, weakness and hypotrophy of the biceps muscle, decrease or
lack of reflex from the tendon of this muscle.

C7 root (disc and intervertebral foramen Cvi-Cvp). Pain radiating from the neck and scapula on the outer-posterior surface of the forearm to fingers II and III, paresthesia in the distal part

this zone, hypalgesia in the C7 root zone, weakness and hypotrophy of the triceps muscle, decrease or
lack of reflex from the tendon of this muscle.
C8 root (Cvp-Thi disc and intervertebral foramen). Pain that spreads from the neck to the ulnar edge of the forearm and to the V finger, paresthesia in the distal parts of this zone. Hypalgesia in
zone of the C8 root, a decrease or loss of the styloradial and instep support reflexes.
Cervical reflex syndromes (cervicobrachialgia). Just like at the lumbar level,
the main vertebral syndrome manifests itself in the form of lumbago - acute neck pain, or churchcalgia, subacute or chronic neck pain. The source of pain impulses are
receptors of the affected fibrous tissues (annulus fibrosus, posterior longitudinal ligament, joint capsules, etc.), as well as tonically strained cervical muscles. Discogenic scoliosis (torticollis) is not as pronounced here as at the lumbar level. Pains wear aching, brawny character, often radiate to the back of the head and shoulder girdle. They increase with movements in the neck or,
on the contrary, with its prolonged monotonous position (in the cinema, in front of the TV screen, after
prolonged sleep, especially on a dense and high pillow). With deep palpation
soreness of individual spinous processes is found. Symptoms are even more common spondyloperiarthrosis - soreness of the capsules of the facet joints on the affected side. For their feeling the head is tilted to the sore side, with the left hand fix the opposite
the frontal tubercle, and the fingertips of the hands exert strong pressure on the joints of the affected side.
These capsules are felt through the thickness of the relaxed cervical muscles along the back of the neck
at a distance of 3-4 cm from the spinous processes. A very characteristic feature of reflex tonic reactions of this level is a significant involvement of muscles located not
only behind, but also in front of the spine. Among them, a special place is occupied by the front staircase.
muscle, as well as muscle that attaches to the upper medial angle of the scapula and lifts it when shortening.
Breast syndromes. Due to the fact that the thoracic spine (as opposed to the lumbar
and cervical) is inactive, there are no conditions for significant macro- and microtraumatization of the discs.
Herniated thoracic discs with root and spinal cord compression are extremely rare.
It is pertinent to recall that, unlike the skin of the trunk, which receives innervation from the thoracic roots,
the muscles of the shoulder girdle, and in particular the shoulder blades, are innervated from the cervical region and, as a rule,
acute and chronic pain in this area is of cervical origin. However, the reflected
spondylogenic chest pains are very real. A similar model of imitation of radicular spondylogenic pain is clearly visible in the clinic of ankylosing spondylitis, where pain in the chest
department - a daily complaint, and compression of the roots - casuistry, contrary to the prevailing representation
niyam. Chest pain due to degenerative lesions of the thoracic spine
usually caused by damage to the costo-vertebral and costo-transverse joints and their capsules.

On radiographs, costovertebral or costo-transverse arthrosis is detected.
On palpation of the costal-vertebral joints (at a distance of 1/2 fingers from the spinous processes), their sharp soreness is found. In a number of patients, painful

nodules in the pectoral and abdominal muscles. Spontaneous pain in these areas can mimic primary
visceral diseases.
Treatment. At all stages - individually selected analgesics, baclofen; in protracted cases, amitriptyline and (or) tranquilizers are used.
Due to the fact that we are talking about diseases caused by degenerative damage spine, the first task is to influence the affected motor segment and the spine as a whole. The spine in the conditions of its pathology with any movements traumatizes as
own structures and neighboring nerve formations, therefore, plays an important role in treatment unloading the affected segment of the spine - ensuring rest.
In the acute period, the patient should be laid on a non-bending bed; under a soft mattress you need to put a shield. The patient can move either with assistance or on crutches.
You should sit with the palms of outstretched hands on the seat. Any change in body position should not be done automatically (as we are used to), but carefully after preparation.
The relative rest of the vertebral segment is created by the “muscle corset”. It should be save, avoiding in the acute period procedures that relax the lumbar muscles, such as local heat, hot bath, etc.
Some clinicians rule out any other orthopedic and physiotherapy impact. The patient lies on the shield throughout the day. If after 2 weeks the pain disappears, then
the patient is allowed to walk around the ward in a corset for another 2 weeks. After that, depending on
the results of treatment, or the corset is removed and the patient is discharged, or rest is prescribed for another 7-14
days. A good effect is noted often, but still no more than 70% of cases.
The second (after passive rest) therapeutic factor, which is at the same time a means of prevention of exacerbation is unloading due to stretching - traction of the affected vertebral segment. The effect of traction is not only to reduce the mechanical the influence of a herniated disc, but also in a change in impulses coming from the muscle tissues of the vertebral
segment, and hence in a change in reflex relations, since afferentation from tense the muscles of the spine is the beginning (part) of the pathological reflex process.
Stretching is especially indicated in the early stages of the disease, primarily when there are symptoms of compression of the root by a herniated disc. If stretching is ineffective and radicular symptoms intensify, one can think of a large and motionless insertion nasal hernia in the canal; in this case, the procedure must be terminated.
The third (after passive rest and traction) factor of immobilization of the affected the spinal segment is to strengthen its "muscle corset" due to physical exercise. Since this should not violate the principle of rest of the vertebral segment, the latter during the procedure should not deform. Therefore, the exercises at first should be to carry out in a prone position. At the same time, it is enough to make active movements in the hands, in
ankle and knee joints so that the lumbar muscles engage in order friendly (synkinetic) activity. Immobilizing measures include removable corset, which is rarely used - such immobilization is simultaneously demobilization of the natural "muscle corset".
If in the first weeks of the disease in the presence of radicular compression is required immobility of the affected vertebral segment, in the future, when the edema of the root decreases and
the latter will adapt to new spatial conditions, a sharp tension in the lumbar muscles are no longer protective. This tension maintains the disturbed posture.

of the spine, is the cause of pain that does not contain a useful signaling load. At this stage blocking and other pain-relieving procedures should be started.
Due to the fact that one of the symptoms of vertebral diseases is compression nervous formations, the second task of treatment is to influence these formations, elimination of stagnant phenomena in them, and subsequently - aseptic-inflammatory changes. it achieved by the appointment of decongestants and desensitizing agents. Very large doses vitamin B12 (3000-5000 mcg per injection) have an analgesic effect.
The analgesic effect is exerted in the acute period by diadynamic and sinusoidal modulated currents. Prescribe novocaine electrophoresis, electrophoresis and phonophoresis of non-steroidal and steroid anti-inflammatory drugs, UHF therapy, microwave treatment. Powerful and the most important means are massage and physiotherapy exercises. In a sanatorium-resort environment

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add radon, sulfide, sodium chloride or iodine-bromine baths. Mud applications are an important curative factor, which, however, can cause exacerbation. Prevent it can be strictly taken into account the indications ("cold period" of the disease) and a combination of mud therapy with the use of dehydrating therapy.
The fourth goal of treatment is to influence the disease-related spine pathological reflex processes - muscle-tonic and myofascial.
Muscle-tonic disorders outside the spine rarely carry a protective load. Voltage muscles of the shoulder girdle, gluteal region, arms or legs should be eliminated by muscle infiltration novocaine - blockades, which often causes a positive shift already during the first procedure. General massage, gentle therapeutic gymnastics without sharp bends of the trunk, and in position lying down - with legs bent at the knees - all these measures are also prevention of exacerbations.
Swimming and other non-fatiguing exercises help to strengthen the cervical muscles. "Collar" and lumbar "corset". Rest in the acute period is required for the affected segment, zone of disc herniation, but by no means for the muscles of the lower leg or forearm. Every day physical exercises are carried out more and more intensively, first in a lying position, and then standing. V conditions of often prolonged pain syndrome, psychotropic drugs are widely used paratha and, in particular, amitriptyline; the latter also has a clear analgesic effect. For for the treatment of patients with vertebral neurological syndromes, manual therapy is widely used.
Manual therapy is a separate method of treatment that is used by the main way for diseases of the musculoskeletal system. At first, it was aimed at elimination of functional blockages of the vertebral and other joints resulting from infringement of intra-articular cartilaginous meniscoids. Then manual therapy began to be used to achieve muscle relaxation, relieve muscle contractures like blocked joints, and adjacent to them. Such effects on the spine lead to the transformation of a common myofixation to the local. The main techniques of manual therapy are relaxation, fixation, mobilization tion, manipulation. Relaxation of spasmodic muscles is achieved by various types of massage (classic, point, segmental, canned, etc.), as well as post-isometric relaxation (PIR). PIR is reduced to muscle relaxation after their volitional tension without changing the distance between the points of muscle attachment (i.e. after isometric tension).

In cases where complex conservative treatment does not relieve within 3-4 months intense pain, there are indications for surgical treatment if CT, MRI or myelography reveals a herniated disc. The uniqueness of the situation with uncomplicated discogenic pain syndromes is that the question of surgical treatment depends to a large extent on the patient (the patient himself must "crawl" to the neurosurgeon). Detection in neuroimaging a herniated disc in itself is not an indication for the abolition of adequate conservative treatment; even under the influence of a hernia on the dural sac, spontaneous and therapeutic remission of pain syndrome. The role of the doctor is to promptly advise a neurosurgeon. An absolute indication for urgent surgery is only an acute development of the picture (pressure cauda equina with motor, sensory and sphincter disorders or acute or subacute spinal cord compression. Surgical treatment is the treatment of choice for spondylogenic cervical myelopathy. It should only be borne in mind that long-term ischemia, despite decompression, causes a relatively moderate effect of operations in comparison, for example, with the removal of a hernia, which can lead to a pronounced regression of paresis. Serious contraindications for decompression in myelopathy are old age and severe accompanying illnesses.

The examination of temporary disability is carried out taking into account the need for bed regime in the first days of any exacerbation. With lumbago, sometimes 6-10 days are enough, after which, if there are no subjective and objective residual effects, the patient can close the sick leave. Exacerbations and with this syndrome, if they are repeated often (several times a year), are a signal to transfer the patient to another job (lighter), if he is busy with heavy physical labor. For persons of sedentary professions, frequent exacerbations are a signal for activation preventive measures and to lengthen the period of stay on sick leave during the period exacerbation. In the presence of lumboischialgic syndromes, stay on sick leave, as as a rule, it is impossible to limit 1-2 weeks - early (up to 3 weeks) discharge from work often entails more frequent exacerbations.

Patients with a severe course of the disease, with persistent pain and other manifestations recognized as temporarily disabled for a period of up to 4 months. If after that it is not determined the prospect of returning to work in the near future, the patient is sent to VTEK to solve

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the question of permanent incapacity for work - disability. Usually after 1-2 years disability withdrawn - the patient manages to return to previous work or to work in lightened conditions.

TUMORS OF THE NERVOUS SYSTEM

HISTOLOGICAL CLASSIFICATION

The basis for most of the available classifications of CNS tumors was based on the histogenetic principle of classification by Baily and Cushing (1926); in the USSR the most common was a modification by L.I.Smirnov (1951) and B.S.Khominsky (1962). It was assumed that the cellular composition neuroectodermal tumors (actually brain tumors) reflect a particular stage of development various cells of mature nervous tissue; the name of the tumor is established by an embryonic element that most closely resembles the bulk of tumor cells; degree malignancy is determined by the severity of cell anaplasia, the nature of growth (invasive, non-invasive) and other biological characteristics of the tumor.

The existing terminological inconsistency between different classifications became one of the main motivating reasons for the development in 1976 of the International (WHO) histological

classification of tumors of the central nervous system.
However, in 1993 WHO adopted a new histological classification of CNS tumors. The basis for the changes made were the results of many years of research by morphologists in the field of in-depth studying the histogenesis of tumors, cytoarchitectonics and biochemistry of tumor cells, factors and their kinetics
growth. To solve these problems, various modern techniques were used, among which a particularly important place was taken by immunohistochemical and ultrastructural immunocytochemical studies.
Some of the tumors more accurately found their place in the classification, built like the previous ones, on the histogenetic principle; a number of terminological inaccuracies were eliminated. Excluded from classification of tumors of the central nervous system section with a list of vascular malformations.
Much attention was paid to the study of the factors of "aggressive" growth of some tumors and their the tendency to relapse after surgical treatment.
As a result, the authors of the new classification considered it expedient to abandon the proposed in WHO classification (1976) the principle of determining the degree of tumor malignancy by life span patients after "radical" surgery. It was proposed to evaluate in detail such signs as atypia of the nuclei, cellular polymorphism, mitotic activity, endothelial or vascular proliferation, and
the presence of necrosis - in direct proportion to the number of signs present and the degree of malignancy of each specific tumor.

INTERNATIONAL
(WHO)
HISTOLOGICAL
CLASSIFICATION
TUMORS
CENTRAL NERVOUS SYSTEM (1993)

I. Tumors of neuroepithelial tissue

A. *Astrospinal tumors*
1.
Astrocytoma:
fibrillar,
protoplasmic, mixed
2. Anaplastic (malignant) astrocytoma
3. Glioblastoma: giant cell glioblastoma,
gliosarcoma
4. Piloid astrocytoma
5. Pleomorphic xanthoastrocytoma
6.
Subependymar
giant cell
astrocytoma (usually combined with tuberous
sclerosis)
B. Oligodendroglial tumors
1. Oligodendroglioma
2.Anaplastic
(malignant)
oligodendroglioma
B. Tumors of ependyma
1. Ependymoma: dense cell, papillary,
epithelial, clear cell, mixed
2. Anaplastic (malignant) ependymoma
3. Myxopapillary ependymoma
4. Subependymoma
D. Mixed gliomas
1. Mixed oligoastrocytoma
2.
Anaplastic
(malignant)
oligoastrocytoma

III. Tumors of the meninges

A.
Tumors

outgoing
from
meningothelial cells of the meninges
1. Meningioma: meningotheliomatous,
mixed,
fibrous,
psammomatous,
angiomatous,
metaplastic
(xanthomatous, ossified, cartilaginous
and others) and others.
2. Atypical meningioma
3.
Anaplastic
(malignant)
meningioma
a) with options
b) papillary
B. Non-meningeal tumors of the meninges
1. Mesenchymal tumors
1) benign tumors
a) osteochondral tumors
b) lipoma
c) fibrous histiocytoma
2) malignant tumors
a) hemangiopericytoma
b) chondrosarcoma
c) mesenchymal chondrosarcoma
d) malignant fibrous histiocytoma
e) rhabdomyosarcoma
f) sarcomatosis of the membranes
3) primary melanic cell lesions

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3. Other tumors
D. *Tumors of the choroid plexus*
1. Papilloma of the choroid plexus
2. Choroid plexus carcinoma
E.
Neuroepithelial
tumors
undefined origin
1. Astroblastoma
2. Polar spongioblastoma
3. Gliomatosis of the brain
G. Neuronal and mixed neuronal
glial tumors
1. Gangliocytoma
2. Dysplastic gangliocytoma of the cerebellum
3.
Desmoplastic
infantile
ganglioglioma
4. Dysembryoplastic neuroepithelial
tumor
5. Ganglioglioma
6.
Anaplastic
(malignant)

ganglioglioma
7. Central neurocytoma
eight.
Olfactory neuroblastoma
-
esthesioneuroblastoma
(option:
olfactory neuroepithelioma)
3. Pineal tumors
1. Pineocytoma
2. Pineoblastoma
3. Mixed pineocytoma-pineoblastoma
I. Embryonic tumors
1. Medulloepithelioma
2.
Neuroblastoma
(option:
ganglioneuroblastoma)
3. Ependymoblastoma
4. Retinoblastoma
5. Primitive neuroectodermal tumors
(PNETs)
with
polymorphism
cellular
differentiation:
neuronal,
astrocytic, ependymal, etc.
a)
medulloblastoma
(options:
medullomyoblastoma,
melanic cell medulloblastoma)
b)
cerebral or spinal PNETs
II. Tumors of the cranial and spinal nerves
1. Schwannoma (neurilemmoma, neurinoma):
dense cell,
plexiform,
melanotic
2. Neurofibroma: nodular, plexiform
3.
Malignant
tumor
shells
peripheral
nerves
(neurogenic
sarcoma, anaplastic neurofibroma,
"Malignant schwannoma")
a) diffuse melanosis
b) melanocytoma
c) malignant melanoma (including
melanomatosis of the membranes)
2. Tumors of undetermined histogenesis
a)
hemangioblastoma
(capillary
hemangioblastoma, angioreticuloma)

IV. Lymphomas and tumors of the hematopoietic tissue
1. Primary malignant lymphomas
2. Plasmacytoma
3. Granulecytic sarcoma
4. Others
V. Germ cell tumors
1. Herminoma
2. Embryonic carcinoma
3. Tumor of the yolk sac (tumor
epidermal sinus)
4. Choriocarcinoma
5. Teratoma: mature, immature, malignant
6. Mixed tumors
Vi. Cysts and tumor-like processes
1. Rathke's pocket cyst
2. Epidermoid cyst (cholesteatoma)
3. Dermoid cyst
4. Colloid cyst of the III ventricle
5. Enterogenous cyst
6. Neuroglial cyst
7. Granular cell tumor (choristoma,
pituicitoma)
8. Neuronal gamartoma of the hypothalamus
9. Nasal glial heterotopy
10. Plasma cell granuloma
Vii. Tumors of the Turkish saddle area
1. Pituitary adenoma
2. Carcinoma of the pituitary gland
3. Craniopharyngioma
VIII. Germination of tumors from nearby fabrics
1. Paraganglioma (chemodectoma, tumor
jugular glomus)
2. Chordoma
3 Chondroma (including chondrosarcoma)
4.
Carcinoma
(nasopharyngeal scaly cell
carcinoma, adenoid cystic carcinoma)
IX. Metastatic tumors
X. Unclassified swelling

TUMORS OF THE BRAIN AND ADJACENT TUMORS
There are no exact data on the frequency of brain tumors, however, according to different authors, they

are the cause of death in 1% of cases. According to averaged data, the most common are the following brain tumors: astrocytoma-15%; oligodendroglioma - 8%; ependymoma - 3%; papilloma choroid plexus - 0.7%; glioblastoma - 15%, medulloblastoma - 4%; meningioma - 15%; angioblastoma - 2%; pituitary adenoma –7-10%; neuroma - 7-8%; congenital tumors, 2%; metastases -8%. For a simplified description of the clinical picture, features of diagnosis and treatment, use various classification schemes that form relatively homogeneous groups of tumors. So, supratentorial (tumors of the cerebral hemispheres and tumors of the base of the anterior and middle cranial fossae) and subtentorial, i.e., tumors located under the tentorial tentorium (tumors cerebellum, brain stem, IV ventricle, VIII nerve, base of the posterior cranial fossa). Perhaps suprasubtentorial location of the tumor, for example, when the trigeminal neuroma penetrates through the tentorial foramen from the middle to the posterior cranial fossa; with growth in both directions meningiomytentorial markings. When the tumor spreads through the foramen magnum into a spinal canal (or vice versa) speak of a craniospinal tumor. Supratentorial tumors

divided into basal and hemispheric (convexital and deep); the same scheme can be applied to tumors of the posterior cranial fossa.
The division of tumors into extra-and intracerebral is of great importance. It haunts practically important goal: most extracerebral tumors can be radically removed (meningiomas, neuromas, pituitary adenomas), and intracerebral (gliomas) are usually characterized by severe infiltrative growth and in many cases radically inoperable. However, some tumors that relying in the deep parts of the brain, are available for radical surgery, in particular, some intraventricular neoplasms.
Sometimes tumors that are particularly difficult for surgical treatment are assigned to a special group. midline: gliomas of the corpus callosum, tumors of the transparent septum, region of the third ventricle, pineal gland, brain stem. These tumors can have both neuroepithelial and other origin.
There are other classification schemes that will be considered when describing some brain tumors.

Clinic

One of the main features of tumors of the nervous system is that they develop in a limited space of the cranial cavity and therefore sooner or later lead to damage as adjacent to the tumor, and distant from it parts of the brain.
Direct pressure or destruction due to tumor infiltration of brain tissue causes the appearance of local (primary, local, nested) symptoms. Dysfunction relative brain structures close to the tumor, resulting from edema, local disorders hemodynamics and other causes, leads to the appearance of an additional group of symptoms, the so-called symptoms "next door".
As the tumor grows, “distant” symptoms may appear, in particular symptoms intercalation of parts of the brain distant from the tumor, as well as general cerebral symptoms that develop due to diffuse cerebral edema, generalization of hemodynamic disorders, the appearance of intracranial hypertension.
This division of symptoms is very arbitrary and not always unambiguous. In particular, it can be difficult separate “local” symptoms and symptoms “in the neighborhood”, and differentiate the latter from symptoms “In the distance”. In addition, when a tumor grows in clinically relatively “silent” areas of the brain the manifestation of the disease can begin with general cerebral symptoms, and in some cases, focal symptoms may not be present at all.
However, such an approach in assessing clinical manifestations is necessary for the correct analysis of their dynamics, which allows: 1) to select from the entire symptom complex the signs necessary for establishing a topical diagnosis, and more reasonably conduct further examination, which often requires the use of invasive and dangerous diagnostic methods; 2) evaluate correctly the stage of the disease (compensation, subcompensation, decompensation) and correctly determine the indications for planned, urgent or emergency treatment of patients. Therefore, in the further description of the clinic we will adhere to the division into primary (focal) and secondary symptoms of head tumors brain.

Primary (focal) symptoms

Some of the symptoms covered in this section are not categorized properly. "Neurological", but are mentioned in connection with the fact that they are included in pathognomonic syndromes that allow to make a confident diagnosis already during an outpatient examination.
Headache. More often it is cerebral, but it can also be a focal symptom with tumors of the brain associated with the richly innervated sensory fibers of the dura mater sheath or with sensitive roots of the cranial nerves. So, with convexital meningiomas headaches, quite clearly localized in the projection of the tumor, are not uncommon. With meningiomas

cerebellar tentorium on the side of the tumor, there are characteristic shooting from back to front in orbital area headaches. With basal meningiomas located in the chiasmatic region, there may be moderate headaches of a shell nature with irradiation to the eyes, with lacrimation, photophobia. These symptoms may be more pronounced when the tumor grows in cavernous sinus. At the same time, their one-sided nature prevails and a combination with others is possible. signs of damage to the cavernous sinus - radicular dysfunction of the oculomotor nerves,

venous congestion in the fundus, less often with exophthalmos. A characteristic pain syndrome is given by neuromas and
other tumors of the trigeminal region. In this case, one-sided pain in the face area can
be combined with hypesthesia or hyperpathy, respectively, in the areas of innervation of the affected branches.
Vomit. Also a common cerebral symptom, however, "isolated" vomiting has been described.
due to irritation of the bottom of the IV ventricle by its intraventricular volumetric formations; her appearance
provoked
change
provisions
heads.
Vomit
such
origin
enters
v
symptom complex of bruns-like seizures, which are characterized by a sharp headache, skin
vegetative-vascular reactions, respiratory failure, pulse arrhythmia and forced head position, with
which improves the conditions for the outflow of cerebrospinal fluid. The reason for the development of bruns-like
seizures (in expanded or abortive form) is mainly acute occlusion of the exit from the IV
ventricle. With continuing and increasing occlusion with the development of an occipital implantation
respiratory and pulse disturbances are aggravated (bradycardia is especially unfavorable prognostically!),
there are disturbances of consciousness, a thrown back position of the head and the danger of sudden
of death.
Irritation of nuclear formations of the lower part of the bottom of the IV ventricle is the basis and other often
accompanying isolated vomiting symptom - hiccups, which can be observed in the form of lingering,
difficult to remove attacks.
Visual impairment. They can be a symptom of focal lesions of the visual pathway throughout its
stretch - from the optic nerves to the cortex of the occipital lobes. A sharp decrease in vision or blindness,
combined with exophthalmos and expansion of the optic nerve canal (determined on radiographs
orbits according to Reze), are pathognomonic for optic nerve glioma.
Chiasmal syndrome (complete or asymmetric) associated with an increase in size
sella turcica, indicates the presence of an endosuprasellar tumor, more often a pituitary adenoma or
craniopharyngiomas. Moreover, craniopharyngiomas are characterized by petrification determined on
craniogram, and the phenomenon of hypopituitarism, and for some types of adenomas (hormone-active)
the presence of a syndrome of increased production of various triple hormones of the pituitary gland is typical.
Chiasmal syndrome with normal sizes of the sella turcica can be a symptom of a basal
meningiomas
(areas
tubercle
Turkish
saddles,
supraphrenic)
or
suprasellar craniopharyngioma. For the first group of tumors, a distinctive feature may be
radiologically detected hyperostosis in the area of the tubercle and the site of the sphenoid bone, and for the second -
petrification in the tumor in combination with hormonal disorders.
Chiasmal syndrome, combined with gross destruction of the bones of the base of the skull and damage
cranial nerves, can be detected with intracranial spread of tumors of the base of the skull.
Asymmetric visual impairment in combination with destruction or hyperostosis of the wings
sphenoid bones are characteristic of meningiomas of this localization. With large meningiomas of the wings
sphenoid bone often develops Foster-Kennedy ophthalmic syndrome (disc atrophy
optic nerve on the side of the tumor and congestion on the other side); there may be a one-sided ek-
zophthalmos.
Assessment of the condition of the fundus is required if a brain tumor is suspected. So,
the appearance of even the initial signs of primary atrophy of the optic nerves makes it necessary
targeted examination of the patient in connection with a suspected tumor of the chiasmatic-sellar
localization. Assessing the rate of increase in primary optic atrophy can be very helpful.
to determine the topic of the focus: when the optic nerve is compressed, atrophy appears early (combined with

various disorders of the visual fields, more often of the bitemporal type); with tumor pressure on the tract descending atrophy of the peripheral neuron appears approximately one year after the onset visual field disorders of the type of homonymous hemianopsia. When the focus is localized along the central neuron (posterior to the geniculate body), a homonymous hemianopsia develops without atrophy of the optic disc
nerve.
Optic disc congestion and secondary atrophy will be discussed in the description of secondary symptoms of brain tumors.
Cranial nerve dysfunctions. We examined this issue within the framework of practical classes.
Symptoms of focal lesions of the cerebral hemispheres. The order of appearance, severity and the nature of these symptoms depends on a number of factors. So, certain differences in the order of appearance
are available with convexitally (more often extracerebral) and deeply located tumors. For

convexital tumors are characterized by the initial predominance of symptoms of "irritation" of the cerebral cortex
brain. For example, epileptic adjustments in meningiomas may appear several years earlier than symptoms of "loss" (paresis, sensitivity disorders). With glioma of the same localization, the opposite picture is observed. For convexitally located tumors, more selective damage to certain areas of the cerebral cortex. So, a tumor in the motor area manifested mainly by paresis without disturbances in sensitivity. With deep tumors, especially located in the area of the inner capsule, even with small sizes can extensive feature drops out.
Thus, the severity and nature of focal symptoms are largely determined by the functional role of the affected area. In addition to the above, it should be noted that early focal symptoms in tumors located in the sensorimotor zone, speech "centers", etc. Tumors the poles of the frontal lobes, the temporal lobe of the non-dominant hemisphere can be clearly manifested already
secondary symptoms, for example, it is not uncommon for a tumor in the frontal lobe to “simulate” a clinic tumors of the posterior cranial fossa and vice versa. Tumors of the lateral ventricles are usually occlusive-hypertensive-hydrocephalic syndrome without obvious local symptoms.
Secondary symptoms
Secondary symptoms of brain damage in tumors include disorders of the brain circulation, edema, intracranial hypertension, dislocation and wedging of the brain. There is a close the interconnection of these processes, often forming a “vicious circle” of cause-and-effect stages pathogenesis of brain death. For example, cerebral ischemia leads to cerebral edema, which further exacerbates insufficient blood supply to the affected area. This causes an increase in intracranial pressure and the appearance of a dislocation with possible wedging, which in turn worsens the conditions blood supply and increases edema.
Cerebral circulation disorders. The brain has a high degree of protection of its own blood supply. There are known phenomena of cerebral blood flow autoregulation, directed mainly to maintain an optimal supply of oxygen to the brain. Brain hypoxia due to "arterial" ischemia with tumors is quite rare: 1) with hypoxemia due to inadequate breathing in terminal conditions or during status epilepticus; 2) with a sharp decrease in systemic blood pressure; 3) with the restructuring of the cerebral circulation due to "robbery" arterial inflow in the perifocal zone of the tumor containing powerful arteriovenous shunts (glioblastoma multiforme), which, apparently, is one of the reasons for the frequent occurrence pronounced cerebral edema around such a tumor. The cause of ischemia and even infarction of the occipital lobe can
be compression of the posterior cerebral artery between the brain and the edge of the cerebellar tentorium with lateral
temporo-tentorial involvement of the brain.
In some cases, ischemia can be caused by tumor compression of large arterial vessels, for example, in basal meningiomas that overgrow the internal carotid artery and often narrowing her lumen. However, due to the slow growth of the tumor and large compensatory the capabilities of the cerebral arterial circle (circle of Willis) ischemia in these cases develops quite rare. In patients operated on for a tumor, the role of "arterial" ischemic

factor in the development of cerebral hypoxia increases in cases of direct damage to arterial vessels, their prolonged postoperative spasm and thrombosis.
More often, hypoxia of the brain with tumors develops as a result of impaired venous outflow.
Recall that the perfusion pressure in the organ is equal to the difference between the systemic arterial pressure and
pressure in the veins of the organ. The pressure in the veins of the brain is almost equal to the intracranial pressure in the passive
corresponds to its changes. Therefore, with an increase in intracranial pressure, the
perfusion pressure up to stopping cerebral blood flow.
At
tumors
located
near
venous
collectors
brain
(parasagittal meningiomas), apparently, it is the obstruction of venous outflow that quickly leads to hypoxia, cerebral edema and increased intracranial pressure. Damage during surgery even
small parasagittal veins can lead to gross neurological defects. The most
an early symptom of impaired venous outflow from the cranial cavity is lengthening of the venous phase blood flow detected by fundus fluorescence angiography.
The next mechanism for the development of cerebral hypoxia lies in the functional and metabolic violations at the cellular level. This includes the phenomenon of ineffective blood flow, when oxygen and glucose is not consumed by the brain tissue, for example, after a period of acute ischemia, even if arterial inflow and venous outflow were quantitatively restored.
Cerebral edema. Cerebral edema is understood as an excessive accumulation of intercellular fluids in his tissue; an increase in the volume of intracellular fluid is called swelling of the head brain. Distinguish between vasogenic, cytotoxic, osmotic and hydrostatic cerebral edema.
Cerebral edema can be limited or diffuse. There is some concurrency
between the prevalence of edema and the degree of increase in intracranial pressure. However, research

recent years have shown that even with significant edema, intracranial pressure can be low.
Neurological diagnosis of edema is very difficult. This is due to the lack of specific edema symptoms (presented in various guidelines, the clinical picture of "cerebral edema" reflects such with intracranial hypertension). This task is not easy to solve. So, in patients with severe perifocal edema around the tumor (olfactory meningioma, glioblastoma, metastasis) powerful decongestant therapy (for example, high doses of glucocorticoid hormones) leads to significant positive dynamics of local and secondary symptoms. However, it is difficult to differentiate the effect that develops by reducing edema, from the effect due to a decrease in intracranial pressure. Therefore, so far, the only way to reliably diagnose edema remains
X-ray and magnetic resonance computed tomography.
It can be assumed that the clinical picture of edema is manifested as an increase in the severity local symptoms and an increase in cerebral disorders due to edema of functionally important parts of the brain (hypothalamus, brain stem, etc.) or due to generalization of edema.
The severity of morphological changes in brain tissues (up to the death of neurons)
corresponds to the severity and duration of the existence of edema and largely determines the prognosis even with
adequate, but untimely started treatment.
Intracranial hypertension. Intracranial pressure is normal, with lumbar puncture and
the lying position is equal to 150-180 mm of water column. (11-13 mm Hg). It consists of three components: pressure of cerebrospinal, interstitial and intracellular fluids. Enhancement
intracranial pressure in brain tumors develops due to: 1) an increase in the mass of the tumor; 2) occlusion of cerebrospinal fluid with impaired outflow of cerebrospial fluid; 3) cerebral edema; 4) imbalance “production - absorption” of cerebrospinal fluid (more often
resorptive component); 5) impaired venous outflow, either "local" genesis, or due to increased central venous pressure with inadequate breathing and constant vomiting.
Defense mechanisms during an increase in pressure in the cranial cavity are presented in the order of their significance; 1) a decrease in the volume of cerebrospinal fluid due to its displacement from the ventricles and

tanks; 2) a decrease in the intracranial volume of venous blood; 3) local atrophy of brain tissue around the tumor or diffuse atrophy with ventricular hydrocephalus; 4) spontaneous wall breakout ventricles. In children, the size of the head may additionally increase due to the divergence of the seams and due to
plastic properties of the bones of the skull (this may be one of the first symptoms of intracranial hypertension in young children).
The clinical picture of intracranial hypertension with its slow increase is manifested uncertain, often "morning" headaches, often at the height of the headache is noted vomit. To the earliest symptoms of slowly progressive intracranial hypertension in adults include plethora of veins, initial edema of the optic nerve head. With fluorescent angiography lengthening of the venous phase of blood circulation (capillary and arterial phases change with a pronounced increase in pressure in the cranial cavity). Parallel or several later, radiological signs of hypertension appear: osteoporosis of the details of the sella turcica, increased pattern of digital impressions, thinning of the bones of the arch.
With the rapid or acute development of hypertension due to occlusion of the cerebrospinal fluid pains often have paroxysmal paroxysmal character, bruns-like seizures are not uncommon (with intraventricular localization of the tumor), characterized by the appearance of oculomotor disorders due to pressing the III and VI nerves to the base of the skull.
With further progression of intracranial hypertension, mental disorders appear, memory loss, changes in the fundus are increasing - a pronounced stagnation with by prominence of the optic discs into the vitreous humor, hemorrhages and white foci (secondary atrophy) in the fundus. Symptoms of advanced intracranial hypertension include the appearance of "Nubilation" (periodic darkening of vision when changing the position of the head, small physical load), which are a poor prognostic sign in terms of maintaining full vision after elimination of the causes of intracranial hypertension. With loss of vision due to secondary atrophy, paradoxical decrease or even disappearance of headache is quite often noted hypertensive nature.
The decompensation phase of intracranial hypertension ends with symptoms of progressive disturbances of consciousness (up to coma) and vital disturbances, one of the causes of which is dislocation and wedging of the brain.
Dislocations and internections of the brain. The reasons and mechanism of their appearance are partially described above.
The most common lateral temporo-tentorial and axial occipital insertion.
The incipient insertion is characterized by pain in the back of the head and neck, stiff neck muscles, forced position of the head. The growth of the interception leads to the appearance of disturbances in consciousness and
bulbar vital disorders, ending with respiratory arrest, unless an emergency help.

Diagnostics
If a brain tumor is suspected, the following diagnostic tests are performed.
1. Thorough neurological examination, including a detailed ophthalmological study of visual acuity, visual fields, fundus; an otoneurologist examines the sense of smell, taste, hearing, vestibular functions.
2. Craniography in two main projections, which, according to indications, is supplemented with images, made with special styling.
3. Electroencephalography (EEG) to identify focal symptoms of brain damage and / or assess the severity of violations of the physiological mechanisms of brain activity in general.
4. Echoencephalography (echoEG). Ultrasound scan results suggest volumetric process in the cranial cavity, primarily by the displacement of the midline of the brain with hemispheric
localization of the tumor. With echoEG, in some cases, you can get a direct signal from the very tumors, especially if it contains a cyst, and also reveal ventricular hydrocephalus.
5. Lumbar puncture is used for pressure measurement and laboratory analysis cerebrospinal fluid. Currently, with brain tumors as an independent diagnostic procedure, it is rarely used, primarily due to the fact that with a high intracranial pressure, it can cause dislocation and wedging of the brain. The puncture is performed in the placing the patient on his side, take no more than 2-3 ml of liquid for the study. After her, a strict

bed rest for 2 days (if a tumor of the posterior cranial fossa is suspected on the 1st day of the patient must lie without a pillow).
6. Gammaencephalography (GEG) - a method of radionuclide scanning on a gamma camera or scanners of a different type. The method is based on registration by extracranially located detectors gamma radiation of a radiopharmic drug (most often Tc-pertechnetate is used), injected into the vascular channel. The method is most informative for richly vascularized tumors (meningiomas, glioblastomas, metastases), actively accumulating the radiopharmaceutical.
7. X-ray computed tomography (CT) and magnetic resonance imaging (MRI) - the most informative diagnostic methods. As they are introduced into practice, they are increasingly are used as the primary survey method, the data of which is supplemented as necessary data from other (invasive) methods used in a neurosurgical hospital.
8. Cerebral angiography is based on X-ray imaging contrasted vessels of the brain. For supratentorial tumors, carotid is used, and with subtentorial - vertebral angiography. The presence of a tumor is judged by the dislocation of blood vessels, identifying her vasculature. Blood supply, relation to large vessels are assessed; in most cases on the angiogram can be fairly confident in judging the histostructure of the tumor. Currently digital computed angiography is being introduced into practice, in which a contrast agent is injected into peripheral vein; after special processing, the computer forms a fairly clear image vessels. Recently, magnetic resonance angiography has begun to be used, which does not require administration drugs directly into the artery.
9. Pneumoencephalography. Due to the widespread use of CT and MRI, pneumoencephalography is used very rarely. When filling only the basal cisterns with air (pneumocisternography) well, tumors of the chiasmatic-sellar region are detected, as well as other basal neoplasms, for example, tumors of the cerebellopontine angle. Pneumocisternography is important in differential diagnosis between an endosellar tumor, a cyst and the so-called empty sella turcica syndrome.
10. Ventriculography is a method based on the introduction of a contrast agent (air, mayodil, amipak) into the cavity of the lateral ventricle by the puncture method after the imposition of the milling hole. Ventriculography is most informative for midline tumors, ventricular tumors, hydrocephalus. Currently, puncture ventriculography is losing its importance due to the space of CT and MRI.
In neurosurgical clinics, a number of special methods are used to study the brain blood flow, intracranial pressure, special electrophysiological techniques, including the use of implanted electrodes for the study of bioelectric and metabolic processes in separate brain structures. Positron emission tomography (PET) is being introduced, based on registration of the accumulation of short-lived radionuclides in the brain, which makes it possible to study the course of metabolic processes. Stereotactic biopsy and tumor surgery, ventriculoscopy with using miniature fiberscopes also equipped with tumor biopsy devices, or emptying the cysts.
The common task of such an extensive diagnostic complex is not only to establish accurate topical diagnosis, but also a judgment on the histology of the tumor, its blood supply, attitude to large vessels, the state of cerebral hemodynamics and circulation of cerebrospinal fluid.

Treatment

Surgery

Indications for surgery. In most brain tumors, the indications for surgery prevail over indications for other methods of treatment. Nevertheless, the problem of determining indications and contraindications to surgical treatment is difficult and its solution is not always unambiguous. So, absolutely shown an emergency operation is considered to be an operation with an increasing hypertensive syndrome with symptoms implantation and dislocation of the brain. In this case, the choice of the type of operation depends on a number of factors: localization and histology of the tumor, the severity of the patient's condition, age, the presence of concomitant diseases, etc. However, in some cases, the operation is not performed even with an immediate threat to life. patient: mainly with inoperable, deeply located malignant gliomas (especially in

case of relapse), in elderly patients who are in a terminal state, as well as in cases
when a tumor is not available for direct surgery, and a palliative one is either impossible or will not give
positive effect. Urgent indications for surgery arise when there is an imminent threat of loss
an important function, for example, in the case of severe visual impairment in the presence of primary atrophy
or
a sharp stagnation in the fundus.
However, in some cases, the operation does not restore the impaired function. Usually not
hearing returns with neuromas of the VIII nerve, vision is not restored in blind patients, especially with
secondary atrophy of the optic nerves. In such situations, we are talking about relative indications for
operations, if there is no immediate threat to the patient's life. Also recognized as relative
indications for surgery, if there is a high probability that it can aggravate the existing defect, for example,
when
gliomas of the motor, speech and some other areas. Relative indications for surgery are also discussed
when another method of treatment may be no less effective, for example, radiation therapy for
endosellar pituitary adenoma or radiation therapy with chemotherapy in deeply located
glioma, etc.
Thus, when determining the indications for an urgent operation, many factors are taken into account,
among which, in most cases, the opinion of the patient himself or his
relatives.
Operations for brain tumors. For surgical access to the tumor, two
Requirements: 1) sufficient visibility must be provided to conduct an effective operation; 2) access
should be as gentle as possible in relation to the functionally important parts of the brain. Most
access is widespread, requiring craniotomy. There are two types of trepanation - bone
plastic (craniotomy) and resection (craniectomy). In the first variant in the bones of the skull
form a window; the bone is put into place after the operation; in the second case, the bone is resected with
nippers.
Resection trepanation is used less often, for example, for tumors affecting the bones of the cranial vault
(the defect can be immediately replaced with a specially treated homograft or plastic), and
when there is a need to create additional space with a high internal
cranial pressure and brain prolapse (for example, bilateral subtemporal decompression along
Cushing). Bone resection is also used in operations on the posterior fossa, where the brain remains
protected by a thick muscle layer.
Almost all extracerebral tumors are removed without incising the brain tissue. In particular, for
basal tumors, approaches have been developed to expose the tumor by lifting
brain.
Unavoidable brain incisions required to remove intracerebral and intraventricular
tumors, it is desirable to produce in the "dumb" areas of the brain. When removing tumors from functionally
significant parts of the brain, it is advisable to make small brain incisions directly above the site
location of the tumor. Modern technology and, above all, the use of microsurgical methods
allow the removal of even large and widespread tumors through small cuts in the cortex.
In addition to operations carried out by craniotomy, the destruction of the tumor is possible
by introducing radiopharmaceuticals into it, emptying the cyst or biopsy of the tumor through a small
milling hole. Such operations are carried out mainly using the so-called
stereotaxic method.
Some basally located tumors can be removed using access through
nose and main sinus (pituitary adenomas, craniopharyngiomas and other endo- and endosuprasellar
tumors) or oropharynx with resection of the clivus (with chordomas). To remove small,
predominantly intracanal neurinomas of the VIII nerve, a translabyrinth approach can be used.
The main types of operations are as follows.
Radical or partial removal of the tumor. Total or subtotal deletion is possible
in most extracerebral tumors - neuromas, meningiomas, pituitary adenomas, and some
gliomas.
Partial removal of the tumor, regardless of its histostructure, is performed in the following cases: 1) radically
unremovable tumor due to its localization and pronounced infiltrative growth, for example, a tumor
the base of the skull, when only its intracranial part is removed; 2) with radically removable

tumors, but if this leads to the appearance of gross neurological defects, for example, in some
basal meningiomas that overgrow the carotid artery and its branches. Partial removal of the tumor may

pursue three goals: 1) relieve compression of functionally important intracranial structures, for example decompress the optic nerves; 2) achieve “internal” decompression at high intracranial pressure, when the removal of part of the tumor reduces intracranial pressure and saves a dying patient; 3) shrink a large, radically inoperable tumor to size, allowing the most effective radiation treatment.

Palliative operations. They are taken when direct intervention on the tumor either it is impossible at all, or, according to the patient's condition, it is advisable to postpone it until the secondary tumor symptoms (mainly occlusive-hypertensive-hydrocephalic syndrome). An example such operations are decompressive craniotomy, various operations on the cerebrospinal fluid system, perforation of the bottom of the third ventricle according to Stuckey, perforation of the interventricular septum, and draining operations.

Among draining operations most often apply Thorkildsen ventriculocisternostomy, ventriculoatriostomy, lumboperitoneal anastomosis.

In addition, for brain tumors, plastic surgery can be performed (plastic surgery dura mater and bone, plastic closure of cerebrospinal fluid fistulas), as well as some analgesic operations (transection of the roots, often of the V nerve, stereotaxic thalamotomy, etc.).

Radiation treatment

Radiation methods can be divided into radiosurgical and radiotherapy. Radiosurgical ones include: 1) implantation of solid pharmaceuticals (yttrium-90) into the tissue of the tumor, mainly the tumor of the base of the skull; there is also evidence of the possibility treatment by this method of endosellar pituitary adenomas, manifested by a sharp pain syndrome; 2) injection of liquid radiopharmaceuticals (yttrium, gold) into a tumor cyst, the technique was previously used mainly with craniopharyngiomas.

Radiotherapy methods of treatment include external beam radiation therapy: X-ray and gamma therapy, irradiation with a beam of protons or other heavy particles.

Drug treatment

Medical treatment options are limited. Perhaps the only effective the drug can be considered parlodel (or its analogs). It is used to treat with microprolactin pituitary gland, when it is possible to stop tumor growth and often restore fertility.

Some brain tumors, especially after surgery, require medication to correct a number of symptoms: anticonvulsants, vasodilators and vitamins (with the phenomena of atrophy of the optic nerve discs), nootropics (with disorders of brain metabolism and asthenic syndrome), hormone replacement therapy, etc.

SPINAL CORD TUMORS

Spinal cord tumors account for about 10-12% of all CNS tumors. They are classified into intramedullary (intracerebral) and extramedullary (extracerebral) and occur in a ratio of 1: 4.

Among extramedullary tumors, meningiomas and neuromas are most common, among intramedullary ependymomas predominate, less often astrocytomas and oligodendrogliomas. Glioblastoma the spinal cord is an exceptional rarity; medulloblastomas - most often metastasis from the posterior cranial pits.

Intracerebral tumors dorsal brain characterized by greater biological benign than the same brain tumors. Extracerebral spinal cord tumors they do not have such differences in their biological properties.

In general, spinal cord tumors are more common in patients of mature age. In addition, there are age-specific distribution within different groups of tumors: neuromas and meningiomas

predominate in adults, and ependymomas and dysgenetic tumors (teratoma, epidermoid cysts) - in children.
Extracerebral spinal cord tumors are subdivided into extradural, intradural and extra-intradural. Intradurally located mainly neuromas and meningiomas.
Extra-intradural membrane-infiltrating meningiomas and hourglass neuromas make up approximately 8% of cases of all neuromas of spinal localization. Extradurally located primary tumors of the spine, often malignant (sarcomas, chondrosarcomas, lymphosarcomas or metastases).
Depending on the relation of the tumor to the spinal cord, they are divided into ventral, dorsal and laterally located neoplasms.

Clinic

The clinical picture of spinal cord tumors consists of radicular-meningeal, segmental and conductive symptoms.

Root-sheath pain is one of the early symptoms of extracerebral tumors, especially if they are associated with the root; however, more often than in 50% of cases, they are also noted with intracerebral tumors, but in the latter case, a “burning” shade of pain is characteristic. Duration of radicular pain with benign tumors can be several years old. They can be local in nature; v in particular, they may be associated with soreness during percussion of the spinous process of the vertebra, corresponding to the localization of the tumor. Often, pains radiate along the root, sometimes simulating other diseases, in particular intercostal neuralgia. The pain usually worsens when lying down, and also with a "liquor jolt" (for example, during a cough) or Quekenstedt's test. They can accompanied by various disorders of sensitivity (hyperalgesia, paresthesia, hypoesthesia) by radicular type.
Segmental disorders are associated with damage to the spinal cord at the tumor level. When squeezed or tumor infiltration of the posterior horns, they are predominantly sensitive; with damage to the motor neurons of the anterior horns, peripheral paresis occurs in the area corresponding to the affected segment. Differentiating radicular from segmental disorders is sometimes difficult: the main difference between segmental defects is dissociated sensory impairment.
Conductive disturbances occur when the tumor is compressed or destroyed by the conductive pathways spinal cord and are manifested in varying degrees of severity by motor and sensory defects downward from the level of the lesion.
Movement disorders are characterized by signs of central paresis and can be a relatively early symptom in intramedullary tumors and are more often more pronounced than sensitive defects. As a rule, anesthesia develops only against the background of complete paraplegia. The nature of the sensitive conduction disorders are determined mainly by the extra- or intracerebral localization of the tumor.
So, for an extramedullary tumor, the “ascending” type is more characteristic, when anesthesia begins with toes and gradually spreads up to the level of the tumor. With intramedullary tumor violations of sensitivity often develop in a “descending” type: first, the upper border, corresponding, as a rule, to the localization of the tumor, and then, as compression of the lying outwards conductors, anesthesia goes down.
Features of radicular, segmental and conductive disorders are determined not only by extra- or intracerebral localization of the tumor, but also its relation to various surfaces of the spinal cord.
So, with a laterally located tumor, especially an extracerebral tumor, a syndrome of half spinal cord lesions (Brown-Séquard syndrome). The latter is manifested by radicular and segmental disorders on the side of the tumor (mainly radicular anesthesia with a narrow zone of hyperesthesia), as well as conductive disturbances downward from the level of the lesion: central paresis combined with anesthesia of proprioceptive sensitivity on the side of the tumor and anesthesia exteroceptive sensitivity on the other side. As the compression of the spinal cord increases a picture of complete transverse lesion with impairment and pelvic functions gradually develops, which in the earlier stages, spinal cord compression is usually preserved due to bilateral innervation organs of the small pelvis.
With a central or dorsal location of the tumor, the symptomatology may initially be symmetrical. Ventrally located tumors are characterized by a predominance of tone disturbances over

paresis. The clinical picture of a spinal cord tumor also depends on the location of the tumor along the length brain and histological structure: symptoms develop especially rapidly in malignant epidural tumors.
Determining the boundaries of the spinal cord tumor is a crucial moment in the topical diagnostics. With intramedullary tumors, the upper limit can be defined quite clearly even in the early stage of the disease in terms of sensitivity.
With extracerebral localization, judgment about the upper border of the tumor based on the analysis sensitive irregularities may not be accurate. This is due to a number of reasons: 1) radicular and segmental disorders may be absent due to functional overlap of the affected area adjacent segments; 2) the level of conduction disturbances has not yet "risen" to the level of the tumor (the probability of an error is especially high when both of these reasons are combined); 3) with tumors large the length of the spinal cord along the length of the spinal cord, the level of sensory conduction disorders can correspond to the lower, not the upper border of the tumor.
Moreover, the level of sensory disturbances may change due to vascular factor or swelling of the brain. Therefore, great attention should be paid when determining the upper border of the tumor.
detection of local tenderness of the vertebrae, a thorough assessment of the radicular-meningeal and segmental symptoms, assessment of the state of reflexes.
Determination of the lower border of the tumor can be based on the analysis of the level of conductors violations of sensitivity, but mainly based on the assessment of the reflex sphere. So, above the level of the tumor reflexes do not change; along the length of the tumor, they are reduced or absent and can supplemented with radicular or segmental disorders of the sensory and / or motor

(peripheral paresis) functions. Below the level of the lesion, there is an increase in reflexes with expansion reflex zones and a combination with other symptoms of central paresis. It should be emphasized that with tumors of small length, it is often possible to determine only one level of damage.
It is also necessary to recall the discrepancy between the segments of the spinal cord and the vertebrae of the same name.
In intramedullary ependymomas, clinically defined tumor boundaries are usually less true due to their tendency to grow in the form of a "spindle" along the central canal.

Diagnostics

Refined diagnosis of a spinal cord tumor most often requires the patient to stay in specialized hospital.
At the outpatient stage, in addition to a thorough neurological examination, which may be supplemented by some special studies (in particular, electromyography), it is necessary X-ray examination of the spine - spondylography on the corresponding symptoms level. With a tumor located in the cavity of the spinal canal, local expansion can be detected its lumen, osteoporosis of the base of the arch corresponding to the tumor, expansion of the intervertebral holes with intraforamenal neuroma, etc.
With tumors of the spine, focal destruction of the vertebral bodies, compression fractures and dislocations due to gross destructive changes.
Lumbar puncture for spinal cord tumors is almost mandatory research. It is produced for laboratory examination of cerebrospinal fluid, conducting liquorodynamic tests and studies of the subarachnoid space of the spinal cord using X-ray contrast agents or radiopharmaceuticals.
In the cerebrospinal fluid with a spinal cord tumor, the content of squirrel. This is more often observed with extracerebral tumors (neurinomas). The protein content is higher, the lower the tumor is located, a high level of protein is characteristic of a complete CSF block. Can to detect tumor cells, more often with sarcomatosis of the membranes, metastases.
With tumors of the cauda equina, a so-called dry puncture is possible. Less often with ependymomas of this localization with a lumbar puncture, you can get a bright yellow cystic fluid.
During a lumbar puncture, if a tumor of the spinal cord is suspected, Kweckenstedt test.
In all cases, myelography using water-soluble contrast media is indicated.
CT and MRI are extremely informative. If MRI is available, all other methods paraclinical studies are usually superfluous.

Treatment

Surgery. Indications for surgery. Operations are considered to be absolutely shown.
for almost any symptomatic spinal cord or spinal tumor
spinal cord compression. The high degree of functional compensation of the spinal cord makes
justified operation even with paraplegia, including in elderly patients. Shown
operations and with a sharp pain syndrome due to compression of the roots of the spinal cord also
practically regardless of the nature of the tumor. However, in patients with metastasis, if
paraplegia surgery is not justified, since there is no hope of restoring the function of the spinal cord. Not
operations are performed in the case of multiple metastases.
Spinal cord tumors are most often accessed through laminectomy, i.e.
resection of the spinous processes and arches of the vertebrae over the site of the tumor. With exact
topical diagnosis for the removal of most extracerebral subdural tumors is sufficient
resect two adjacent arches; with tumors of a large extent, in particular ependymomas, sometimes
it is necessary to carry out a more extensive resection of the arches. Hemilaminectomy (half arch resection)
and interlaminar access (spreading the arches without resecting them) with spinal cord tumors practically
do not apply. In exceptional cases, to remove small neuromas, it is used
transforaminal access.
Anterior
and anterolateral approaches with resection of a part of the vertebral body (or bodies), followed by fusion.
Usually, autografts from the iliac are used to replace fragments of the removed vertebrae.
bones or ribs.
With severe pain syndrome in the case of an inoperable tumor (more often a spinal tumor)
carry out
analgesic
operations:
crossing
stifled
roots,
cut
spinothalamic pathway above the lesion level.
Radiation treatment. Radiosurgical methods are rarely used.
External beam radiation therapy, mainly gamma therapy, combined with decompression
laminectomy or as the main method of treatment is used for primary and secondary
malignant, as well as some benign (hemangioma) tumors of the spine. V
in combination with a surgical method, external beam therapy is used after decompression

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laminectomy for inoperable gliomas or after partial removal of radiosensitive tumors
(more often ependymoma).
Preventive irradiation of the entire spinal cord is performed after removal of malignant,
tumors metastatic along the liquor pathways (medulloblastomas, malignant germinomas, etc.).
Medical treatment. Chemotherapy is mainly used in combination with radiation therapy.
Its effectiveness is low.

CRANIAL INJURY

Significant dynamism and often delayed deployment of clinical manifestations
traumatic brain injury (TBI) is often the cause of misdiagnosis and treatment
evacuation tactics for this type of pathology. The high mortality rate among
victims with severe TBI and high disability of persons who have undergone even the so-called
easy TBI. At the same time, long-term forecasts show a tendency towards an increase in the
number of
suffered from TBI and the specific gravity of certain forms of TBI.
Classification [Konovalov AN et al., 1986]. **...** There are 5 clinical forms of TBI:
a) concussion;
b) mild brain contusion;
c) moderate brain contusion;

d) severe brain contusion;
e) compression of the brain.
A concussion is characterized by the shutdown of consciousness for a time from a few seconds to
a few minutes. Mild brain contusion is characterized by switching off of consciousness after injuries lasting from a few minutes to an hour. Moderate brain contusion
characterized by switching off of consciousness after trauma lasting from several
tens of minutes up to 4-6 hours. Severe contusion of the brain is characterized by
consciousness after trauma lasting from several hours to several weeks. Compression
the brain is characterized by a life-threatening growth through one or another interval
time after injury or immediately after it cerebral, focal and stem
symptoms.
The severity of the victim's condition is determined primarily by dysfunction of the trunk
the brain and life support systems of the body (respiration, blood circulation). One of
leading signs of damage to the trunk and overlying parts of the brain is impaired consciousness.
There are 7 gradations of the state of consciousness in TBI: a) clear consciousness; b) moderate stunning; v)
deep stun; d) stupor; e) moderate coma; f) deep coma; g) transcendental coma.
Clear consciousness is characterized by complete preservation of consciousness with adequate reactions
to the environment. Stunning is characterized by impaired perception when intact
limited verbal contact against the background of an increase in the threshold of perception of external stimuli
and a decrease in their own activity. Stupor is characterized by switching off consciousness with the preservation of coordinated protective reactions and closing the eyes in response to pain, sound and other stimuli. Coma is characterized by switching off consciousness with complete loss
perception of the surrounding world and oneself.

Clinical presentation and diagnosis of various types of trauma
Brain concussion. This is one of the most common forms of TBI, while
the same time the least studied and clinically delineated. According to modern classification, concussion
the brain qualifies as a single, functionally reversible form and is not
subdivided. Many of the signs previously used to distinguish concussion into
degrees, represent manifestations of brain contusion. Concussion refers to those of him
lesions in which there are no structural changes. It is very important to distinguish between concussion
as a mechanism of trauma and concussion as a clinical form of TBI. It is no coincidence that until now
Many people with ephemeral head trauma are diagnosed with
concussion when it comes to completely different diseases. On the other side,
under the “mask” of concussion, more severe forms of TBI, including the required
surgical treatment. The paucity of objective data creates sometimes insurmountable
Difficulty differentiating concussion from head contusion. In this regard, it is important
scrupulous assessment of clinical symptoms and data of instrumental methods.
It is necessary to follow the principle: the clinical form (in this case, a concussion of the head brain) must have clinical signs. In the absence of such, we are talking about a bruise
soft tissues, or no head injury at all. Absence, as a rule, by the time
examination of objective signs of organic brain damage, including when
paraclinical examination, leads to the fact that the diagnosis of concussion is usually based on

on the anamnestic data - the presence of traumatic amnesia and (or) vomiting. The fact of loss consciousness can be amnesized by the patient.
To understand the clinical manifestations of concussion, it is important to consider some aspects of its pathogenesis. Shock-shaking effect of mechanical energy covers the brain as a whole, however, the peculiarities of intracranial topography determine selective violation of vulnerable structures. What happens during the concussion is transient movement of the brain relative to the skull almost always leads to microdeformation of the hypothalamus
in the area of the funnel directly connected with the non-displaceable pituitary gland, which is its kind of "brain anchor".
The underlying stem structures can also undergo transient deformation, which defines a short-term loss of consciousness. Concussion as a mechanism of trauma is a factor overstimulation of the vestibular complex, which forms a picture of vestibular hyperesthesia. Among the subjective symptoms, headache is noted (often in the area of impact head), nausea, dizziness, retrograde amnesia. Duration of loss of consciousness after an injury does not exceed a few minutes, it is often generally denied by the victim.
The main objective signs: clear consciousness, may be moderate for a short time stunning. Moderate arterial hypertension is often noted (in the absence of it in anamnese), lability of facial vascular tone, hyperhidrosis. A few vomiting.
Neurologic symptoms are vague: convergence weakness, shallow sweeping nystagmus, decreased abdominal reflexes. In the vast majority of patients with local no symptoms. In some patients, certain signs of damage to soft head tissues.
Some patients with a clinical picture of a concussion of the brain are admitted to hospital with symptoms of severe arterial hypotension, which is associated not with the severity of the brain injury, but
with wound injuries of the scalp in the projection of the branches of the external carotid (superficial temporal,
occipital artery) or internal carotid (supraorbital artery) artery. These vessels are fixed are formed in the thickness of the scalp by connecting bridges that form cells, and therefore do not
subside, which prevents the spontaneous cessation of bleeding.
Instrumental examination includes craniography and echoencephaloscopy. These two methods are obligatory. EEG does not provide essential information. As for lumbar puncture, the indications for it are extremely rare. CT and MRI changes are not reveal.
The most important element of diagnostic tactics is the repetition of examination and echolocation. The condition of patients usually improves quickly (within 1 day), and then normalizes.
Brain contusion. Brain contusion is morphologically characterized by the presence areas of brain damage. Due to the fact that these areas have different locations, different length and depth, brain contusion is divided into degrees: mild, moderate and severe.

Despite the existing criteria for assessing the severity of the injury, during the initial examination it is often
it is difficult to distinguish mild and sometimes moderate cerebral contusion from a concussion, and
that's why. When bruised, focal destruction of brain tissue is usually combined with the same functional shifts that are observed during concussion, and the areas of damage
are often located in areas, the loss of function of which does not give a distinct neurological symptoms (pole-basal parts of the frontal and temporal lobes). Duration of loss

consciousness is not always reliably established. Clinical diagnosis is helped by a large
the severity of cerebral disorders (stunning, repeated vomiting). Reliable
the criteria for the diagnosis of brain contusion in such cases are: 1) detection of bone fracture
cranial vault on survey craniograms or clinical signs of a skull base fracture;
2) the presence of blood in the cerebrospinal fluid. Reliable information in this regard is usually
give CT and MRI of the head.
When analyzing craniograms, it should be borne in mind that linear fractures of the cranial vault
are not
grow together, moreover, over time, diastasis may even increase. Therefore, without reliable
anamnestic data can be extremely difficult to distinguish a “fresh” fracture from an “old”
one. Local
changes in the projection of "fresh" fractures, as well as subjective sensations on palpation
can often be almost absent.
Mild brain contusion is characterized by switching off consciousness after trauma.
from a few minutes to an hour. As a rule, retro-, con- ianterograde amnesia is noted,
repeated vomiting. Vital functions are not grossly impaired. There are light focal and
meningeal symptoms. Fractures of the bones of the skull, subarachnoid hemorrhage are possible.
A moderate brain contusion is characterized by switching off consciousness after
trauma for a time from tens of minutes ”to 4-b hours. Amnesia is expressed. There is repeated
vomiting.
Mental disorders, moderate transient violations of vital functions are possible.
Meningeal symptoms are common. Revealed secondary stem symptoms, focal
symptoms that usually subside within a few weeks.
Fractures of the vault and base of the skull, significant subarachnoid
hemorrhage; CSF pressure is often increased.
Severe brain contusion is characterized by switching off consciousness after
injuries for a period of several hours to several weeks. Movement is often observed.
excitation. Dysfunctions of vital organs are threatening. Often dominates
primary stem neurological symptoms, which "obscure" focal
hemispheric symptoms. Paresis of the extremities, subcortical disorders can be detected
muscle tone, epileptic convulsions. Symptoms regress slowly, often
severe residual effects. Such a bruise, as a rule, is accompanied by fractures of the skull and
massive
new subarachnoid hemorrhage.
Echolocation for brain bruises often shows the displacement of the M-echo, which is
an indication for cerebral angiography. CT data for brain contusion are diverse.
For severe brain contusion, primary stem lesions
are characterized by gross persistent disturbances of consciousness up to coma. Recovery of
consciousness
the surviving patients go through the phases of apallic syndrome and akinetic mutism. Level
stem lesions help to establish the following symptoms (in addition to those discussed above
when describing hormetonia): rhythmic movements of groups of mimic, masticatory muscles
(mesodiencephalic level); bilateral miosis (mesencephalic level); bilateral
fixed mydriasis, muscle hypotonia (mesencephalic-bulbar level). Symptoms
develop immediately after injury, are characterized by the stability of manifestations. One of the
most difficult
The most common forms of brain contusion are diffuse axonal injury,
characterized by multiple small hemorrhages in the semioval center,
corpus callosum, stem sections against the background of generalized cerebral edema.
In most cases, as indicated, with TBI, the trunk lesions are secondary, from direct
injuries it is protected by cerebrospinal fluid in cisterns, from all sides surrounding

stem formations. The development of primary focal symptoms in contusion is mainly associated with
with damage to the cerebral hemispheres. The most severe foci arise by the mechanism counterstrike, and the severity of early focal symptoms will be much greater when frontal orientation of the force vector (damage to motor, speech zones) than with sagitis tal (pole-basal lesions of the frontal, temporal lobes). At the same time, one of the most a common mechanism of injury is a fall to the back of the head. Moreover, the absence

visual control and shock absorption by the hands can contribute to severe cerebral ral injuries in the sagittal direction of the force impact.
In this regard, it is necessary to consider the methods of early recognition of hidden severe the consequences of a blow to the occipital region with acceleration. Recreation of the nature of the injury (with
the presence of amnesia, loss of consciousness) is helped by a targeted assessment of local changes in soft
tissues in comparison with craniographic data. For injuries to the occipital region, in addition to overview craniograms, it is necessary to perform (urgently) an additional X-ray posterior semi-axial projection (supine position, the tube is deflected at an angle of 45 ° when the direction of the rays to the frontal region). At the same time, fractures of the occipital bones, often extensive, not detected by plain craniography. Fractures are not always are located in the midline. More often they deviate to the side. The following options are typical: passage of the fracture through the scales of the occipital bone downward with the intersection of the middle sections
pyramids of the temporal lobe, and lateral from top to bottom with the transition to the aspirate region and posterior
departments of the pyramid. These common variants can be recognized clinically: a combination traces of bruising of the soft tissues of the occipital region with bleeding, liquorrhea from the corresponding
external auditory canal, unilateral cochleovestibular disorders are characteristic
for the first option. In the second variant, an isolated behind-the-ear hematoma is noted, Niksha due to damage to the mastoid venous graduates. With significant
bleeding from them, blood spreads through the sheath of the sternocleidomastoid muscle down, causing muscle irritation and torticollis phenomenon. Distinguish behind-the-ear hematoma as early
a sign of severe trauma allows limited skin staining, whereas with a direct impact the hemorrhage also spreads to the skin covering the auricle.
A thorough assessment of the external signs of trauma often makes it possible to clinically establish
the presence of fractures of the base of the skull, which are poorly detected by x-ray. Wherein it is also important to define the "masks" of this severe injury, which has not only clinical but and important legal significance. For a fracture of the anterior cranial fossa, it is considered the characteristic delayed appearance of periorbital hemorrhage ("glasses symptom"). Being bright and long-lasting, this symptom does not go unnoticed. But not always
it indicates the presence of a fracture. The most common "mask" is
signs of blood migration with hematomas of the soft tissues of the frontal region. At the same time, the delayed (1/3
- 1 day) the manifestation of "points" in the absence of signs of direct trauma to the orbital region (absence of subconjunctival hemorrhage) is alarming for a fracture of the anterior
the cranial fossa, which is the upper wall of the orbit. However, in such cases, staining of the skin

the eyelids are usually significant, and the existing hematoma of the frontal region decreases markedly in size.
The lower "half-lines" often indicate a fracture of the bones of the nose and subcutaneous bleeding from
branches of ethmoid arteries.
A real suspicion of a fracture of the anterior cranial fossa arises upon registration
delayed developing, moderately pronounced hemorrhage in the upper eyelid
(The tarsoorbital fascia prevents the blood from spreading downward). In addition, often
periorbital hemorrhages as a sign of a fracture of the base of the skull develop immediately after TBI, combined with damage to the upper jaw (type Le Fort 2, 3), while the diagnosis facilitated by the establishment of the mobility of the upper jaw.
Thus, the timing of the appearance of periorbital hematomas in isolation from other signs is not are decisive in terms of diagnosing a fracture of the anterior cranial fossa. Undoubted
the fact of a fracture of the base of the skull will be in the presence of nasal
liquorrhea. Distinguish profuse
liquorrhea with an admixture of blood from bleeding allows the assessment of the symptom of a stain on gauze
napkin: a falling drop of bloody cerebrospinal fluid forms a red spot in
center with a yellowish halo along the periphery. blood, altering in the stomach, with vomiting stands out as "coffee grounds". In such cases, diagnostic actions should be
especially responsible (excluding TBI, combined with damage to the abdominal
cavities, assessment of the premorbid state with the involvement of related specialists).
Nasal liquorrhea can be the result of a fracture not only of the anterior cranial fossa, but
and pyramids Reliable diagnosis of liquorrhea, especially latent, is provided when endolumbar administration of a radiopharmaceutical and laying for an hour in the nasal passages of small
tampons on threads with subsequent calculation of the radioactivity of these tampons. Excess background values indicate liquorrhea.
Fractures of the base of the skull may be accompanied by internal liquorrhea and / or
bleeding into the nasopharynx and further into the digestive tract. In this case, the temporal bone

(the outflow of cerebrospinal fluid into the nasopharynx through the auditory tube). More often fracture
the pyramid of the temporal bone is accompanied by an otorrhea with an admixture of
blood. The "mask" is flowing
blood into the auricle from an external wound with subsequent outflow through the lower sections
shells. However, in such common cases, the deeper lying areas of the external
the ear canal remain free of blood, hearing is preserved in the corresponding ear. For
fracture is characterized by early hypacusia.
An important sign is the Batle symptom (hematoma in the area of the mastoid process). Wherein it is very important to note unilateral nystagmus or bilateral, but with different amplitude movements to the right and left. Prosoplegia, if it develops, is more often delayed (ischemic disturbances in a nerve passing in a narrow long canal). For fractures of the base of the skull, develop cerebrospinal fluid hypotension. The most formidable and frequent complication of a base fracture
skull is an early traumatic purulent meningitis, the development of which is complete neo-liquidity in cases of latent liquorrhea.
Rare complications of a fracture of the middle cranial fossa include carotid-cavernous anastomosis and profuse nosebleeds. Both complications are associated with wall tear
internal carotid artery adjacent to the damaged lateral wall of the sphenoid sinus on

different levels. In the first case, blood flowing into the cavernous sinus causes a characteristic symptomatology: pulsating exophthalmos, conjunctival edema, noise in the head, determined when
auscultation, usually in the fronto-orbital and temporal regions on the corresponding side, decreased vision in one eye, oculomotor disorders (compressed III, IV, VI cranial
nerves in the cavernous sinus), neuralgia of the I branch of the trigeminal nerve (for the same reason).
If the internal carotid artery is damaged before it enters the cavernous sinus
blood flows through a crack in the wall of the sphenoid sinus through the upper nasal passage out. In this case, blood loss is often life-threatening. Both complications
usually occur several days after injury (often with satisfactory
condition of the victim) and require urgent treatment in a specialized
neurosurgical department. This treatment includes endovasal reconstructive
intervention using balloon catheters.
Brain contusions are often accompanied by subarachnoid hemorrhage. It
although it is not an independent clinical form, it introduces specificity into clinical manifestations of TBI. Hemorrhage is manifested by tonic meningeal symptoms,
acyclic fever (irritation of the thermoregulatory center), severe pain
syndrome, primarily in the frontal-orbital region (irritation of the receptors of the tentorium of the cerebellum -
analogue of the Burdenko-Kramer symptom). Subsequently, predominant pain in the clearing (irritation of the cauda equina roots accumulating in the lumbar sac
decaying blood). It is important to note that sometimes with massive subarachnoid hemorrhages, tonic meningeal symptoms are almost or not defined at all (more often in persons with chronic alcoholism).
Subarachnoid hemorrhage disrupts the process of cerebrospinal fluid circulation. Reactive hypersecretion of cerebrospinal fluid leads to the development of acute hydrocephalus, which is often
is the morphological basis of hypertensive syndrome in cerebral contusions. V
subsequent blockage of the drainage pathways of the cerebrospinal fluid disintegrating blood circulation can lead to hyporesorbent internal hydrocephalus. She can be moderate and local, limited only to some expansion of the third ventricle without significant clinical manifestations. In some cases, the process progresses roughly and dropsy becomes so pronounced and widespread that it is necessary to use
interventions (lumboperitoneal, ventriculoitrial bypass grafting).
The presence of subarachnoid hemorrhage with penetrating TBI significantly increases the risk of developing purulent meningitis.
Brain contusions of moderate to severe degree may be accompanied by lesions
crush injuries, which, by their nature, become formations of a volumetric type. Violations barrier functions and microcirculation lead to local overhydration of the damaged
tissue, which causes the increasing compression of the surrounding structures. In such cases the distinction between the concepts of contusion and compression of the brain is lost.
Traumatic compression of the brain. This clinical form is qualitatively
differs from the previous ones in violation of the volumetric ratios of the cranial cavity and its contents
with redistribution of intracranial volumes. According to the criterion of danger to life

traumatic compression of the brain refers to severe TBI and, as a rule, requires urgent surgical intervention. Causes of traumatic compression of the brain; 1) internal
cranial hematomas (hydromas); 2) depressed fractures of the cranial vault; 3) foci of crush injury volumetric type; 4) cerebral edema; 5) hydrocephalus; 6) pneumocephalus. Many of the listed

morphological species can be combined with each other, while the presence of pneumocephalus (accumulation of air in the intracranial spaces) practically excludes the presence intracranial hematoma.

Traumatic intracranial hematomas and other causes of brain compression.

Intracranial hematomas are the most common form of acute escalating traumatic compression of the brain. Representing deposited blood, dissociated from pathways of blood (cerebrospinal fluid) flow, they cause a clear volumetric effect, leading to compression the brain and, as a consequence, to its dislocation. The volume of the cranial cavity in adults is constant value. The volume of its contents is also constant: brain parenchyma, cerebrospinal liquids, blood (practically incompressible substrates). Adding pathological intracranial volume in the form of extravascular blood accumulations is the cause of over-distribution of the volumes available in the norm. The most labile volume of the cerebrospinal fluid, which can be displaced into the spinal subarachnoid space (according to this reason, traumatic subarachnoid hemorrhage is not attributed to compression of the brain, since blood enters the "flowing" cerebrospinal fluid). However, this compensation factor triggered when the pathways of the cerebrospinal fluid are preserved and with the initial sufficient amount cerebrospinal fluid. The decrease in intravascular blood volume is practically unrealistic. The movement of the cerebral parenchyma is a dislocation process: temporo-tentorial insertion as an attempt to move a part of the brain from the supratentorial spaces, the lower stem - from the cranial cavity in general.

Intracranial hematomas not only constitute an additional volume themselves, but also lead to an increase in the available volumes: 1) due to an increase in the volume of the brain due to its edema (compression postischemic local edema, arterial hypertension, impairment, venous outflow); 2) due to an increase in blood volume (reflex arterial hypertension, violation of venous outflow); 3) due to an increase in the volume of cerebrospinal fluid (its reactive hypersecretion and blocking of the cerebrospinal fluid at the level of the tentorial ring).

Consideration of acute traumatic cerebral compression only from biomechanical positions is important but one-sided. It has been shown that acute compression disrupts rheological properties of blood due to a decrease in the plasticity of the walls of the shaped elements, leads to the development disseminated intravascular coagulation, disorganizes and distorts many neurohumoral processes and reactions. However, the clinical picture of intracranial hematomas according to the timing and nature of the manifestation of symptoms is largely determined by the morphological features of hematomas.

Epidural hematomas. The dura mater, which is the internal periosteum skull, firmly attached to it, especially in the area of the seams and the base of the skull. That's why accumulations of blood between the bone and the hard shell can form only with powerful sources of bleeding and usually within one of the bones of the cranial vault. Fusion of solid shells with bones in old age makes clear the casuistically rare formation epidural hematomas in the elderly and, conversely, the frequent development of this type of hematoma with TBI in young people. The mechanism of injury in cases of development of an epidural hematoma is itself, as a rule, a direct blow, as a result of which a fracture of the skull and the formation hematomas in the projection of this fracture.

The sources of bleeding are most often the branches of the middle meningeal artery, in a number

cases - the superior sagittal sinus. For epidural hematomas in most cases
the contours of the hematoma are similar to a biconvex lens both horizontally and frontally
section. This is especially clearly seen with CT of the head, which is due to punching
hard shell deep into the area between the seams. Major sources of bleeding
usually cause a large volume of hematoma, which is often represented by dense clots.
In some cases, bilenticular hematomas in the shape of an hourglass are noted. Similar type
the focus is especially difficult to recognize and radically remove,
The rate and intensity of bleeding determine the formation of the focus in the first hours after
injury. There are cases of delayed formation of epidural hematomas with combined

TBI and shock. Then the bleeding source begins to function after stabilization
tion of hemodynamics. Chronic hematomas are rare.
The clinical picture. 1. State of consciousness. Its primary loss from
subsequent restoration. The "light gap" is very significant precisely with epidurals
hematomas, as it is short. During this period, one can notice the insufficient adequacy of the
conducting, usually there is a stun. Subsequent loss of consciousness is usually sufficient
swiftly. A distinct three-phase change in consciousness in TBI is typical for
epidural hematomas, in which more often than in other types, compression of the brain occurs
in the absence of severe bruising.
2. Somatic status. Changes in hemodynamics are characteristic: bradycardia, cardiac
arrhythmia in the form of quantitative differences in heart rate at different time intervals; arterial
hypertension. An increase in blood pressure often predominates on the brachial artery with
the side opposite to the location of the hematoma. Violation of the control of the function of the
pelvic
organs may be a sign of developing decompensation of the state.
3. Primary focal neurological symptoms. Severe localized compression is usually
although not always, it causes contralateral pyramidal insufficiency, more often not
uniformly distributed along the axis of the body (phaciobrachial paresis with predominantly
basal localization of hematoma due to rupture of the middle meningeal artery, paresis in
lower extremities - with a convexital focus due to damage to the sagittal sinus). At
left-sided foci usually develop dysphasic disorders, the nature of which depends on
compression topography. Other symptoms of prolapse are difficult to detect. Irritation symptoms
-
epileptic seizures are extremely rare.
4. Dislocation symptoms. Mostly developing rapidly: signs of deficiency
oculomotor nerve (more often on the side of the focus), bilateral Babinsky symptom,
homolateral hemiparesis, which significantly complicates the clinical assessment of the side of
the lesion.
Often, hormone and other dislocation symptoms quickly join.
Diagnostics. Although epidural hematomas require intensive
shock impact, in some cases the clinical assessment may be erroneous not only in terms of
recognition of hematoma, but also regarding the presence of TBI in general. Compression of the
epidural
a hematoma can be interpreted as an acute violation of cerebral circulation, and with
lumbar puncture can obtain colorless cerebrospinal fluid. In from-
avoiding gross errors, it is necessary to try to more accurately establish the anamnesis of the
disease and
adhere to a strict sequence in the use of instrumental methods
diagnostics. Craniography - a mandatory study - in most cases allows
detect a fracture of the cranial vault. Comparison with lateralization of prolapse symptoms
allows to establish a direct mechanism of injury (hemiparesis contralateral to the fracture site and

etc.). Echoencephaloscopy allows you to establish the volumetric nature of the lesion and prove it
sideliness. Lumbar puncture is contraindicated. The final diagnosis is carried out in
depending on the equipment of the hospital and the severity of the victim's condition by angiography,
CT or overlapping milling holes.
Subdural hematomas. This is the most common, varied and "insidious" form.
traumatic compression of the brain. The subdural space is formed easily, so
how its walls are not soldered to each other, and with age, the gap between the surfaces of solid and pau-
the muddy membranes increases. The mechanism of injury is often indirect. In such cases, the source
hemorrhages are either translocating pialodural convexital veins or
basal parts of the brain, or destroyed intracerebral vessels in the focus of brain injury with
crushing. It is important to note that venous tear often occurs at minimal intensity.
traumatic impact, while the very fact of injury in the future can be denied as
victims and their loved ones. This is especially true for the elderly.
(tension of the pial veins due to cerebral atrophy). In acute cases, the hematoma has
a lunate form, spreading from the convex to the basal parts of the brain. With chronic
the flow of disintegrating blood is delimited by membranes and the hematoma takes a form close to
to the spherical. Over time, hematomas liquefy, and in the chronic phase, the contents in
mostly liquid. Appearing in most cases in the next few hours after the injury,
subdural hematomas are characterized by acute, subacute (after more than 3 days from the moment of injury) or
chronic course with subsequent decompensation.

The clinical picture. Acute hematomas form more often from damaged vessels
areas of brain crush, in connection with which the symptoms are due to both compression and
the consequences of focal brain destruction.
1. State of consciousness. Characterized by a three-phase nature of oppression of consciousness, however
The “light gap” is often not as distinct as in epidural hematomas.
2. Focal symptoms. Typical signs of pyramidal insufficiency of various
severity, aphatic disorders can be migratory in nature. Are characteristic
epileptic seizures, often secondary generalized with post-seizure
hemiparesis.
3. Dislocation symptoms develop early and determine the severity of the process.
In this case, often a breakdown in compensation occurs after the first or next epileptic seizure.
Diagnosis of hematoma in the presence of a "light period" is not difficult, almost always available
signs of injury. Suspect the presence of a hematoma during the period of persistent subcompensation
more difficult. Craniography can give negative results, and a single echoscopic examination
The scan will not show the offset of the M-echo. In such cases, in a non-specialized hospital
the correct diagnostic technique is the organization of the hourly (or with a greater frequency)
the observation mode with the registration of the parameters of hemodynamics described above, the state of
knowledge, focal and dislocation symptoms with obligatory echo control. From the spinal
puncture should be abstained. The final diagnostic tools are angiography,

showing a semilunar avascular area (often with coarse lateral dislocation of the brain), CT or trepanation.
Subacute subdural hematomas are more often formed due to rupture of pial vessels with minor head injury. Clinical manifestations are often not drawn. It has been established that subdural hematomas can appear delayed, and not only late to manifest itself: as the cerebral edema and hydrocephalus symptoms decrease pressing down on the source of bleeding stops. All this shows that in cases of erased clinical picture, paucity of anamnesis requires a special “hematoma alertness”.
The clinical manifestations themselves are often caused not so much by compression as by irritation.
brain. For hematomas of this type, the development of recurrent epileptic seizures with subsequent decompensation of the state according to general cerebral and focal criteria.
The diagnostic tactics are the same as for acute hematomas.
Chronic subdural hematomas are a special type of traumatic compression. Such hematomas develop at a minimum intensity of shock, in the presence of cerebral atrophy. The sources are pial vessels. In these conditions, even the centers of large volume cause only transient focal disturbances. Ectasia of small vessels of the solid the membrane that forms the membrane of the hematoma as a reactive structure, often causes repeated bleeding into an existing hematoma, which can cause a breakdown in compensation and rapid deterioration of the patient's condition. Clinical evaluation is very difficult differential diagnostic character.
Most typical diagnostic assumptions: senile or presenile dementia (in the absence of focal loss), brain tumor (slowly progressive course, in 50% of cases - stagnation in the fundus, denial of trauma in the anamnesis due to its insignificance), acute cerebral impairment blood circulation (hypertensive crisis with cerebral edema as a resolving factor, etc.), transient violation of cerebral circulation (flickering nature of focal symptoms).
In all cases, diagnostic tactics, as a rule, allow you to accurately establish the nature and extent of the defeat. After surgery, the prognosis is often favorable.
Intracerebral hematomas are more often localized in the areas of brain crush, that is, in the temporal or / and the frontal lobe, resulting from damage to the intracerebral vessels. Therefore, hematomas usually adjacent to foci of brain damage. CT has proven the possibility of delayed formation of intracerebral hematomas. All this determines polymorphism clinical manifestations. At the same time, it is possible to catch signs of damage to the deep sections hemisphere (extrapyramidal shade of muscle hypertension, the predominance of paresis in proximal extremities, etc.). Even with large intracerebral hematomas, there is no M-echo offset. The diagnosis can be clarified with angiography or CT.

Intraventricular hematomas. Isolated massive accumulation of blood in the ventricles brain - a rare, usually fatal injury in TBI - characterized by gross vegetative stem disorders, hormetonia, and other dislocation symptoms. Moderate accumulation of blood in the ventricles is combined with various forms of TBI and does not always have life-threatening character. The clinical aspects of this issue are still awaiting resolution.

The need for early sanitation of cerebrospinal fluid using
ventricular drainage, set initially for diagnostic purposes.
Crush foci refer to the form of a brain injury, however, they often acquire volumetric
character due to local edema and become a form of compression. To select a therapeutic
tactics, dynamic clinical and instrumental assessment and condition monitoring are important
victim. With increasing compression, removal of non-viable areas is indicated
brain.
Impressed fractures . It is a form of brain compression with a direct mechanism of injury.
Despite the severity of bone damage, palpation to diagnose a fracture is often
difficult due to local scalp edema. On the other hand, the edge of the subgaponeurotic hematoma
can be
May be mistaken for bone indentation. Pain syndrome does not reflect the degree
destruction of the bone. The size of the fracture also does not correlate with the severity of the
neurological
symptoms. So, a slight depression in the projection of speech zones can cause rough
dysphasic disorders. At the same time, extensive fractures with massive implantation of
fragments into
the cranial cavity may not give any neurological symptoms, and when
the operation reveals that the dura mater and the underlying brain are not damaged. At the same
time it is possible
install the bone fragments in place.
The main diagnostic method is craniography, especially multi-projection, which
allows you to determine the details of the fracture, the possibility of damage to large vessels, in
particular
sinuses of the hard shell.
Treatment. Disruption of the life-support systems of the body in a significant
the least determines the severity of the condition of the victims, and untimely or insufficient
correction
of these violations often leads to secondary difficult to eliminate or uncompensated
lesions that predetermine unfavorable outcomes even with mild primary
brain damage. In most cases, in the first minutes and hours after the injury, pre-
have breathing disorders.
Treatment of external respiration disorders . Peripheral and central breathing disorders
are often combined with each other, increasing hypoxia is one of the leading links in
the pathogenesis of the development of subsequent complications. Therefore, an early, fast and
reliable
elimination of respiratory disturbances or replacement provision of respiration. Only after that
you can begin to eliminate other lesions. Fundamentally the following position: up to
elimination of pronounced violations of the airway patency, measures cannot be taken
to restore biomechanics and breathing rhythm. Any other tactical decision is a gross lie.
a common mistake.
The main ways to restore the patency of the airways are determined
the predominant localization of the occlusion and its severity. Upper respiratory tract
freed from the contents with a swab on the forceps or a catheter inserted through the mouth or
nasal passages and connected to a vacuum aspirator. Extending the lower jaw forward with
the subsequent introduction of the air duct often normalizes external respiration due to
walking of the root of the tongue from the back of the pharynx and from the soft palate. The
ineffectiveness of these
the simplest activities, maintaining the sonority of breathing with the participation of auxiliary
respiratory muscles and cyanosis indicate damage to the underlying segments of the respiratory
tubes and usually require intubation. Indications for tracheostomy are usually
a few days after severe traumatic brain injury in cases requiring prolonged

assisted ventilation of the lungs, to prevent pressure ulcers of the laryngeal mucosa in the zone of contact with the walls of the endotracheal tube. Tracheostomy is indicated for persisting coma even in the absence of pronounced respiratory disorders due to the threat leakage of mucus, saliva, gastric contents into the larynx, trachea and bronchi. Reducing the invasiveness of the intervention while providing access to small branches of the tracheobronchial tree is achieved when using a medicinal tracheobronchoscopy.

To combat peripheral bronchospasm, the well-known bronchodilator effect of xanthine derivatives, they are usually used in the form of 10 ml 2.4% aminophylline solution. It should be noted that the drug has a multivalent effect and almost its use is always indicated for acute TBI. The prevailing opinion about the possible in this case resumption of intracranial bleeding is completely unfounded.

Correction of central breathing disorders. Tactics for correcting rhythm disturbances breathing mainly depends on the frequency, depth of respiratory movements and their ratio with the level of ventilation of the lungs. Most often with TBI, hyperpnea is observed, i.e. an increase in compared with the normal respiratory minute volume. In conditions of impaired blood circulation increased energy requirements of the respiratory muscles can lead to the syndrome "Robbing" the brain. Therefore, a significant increase in the frequency and amplitude of respiration in most cases, TBI in the presence of signs of damage to systemic hemodynamics requires connecting the patient to a ventilator. Moreover, the continuation apparatus breathing is absolutely necessary if the patient is disconnected from respirator causes tachycardia, arterial hypotension or increased venous pressure.

With a frequent combination of hyperpnea and hyperventilation, the resulting hypocapnia promotes the redistribution of cerebral blood flow with its improvement in the affected areas for by reducing the blood supply to unchanged parts of the brain (Robin Hood phenomenon), however significant decrease in PaCO3 less than 25 mm Hg. Art. (3.35 kPa) leads to a decompensated gas alkalosis, capillary overflow. Therefore, patients with excessive hypocapnia reco- it is recommended to transfer to apparatus breathing with an underestimation of its minute volume and maintenance moderate hypocapnia (PaCO2 30-32 mm Hg, i.e. 4-4.27 kPa). In addition, the mode is moderate hyperventilation reduces intracranial hypertension.

Indications for extubation or removal of the tracheostomy tube are persistent normalization of external respiration according to clinical signs and according to the assessment of the gas composition blood, preservation of consciousness and cough reflex, sufficient drainage function tracheobronchial tree.

Treatment of disorders of systemic hemodynamics. Moderate increase in arterial pressure in TBI is essential to maintain a sufficient level of cerebral blood flow in conditions of intracranial hypertension and therefore usually does not require therapeutic correction. A significant increase in blood pressure leads to rapid filtration water from the blood in the vessels of the brain, which is one of the leading causes of the development vasogenic edema, brain. The latter requires correction using traditional means.

(clonidine, dibazol, chlorpromazine) with the expectation of reducing systolic pressure by no more than 1/3
original. Various lytic mixtures are used containing neurotropic,
antihistamines and vasoplegics: pipolfen 2 ml, tisercin 2 ml, analgin 2 ml,
droperidol 4-6 ml or pipolfen 2 ml, chlorpromazine 2 ml, pentamine 20-40 mg, analgin 2 ml.
the mixture is used for persistent, non-arresting increases in blood pressure.
Lytic mixtures are injected intramuscularly 4-6 times a day.
Hypotension in acute TBI is less common. Her appearance more often
indicates either gross and irreversible lesions of the brain stem, or the presence of
continuing extracranial bleeding with the development of shock in concomitant TBI. Curative
tactics is determined by the reasons that caused a decrease in blood pressure.
Significant and / or persistent hypotension requires immediate correction. Start off
infusion therapy is necessary until the cause of the decrease in blood pressure is clarified.
The drug of choice is a solution of low molecular weight dextran (rheopolyglucin), with
the introduction of which, in addition to quickly replenishing the deficit in circulating blood volume,
blood viscosity and aggregation of corpuscular elements decrease, organ
microcirculation, increases plasma osmolarity, which is a factor in preventing
development of cerebral edema. Other infusion drugs are
glucosonocaine mixture, 5% albumin solution. Liquid loading volume and distribution
its time depends on the massiveness of blood loss and the state of the myocardium. Infusion therapy
should therefore be carried out against the background of the introduction of cardiotonic drugs (cardiac glycosides,
calcium preparations) at the rate of 0.5-1 ml of 0.6% corglikon solution, 10 ml of 10% chloride solution
calcium for every 500 ml of injected fluid. Criteria for the effectiveness of the
infusion therapy is stabilization of hemodynamic parameters (systolic

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blood pressure 90-100 mm Hg. Art., central venous pressure 100-150 mm of water. Art. at
absence of tachycardia), which indicates a good return of blood to the heart, as well as the level
hour diuresis (more than 15-20 ml), indicating the sufficiency of organ blood flow.
Vasopressor drugs can be used only after the elimination of severe
hypovolemia. The introduction of corticosteroid hormones, in addition to the direct pressor
effect, has a compensatory effect on the function of the hypothalamic-pituitary system.
Specific components of treatment. Brain lesion substrate and features
treatment tactics. Brain concussion. With this injury, it is necessary to observe
bed rest for 1-2 days under the control of health and condition. In the overwhelming
in most cases, except for analgesics and tranquilizers, drug treatment is not
required. Subsequently, nootropil can be prescribed. The length of hospital stay is usually
limited to 7-10 days. During this period, dynamic observation of patients is carried out, since
under the guise of a concussion, a compensated phase of its compression may occur
an intracranial (usually epidural) hematoma (“lucency”). It was this moment in
to a large extent determines the obligatory hospitalization of patients with concussion.
In many clinics, in good condition, patients are in the hospital for only 2 days, that is, that
the period when a hematoma can manifest.
The length of time spent on sick leave is determined by an objective assessment
states. It has been proven that the duration of bed rest and inpatient treatment does not affect
the incidence of postcomotional syndrome. In the genesis of the latter, the leading role is usually played by
neurotic components; sometimes you should consider the possibility and rental trends.

The controversy of the pathogenesis of post-concussion syndrome dictates the need for compliance
caution in the unambiguous interpretation of it as a neurosis.
Mild to moderate brain contusion. Treatments include the same
components as in a concussion, to which more powerful agents are added.
The main goals of therapy:
1) improvement of cerebral blood flow; 2) improving the energy supply to the brain; 3) restoration of the function of the blood-brain barrier; 4) elimination of pathological changes water sectors of the brain; 5) anti-inflammatory and 6) metabolic therapy.
Restoration of cerebral microcirculation is the most important factor in determining the effectiveness of other therapeutic effects. The main trick here is to improve rheological properties of blood: increased fluidity, decreased aggregation ability shaped elements, which is achieved by intravenous drip infusions of rheopolyglucin, albumin solution under the control of hematocrit (control indicators - 30-40), the introduction cavinton, xanthine derivatives.
Improving cerebral microcirculation is necessary to enhance energy supply brain and prevent its hypoxia. And this is important for recovery and maintenance functions of cellular structures that make up the blood-brain barrier. Thus a nonspecific membrane stabilizing effect is carried out. In turn stabilization of membrane structures normalizes the volumetric relationships of intracellular, intercellular and intravascular water sectors, which serves as a correction factor intracranial hypertension.
V
quality
energy
substrate
used by
glucose
v
the form
glucose-potassium insulin mixture, proposed by A. Labori. The addition of insulin promotes not only the transfer of glucose into cells, but also its stabilization according to the energetically favorable pentose
paths. Intravenous administration of glucose contributes to the inhibition of gluconeogenesis and performs, thus
thus, the protective function against the hypothalamic-pituitary-adrenal system (decrease in the release of corticosteroids) and parenchymal organs (decrease in the level of nitrogenous
slags).
Tissue hypoxia with mild and moderate brain injuries develops in mainly with untimely or inadequate elimination of respiratory disorders in the early terms after injury or when pneumonia appears.
Specific effect on the restoration of blood-brain barrier function have xanthine derivatives (euphyllin), papaverine, which contribute to the accumulation cAMP, which stabilizes cell membranes. Given the multifactorial effect of aminophylline on cerebral blood flow, cell membrane function and airway patency, i.e. on those

processes and structures that are especially vulnerable in acute TBI, the use of these drugs for any kind of brain damage is justified. To save the function blood-brain barrier, it is necessary to eliminate sharp fluctuations in blood pressure in the side of both an increase (vasogenic cerebral edema) and a decrease (circulatory hypoxia).

It is also necessary to prevent and correct a deficit in circulating blood volume.
(replenishing blood loss, taking into account the presence of fever, assessing urine output during dehydration therapy), thereby preventing the release of aldosterone, which activates hyaluronidase, which destroys glycosaminoglycans (mucopolysaccharides) and affects the barrier
vascular level. With increased vascular permeability, intravenous administration of 5% is indicated
solution of ascorbic acid for 1-2 weeks.
Due to the fact that with a brain injury there is a mechanical "breakthrough" in the blood-brain barrier in the damaged area, and the nervous tissue is foreign to the immune system, the immunological status deserves serious attention, and in some cases pathological brain responses are the result of autoimmune aggression.
Timely and rational use of many of the above means for
mild brain contusion usually prevents or corrects distribution disorders
water in various intracranial sectors. If they nevertheless develop, then we are usually talking about
extracellular fluid accumulation; or moderate internal hydrocephalus. Wherein
traditional dehydration therapy with saluretics (oral or in
injections), and in more severe cases of osmodiuretics (mannitol, glycerin) quickly gives a good the effect. Dehydration therapy should be carried out under the control of plasma osmolarity blood (norm 285-310 mosm / l).
In the presence of subarachnoid hemorrhage, the treatment complex includes 5% aminocaproic acid solution, less often - countercal, trasilol, gordox. The drugs are administered intravenously, 25,000-50,000 units 2-3 times a day.
Anti-inflammatory therapy for mild to moderate brain contusions, of course,
indicated in cases of the presence of subarachnoid hemorrhage, especially wounds on the head, since
with multiple anastomoses of extra- and intracranial vessels in conditions of impaired barrier function in the presence of blood in the cerebrospinal fluid, there is a threat of development
early traumatic purulent meningitis. This danger increases manifold when
liquorrhea. It is advisable to use a combination of penicillin and
sulfonamide of prolonged action. With liquorrhea, the daily dose of penicillin can be increased and should be up to 8-10 million units. Antibiotic therapy is also indicated for the elderly
people and victims with chronic pulmonary diseases in order to prevent
hypostatic pneumonia. The diagnosis of a contusion of the brain does not exclude its compression
intracranial hematoma, the signs of which may appear delayed and with greater probability - in the presence of subarachnoid hemorrhage, therefore, during treatment and monitoring patients with mild and moderate bruises requires a special “hematoma alertness ”.
The length of stay in the hospital for mild brain injuries is again determined
assessment of the patient's condition.
An obligatory component of treatment in a hospital and a polyclinic (6-12 months) is anticonvulsants (phenobarbital, carbamazepine). In treatment and recovery
the complex includes metabolic therapy (nootropics) and course use of drugs,
improving cerebral microcirculation (cinnarizine, cavinton).
Severe contusion of the brain and acute traumatic compression. At
the above forms of acute TBI, brain damage has the character
subcompensation or reversible moderate decompensation while maintaining central
the control link and mild damage to the peripheral apparatus. Therefore, treatment in such

cases corrective in terms of relieving acute transient excessive brain reactions and the organism as a whole and creating favorable conditions for the action of natural mechanisms sanogenesis. In severe TBI, which includes severe brain contusion and acute traumatic compression of it, a gross violation occurs, and then a disruption of the processes self-regulation both at the cerebral and systemic levels with depletion of the substrate energy resource on the periphery. In the absence of intensive complex measures this leads to irreversible disruption of the homeostasis system and death of the patient. In this case the problem of the choice of treatment tactics becomes much more complicated and the doctor's decisions are more responsible.

The universal pathogenic effect of the compression factor leads to a redistribution intracranial volumes with the development of progressive intracranial dislocation, with non-elimination of which the death of the patient is inevitable. Therefore, the detection of traumatic compression of the brain requires urgent surgical intervention aimed at elimination of the cause of compression and its consequences (intracranial dislocation, dislocation hydrocephalus). In this case, the operation can act as a resuscitation measure. Surgical the intervention includes, in addition to removing the focus (foci) of compression of the brain (hematomas, hydromas, crush sites), the following complementary techniques performed in the presence of corresponding indications:

1) external bone decompression - removal of a bone flap formed in the process craniotomy, and plastics of the dura mater with the creation of a reserve subdural space (in the presence or threat of development of cerebral edema); 2) interventions on connective tissue frame - dissection of the tentorium of the cerebellum, large sickle of the brain (tentoriotomy, falcytomy) to free the wedged brain areas and restore them blood flow; 3) intervention on the cerebrospinal fluid system - ventriculopuncture with organization continuous or intermittent drainage. Drainage of the ventricles, in addition to therapeutic decompression, is essential for constant monitoring of intracranial pressure and selection of appropriate means of conservative decompression. A critical indicator is the intracranial pressure equal to 30 mm Hg. Art.

Surgery is an integral part of the treatment of acute traumatic compression of the brain. However, the operation, eliminating many pathogenic influences and including some compensatory mechanisms, can disrupt others, in particular, reactions in response to surgical trauma. The topography and multiple nature of crush foci are often not allow you to remove them radically. Therefore, in the postoperative period, patients are shown intensive therapy.

Intensive specific therapy for severe TBI . Intensive planning therapy carried out in conditions of impaired self-regulation of brain functions should provide for a predominant effect not on the final results of pathological processes, but on the mechanisms of their development. When planning such treatment, in particular its specific components, you need, first of all, sufficient energy supply to the brain.

Energy supply to the brain and the fight against cerebral hypoxia. The main energy the substrate is glucose in combination with insulin and potassium ions, usually in the form of 10%

solution. The amount of the mixture introduced depends on the state of the body's resources, approximately
assessed by clinical and laboratory parameters (body temperature, breathing pattern,
urine output level, creatinine, sodium clearance, etc.), averaging 2 g of glucose per 1 kg of body weight
body per day.
Sufficient cerebral blood flow is required for the substrate to enter the brain. With severe trauma, the self-regulation of this link in the life-support of the brain is in one way or another always impaired,
which requires external correction. In general, the mechanisms of action on cerebral blood flow can be
can be easily subdivided into: 1) intravascular (change in the rheological properties of blood, intravascular pressure); 2) proper vascular (change in vascular tone) and 3)
extravascular (change in the degree of vascular compression from the outside); Improvement of rheological properties
blood (a decrease in viscosity, an increase in the deformability of formed elements, a decrease in their
aggregation ability) provide the introduction of previously considered drugs
(rheopolyglucin, albumin solution, etc.). Improvement in cerebral blood flow can be achieved by infusion of chilled blood into the main arteries of the head. This leads to an improvement
increase in brain oxygenation due to a left shift of the hemoglobin dissociation curve. However, one should
take into account that hypothermia contributes to a decrease in blood flow. Significant impact on cerebral blood flow has a level of arterial and venous pressure. Moderate ar-
terial hypertension contributes to the maintenance of cerebral perfusion under conditions of intracranial
hypertension, although there is a threat of vasogenic edema.
The effect on vascular tone, in addition to changing the gas composition of the blood, is provided the use of traditional vasodilator and vasoconstrictor drugs, but in
conditions of impaired cerebral vascular regulation, their effect may be
paradoxical. More reliable vascular decompression - elimination of perivascular edema due to decrease in the volume of intercellular fluid (saluretics, osmodiuretics).
An important condition for metabolic processes in the brain is to ensure

optimal temperature conditions. At the same time, hyperthermia is the most dangerous, sharply increasing the needs of the brain for both the energy substrate and oxygen against the background of a decrease
activity of enzyme systems for utilizing glucose and other metabolites. Emerging uncoupling of oxidative phosphorylation and respiration leads to tissue hypoxia, and oxygen continues to flow. Free radical oxidation leads to the formation
peroxides toxic to cells. In addition, hyperthermia causes severe disorders
external respiration (tachypnea) and systemic hemodynamics (decreased cardiac output, increased peripheral vascular resistance). In this regard, the elimination of hyperthermia is one of the important measures to improve the life support of the brain and the body as a whole. To distinguish
hyperthermia of infectious and central origin is helped by an acyclic nature
last. Lytic mixtures are used to reduce body temperature; elimination of deficit
the volume of circulating blood as the main condition for the efficiency of heat transfer; vasodilator drugs, glucocorticoids; physical cooling.
In recent years, the possibilities of antihypoxic effects on the brain have been intensively studied.

barbiturates. The mechanism of their protective action is to reduce the level of metabolic processes and consumption of oxygen by cells in the lesion focus, in the removal of free radicals, stabilization of lipid cell membranes. At the same time, intracranial pressure decreases. Optimal drug doses have not been determined. When using thiopental sodium, it is recommended
administration of the drug at the rate of 2-3 mg per 1 kg of body weight per hour for 8-10 days after severe
TBI. Another powerful antihypoxant is gamma-hydroxybutyric acid (GHB). For elimination of post-hypoxic reactions, it is administered at the rate of 25-50 mg per 1 kg of body weight per hour (20%
solution) for the same time as for barbiturates. When using this
the drug decreases the concentration of potassium ions, which requires appropriate correction. There is no final assessment of the effectiveness of antioxidants yet.
Another area of treatment for hypoxia is to increase the oxygen saturation of the brain. In addition to conventional oxygen therapy, hyperbaric therapy is used for severe TBI. oxygenation, which in many cases compensates for various forms of hypoxia, increases the efficiency of oxygen diffusion into the brain (especially with its edema), however, it is possible
damaging toxic effects on the brain of peroxides formed as a result
free radical oxidation. Therefore, the question of using hyperbaric
oxygenation in severe TBI requires further study.
Correction of intracranial hypertension. Causes of intracranial hypertension the following:
1) cerebral edema (treatment - dehydration, corticosteroids, aldosterone antagonists); 2) cerebral hyperemia (treatment - controlled hyperventilation, therapeutic anesthesia with barbiturates,
GHB, hypothermia); 3) hyperosmolarity (treatment - rehydration, corticosteroids); 4) compression of the brain (surgical treatment).
Extracranial causes of intracranial hypertension include:
1) impaired airway patency (treatment - intubation, mucolytics,
aspiration of contents, bronchoscopy); 2) desynchronization of breathing (elimination - hyperventilation, sedatives and drugs); 3) "shock" lung (treatment -
increased expiratory resistance, oxygen therapy); 4) violation of venous outflow (treatment - postural venous hypotension).
With excessive accumulation of intercellular fluid, cerebral edema develops. He may be vasogenic due to increased intravascular pressure or edema
develops in connection with a decrease in the osmolarity of blood plasma, which is often observed with an excess
precise administration of fluid (hypervolemic hypoosmolarity) or
hormonal link of osmoregulation (unbalanced hypersecretion of antidiuretic
hormone with a decrease in free water clearance). The appearance or intensification of edema is facilitated by
violations
structural and functional
integrity
hematoencephalic
barrier
(compression postischemic, perifocal cerebral edema). In such cases, effective
traditional means of dehydration therapy, starting with the intravenous administration of lasix or another saluretic, then osmodiuretics are administered. The latter have phase properties: in In the 1st phase, the volume of tissue fluid decreases and the volume of circulating blood increases; in

2nd phase (hypotensive plateau) increases the excretion of osmodiuretic and stabilizes intracranial pressure; in the 3rd phase, the volume of tissue fluid increases. In this case, it is possible
repeated overhydration (the phenomenon of "recoil"). Reuse of osmodiuretics

carried out under the control of osmolarity, which in blood plasma should not exceed 310-320 mosm / l. For the manifestation of the best effect of osmodiuretics, it is advisable to inject together with
rheopolyglucin (binding of water leaving the brain). Osmodiuretics are used based on 1-1.5 g per 1 kg of body weight. Mannitol and sorbitol are effective. Urea currently practically not used, since it increases the possibility of intracranial bleeding,
causes a pronounced phenomenon of "recoil", increases the content of residual nitrogen in the plasma. How
a dehydrating agent, glycerin begins to occupy a leading position. In addition to the osmotic effect,
it is metabolized by cells, binding free fatty acids that accumulate during hypoxia.
Forced osmotherapy is dangerous due to the appearance of hyperosmolar syndrome and hypertensive dehydration with the accumulation of fluid in the brain cells themselves. This leads to
an increase in their volume (cytotoxic edema or brain swelling). In this case, the volume of extracellular
fluid is reduced. Moderate transient hyperosmolarity is observed with favorable the course of TBI. The steady increase in this indicator (up to 350 mosm / l) requires urgent correction. In such cases, dehydration therapy is contraindicated, as it will lead to even greater hyperosmolarity and a sharp aggravation of the condition of the victims. Needed reimbursement of negative water balance (use of 2.5% glucose solution) in the background the introduction of glucocorticoids, in the absence of effect - artificial ventilation in the mode hyperventilation and long-term therapeutic anesthesia with barbiturates. To the use of large doses corticosteroids (dexazone) for the treatment of edema - traumatic brain swelling
genesis, are currently treated with restraint, since the effectiveness of such doses of dexazone questionable, and complications are natural.
The volume of cerebrospinal fluid, which is easily displaced from the cranial cavity, when intracranial hypertension is often reduced. Compression of the ventricular
system and subarachnoid cisterns. With temporo-tentorial wedging occurs
blockade of the outflow tract of cerebrospinal fluid, but its production continues and arises like this
called dislocation hydrocephalus. Cerebrospinal fluid secretion is reduced
diacarb, furosemide, strophanthin (in small doses). Most effectively intermittent
removal of this fluid through the ventricular drainage.
The volume of blood in the vessels of the brain is determined by both indicators of central hemodynamics, so
and the state of the microcirculatory bed. Arterial hypertension against a background of impaired self-regulation of cerebral blood flow leads to an increase in intracranial hypertension. Reducing cerebral perfusion briefly reduces hypertension, but soon leads to
ischemic edema. Physiological is a decrease in blood volume due to increased
venous outflow from the cranial cavity. This is achieved by giving the patient a position with raised head. Hyperemia of the brain leads to its edema. So, with diffuse malignant
cerebral edema, the phenomenon of increased cerebral perfusion is noted. The positive impact of control
controlled hyperventilation on intracranial pressure is largely due to

hypocapnia, which increases the tone of cerebral vessels, which leads to a decrease in volume intravascular blood in the cranial cavity.
All four intracranial volumes are separated from each other by membrane structures, constituting the blood-brain barrier (blood-brain barrier, hematocerebral, CSF barriers), maintenance or restoration of morphofunctional structure of which is an important way to correct disturbed intracranial volumetric relationships. Therapy for violations of the blood-brain barrier is based on the following steps boards: 1) elimination of circulating blood volume deficit, use of antagonists aldosterone (aldactone); 2) prevention of sharp hemodynamic fluctuations; 3) provision the brain with a sufficient amount of energy and plastic substrate; 4) the use of special digital membrane stabilizing agents (corticosteroids, papaverine, aminophylline, etc.).

DAMAGE OF THE NERVOUS SYSTEM IN DISEASES OF THE INTERNAL ORGANS
General mechanisms of pathogenesis.
The pathogenesis of damage to the nervous system in somatic diseases is due to mainly metabolic, toxic, vascular and reflex disorders.
Shifts in homeostasis resulting from a violation of protein, carbohydrate, fat, water-electrolyte, vitamin metabolism, hypoxemia and tissue hypoxia, accumulation various toxins to be removed from the body have a toxic effect on nervous tissue (neurons and gliosis cells, synapses, axons). A certain role belongs to and reflex disorders. Excessive release of neurotransmitters may occur, and then depletion of their tissue reserves, for example, norepinephrine activates the enzyme adenylate cyclase, catalyzing the formation of cyclic adenosine monophosphate (cAMP) from ATP. Last is necessary for the normal course of complex intracellular metabolic reactions, since with a decrease in cAMP decreases the activity of the genetic apparatus and enzyme systems. Consequently, in most cases, a number of interrelated factors affect, one or two of which are the most significant. With pulmonary embolism, this is an acute hypoxia and reflex disorders (shock-anoxic syndrome), with long-term current nonspecific lung diseases - chronic hypoxia with severe changes in nervous cells and glia, kidney pathology - metabolic disorders and toxicosis (hypo-, and later hyperkalemia, creatininemia, azotemia, the effect of the whole complex accumulating in the blood metabolites with the development of cerebral edema), obstructive jaundice - bilirubinemia.
Clinical manifestations depend, in addition, on heredity, the patient's constitution, age, living conditions and nutrition, bad habits, previous pathology, features of the work performed. Somatogenically caused disorders of the regulatory the influence of the nervous system on the activity of internal organs and endocrine glands create a vicious circle, contributing to the deepening of the failure of both internal organs and nervous systems. A well-known commonality of the clinic of neuropsychic disorders in response to pathology internal organs, endocrine glands confirms that they are based on close pathogenetic mechanisms - a combination of irritation and loss on various levels of the nervous system (cortex, subcortex, trunk, spinal cord, etc.) with some emphasis on one, then on the other of them.
It was found that even a relatively compensated failure of functions an internal organ or endocrine gland with an increased predisposition may lead to clinically pronounced changes in the nervous system (fatigue, headache pain, dizziness, memory loss, etc.). Against the background of somatic disorders and associated shifts in hormonal-mediator-electrolyte balance and hypoxia earlier and more often neuropsychiatric disorders develop with infections, intoxication,

injuries, chronic and acute disorders of cerebral circulation, more severe
hereditary and chronically progressive diseases.
The first signs indicating the involvement of the nervous system with somatic
diseases - this is increased fatigue, irritability, headache, sleep disturbance,
paresthesia and dysesthesia in the zones of Zakharyin-Ged.
So, with heart damage, pain often spreads to the upper chest
and on the inner surface of the shoulder and forearm on the left (segment Cvp-Thi-Thiv), in case of damage
lungs - on the neck and shoulder girdle (Csh-Civ), liver - on the right hypochondrium (Thvш-Thix),
stomach and pancreas - to the epigastric region (Thvp-Thix), kidney and ureter
- on the lower back and the anterosuperior surface of the thigh (Thxп-Li), small intestines - on the umbilical
area (Thx-Thxi). When the vagus nerve is involved, pain is often felt in the face.
(trigeminal nerve) and occiput (segment Cn); phrenic nerve - in the shoulder girdle and neck (Csh-Civ).
All these symptoms are mild and inconsistent at first. Further, if
dysfunction of one or another organ or endocrine gland is growing, then gradually they can
develop disorders of an organic nature - nystagmus, symptoms of oral automatism,
reflex changes, movement and sensory disorders. Sometimes acute illness
internal organ (pulmonary embolism, pancreatitis, obstructive jaundice,
hepatitis) debuts with neuropsychiatric disorders: agitation, motor
anxiety, hallucinations, meningeal phenomena, etc.
muscle irritability, spasms and paresthesias in the limbs are the first signs of hypocalcemia

due to insufficient function of the parathyroid glands and kidneys. Even with
a clear picture of diseases of the liver, kidneys, lungs, pancreas, organs of small
the establishment of a pathogenetic relationship between this disease and the lesion
the nervous system should be based on data from anamnesis, clinic and additional methods
research. Neuropsychiatric disorders develop, as a rule, against the background of already
identified somatic or endocrine disease. The latter may outstrip development
neuropsychic pathology for several years. Less often, there are inverse relationships: nervous
mental disorders outpace the clinical manifestations of the somatic. In this case
the interval between them is usually shorter - it rarely exceeds a period of 2-3 months, sometimes
half a year - a year. During this period of time, the symptoms of a physical illness, as a rule,
become explicit enough. You need to pay attention to the relationship between severity and
course of the underlying disease and existing disorders of the nervous system.
Diseases of the heart and great vessels. Diseases of the heart and large vessels -
congenital and acquired defects, myocardial infarction, heart rhythm disorders,
septic endocarditis, aortic aneurysm and coarctation, nonspecific aortoarteritis
(Takayasu's disease), thromboangiitis obliterans and some others may be accompanied by
various neuropsychiatric disorders. The pathogenesis of these disorders is due to
changes in blood circulation and cerebrospinal fluid circulation, vascular occlusion, embolism,
reflex disorders, sometimes the spread of the inflammatory process to the vessels
brain (for example, with vasculitis, septic endocarditis).
The initial period of heart and vascular disease is most often characterized by asthenic
vegetative-vascular disorders - general weakness, rapid fatigue, sleep disturbance,
sweating, instability - pulse and blood pressure (the so-called
neurocirculatory asthenia). Cephalgic syndrome is manifested by paroxysmal or
almost constant diffuse or more limited (temple, occiput) pain. When significant
duration and severity of the disease, except for headache and other cerebral

symptoms (nausea, dizziness), there are small focal symptoms - nystagmus, reflexes of oral automatism, hand tremors, pathological reflexes, etc.
Symptomatology of congenital heart defects manifests itself in childhood, while there is a lag in physical and mental development, syncope or epileptiform seizures, paresis and other focal symptoms. Often noted neurosis-like disorders - fear, anxiety, constant internal anxiety, sleep disorders, general weakness. The severity of symptoms depends on the severity of the underlying diseases, premorbid personality traits, age and gender.
Extensive myocardial infarction can be complicated by various cerebral disorders circulation (lethargy, lethargy, drowsiness or agitation, headache, meningeal symptoms, suppression of reflexes, etc.), sometimes turning into cardiogenic shock, cardiocerebral syndrome (dizziness, impaired consciousness, motor and sensory disorders, pathological reflexes) or cardiospinal syndrome (weakness in the limbs, changes in tendon and periosteal reflexes, conductive or segmental sensory disturbances and pelvic disorders). One of the consequences myocardial infarction can be a reflex shoulder-hand syndrome, which is characterized by severe pain in the shoulder joint, arm, especially in the hand, swelling of soft tissues, vasomotor disorders. In the future, trophic disorders are revealed - atrophy muscles and skin of the hands, osteoporosis, etc.
Cerebral circulation disorders occur especially often against the background of a disorder hemodynamics in children with congenital heart defects or in older patients, suffering from acquired heart defects, hypertension or cerebral atherosclerosis.
In patients with congenital heart defects, paradoxical embolism often occurs in the brain, the source of which is thrombosis of the veins of the lower extremities or hemorrhoids veins. The development of acute disorders of cerebral circulation in these cases is promoted as ec-
zogenic factors (physical stress, bending or turning the body, etc.), and compensatory polycythemia and increased blood viscosity. These patients often have early and late post-stroke pneumonia, which develop, respectively, in the first 3 days or 2-6 weeks after a stroke, and

violation of carbohydrate metabolism (post-stroke diabetic syndrome).
The rapidity of development of early pneumonia, its occurrence mainly with extensive foci with an effect on the hypothalamus and brain stem, more frequent development on the side, contralateral focus in the brain, the presence in the lungs of signs of circulatory disorders in the form of plethora, hemorrhage and edema indicate the important role of central neurotrophic disorders in the pathogenesis of complications.
The hypostasis factor plays a lesser role in the development of early pneumonia, but it is very important, and in some cases, a decisive role in the development of late forms of complications. In sick stroke, the activation of which is delayed for one reason or another (cardiac pathology, thrombophlebitis), prolonged stay in bed almost always leads to a violation ventilation capacity of the lungs.
In addition, for the development of both early and late forms of pneumonia, it is important the initial background of the patient's health. They are more likely to occur in individuals with repeated cerebral circulation disorders and pseudobulbar and bulbar syndromes.
Swallowing disorders in these patients contribute to the aspiration of saliva, food pieces, mucus, vomit into the respiratory tract.

An important provoking factor is ischemic heart disease with small and large-focal angiogenic cardiosclerosis, chronic lung diseases (bronchitis, bronchial asthma) with an outcome in pneumosclerosis (diffuse or limited) and emphysema, which leads to the development of pulmonary heart failure. The occurrence of a stroke in these patients even more worsens the already impaired aeration of the lungs.
Disorders of carbohydrate metabolism in the acute period of stroke develop very often. They differ in lability and the absence of the phenomena of ketoacidosis. The severity of violations depends on the severity of the stroke, the size of the focus and the nature of the process, as well as on the outcome of the pancreatic condition the mammary gland. In the recovery period after a stroke, carbohydrate metabolism gradually normalizes, however, if there is compensated organ failure (primarily pancreas), involved in the regulation of glycemia, then with the survival of the patient the risk of developing diabetes is aggravated. The postponed stroke is such thus, one of the risk factors contributing to the onset of the disease, especially in individuals with residential age.
Changes in heart rhythm (paroxysmal tachycardia, atrial fibrillation, bradycardia) are a common cause of fainting.
Fainting (syncope) conditions occur most often with atrioventricular blockade (Morgagni-Adams-Stokes syndrome) against the background of a decrease in heart rate up to 30 - 10 beats / min.
There is a feeling of lightheadedness, dizziness, general weakness, then loss of consciousness. Objectively - the face is pale, the pulse is very rare, weak filling. In severe cases tonic and clonic seizures develop, urine loss. Frequent paroxysms gradually lead to the formation of encephalopathic syndrome.
The cerebral form of thromboangiitis obliterans is characterized by simultaneous involvement of the vessels of the brain, limbs and internal organs, for Takayasu's disease - obliteration vessels extending from the aortic arch. Both forms are manifested by symptoms of dyscirculatory encephalopathy and repeated transient ischemic attacks, accompanied by dizziness, impaired consciousness, speech, visual and movement disorders you.
With septic endocarditis, damage to the nervous system is possible due to cerebral embolism. The penetration of infected emboli into the vessels of the meninges can lead to the development of purulent meningitis, and in the deeply located vessels of the brain - a single or multiple brain abscesses.
Coarctation of the aorta due to increased blood supply to the upper half of the body and insufficient lower half leads to hypertrophy of the chest, shoulder girdle, hands and atrophy of the pelvic girdle and legs. Against this background, symptoms usually develop.
discirculatory encephalopathy and acute cerebrovascular accidents - plethoric crises, parenchymal and subarachnoid hemorrhages. Symptoms aortic aneurysm - girdle pain at the level of its location, the intensity of which may vary depending on the position of the patient; further signs appear ischemic myelopathy.
In the case of localization of the aneurysm in the area of the aortic arch, located

next to the formation - the left recurrent nerve (hoarseness, bouts of coughing, choking),

phrenic nerve (shortness of breath, hiccups), borderline sympathetic trunk (Horner's symptom, burning pains in half of the face, lacrimation and redness of the eye, rhinorrhea).
The clinical picture of dissecting aortic aneurysm is the sharpest radical pain in areas of the chest or back with irradiation to the lower abdomen and legs, sometimes the development of collapse or shock.
Acute occlusion of the abdominal aorta and great arteries of the lower extremities characterized by pronounced paleness of the legs and pain in them, the disappearance of pulsation large vessels, the development of lower flaccid paralysis or paraplegia with impaired function pelvic organs, as well as conductive-type sensory disorders.
Chronic obliteration of the abdominal aorta, bifurcation of the aorta and great vessels lower limbs manifests itself gradual development symptoms of dyscirculatory myelopathy. At the same time, vegetative-trophic, sensitive and movement disorders are especially pronounced in the distal parts of the limbs (up to gangrene of the feet).
Treatment and prognosis. The most rational is a complex treatment regimen, taking into account features and underlying disease and complications. If neurological disorders develop against the background of myocardial infarction or congenital and acquired defects heart, then therapeutic measures should be aimed primarily at compensation cardiovascular failure. For cardiac arrhythmias, antiarrhythmic drugs. Patients with a sharp decrease in pulse rate (atrioventricular block) anticholinergics are prescribed, and in cases of insufficient effectiveness with appropriate indications - electrical stimulation. Septic endocarditis is treated with high doses of antibiotics.
Therapy of systemic diseases with impaired vascular patency (Takayasu disease, atherosclerotic obliteration of the vessels of the extremities) at the initial stage usually conservative with using antispasmodics " vasodilator funds, ganglion blockers, sympathetic blockade, oxygen therapy. With epileptic seizures - anticonvulsants, cerebrovascular accidents - treatment, corresponding to the clinical picture of stroke.
The prognosis is determined by the course of the underlying disease, the nature of the neuropsychic complications, timeliness and volume of treatment. It is relatively less favorable for severe chronic diseases of the heart and great vessels, complicated by cardiogenic shock, cerebrovascular accident, with discirculatory encephalopathy of II-III stages.
Diseases of the lungs. Neurological disorders can develop against the background of both acute lung diseases (thromboembolism of the main trunk, large, medium and small branches pulmonary artery disease, infarction pneumonia, severe bilateral pneumonia), and chronic nonspecific lung diseases (COPD) (pulmonary emphysema, chronic bronchitis, bronchi-asthma, pneumosclerosis).

Pathomorphologically in the brain of patients who died from acute lung diseases, edema, diapedesic hemorrhages and plasmorrhages, foci of thrombotic and non-thrombotic softening, a combination of focal ischemia in the cortical regions with areas plethora in deeper ones, as well as venous stasis with arterial ischemia. Observed hyaline and annular thrombi in capillaries, dystrophic changes in nerve cells and glia, areas of massive primary karyocytolysis.

Chronic hypoxia is manifested primarily by neurocellular pathology - severe a form of damage to nerve cells with a slowly growing degenerative process in the nucleus and cytoplasm of neurons and glial cells.

In the pathogenesis of damage to the nervous system in lung diseases, the leading role is played by exposure to factors of hypercapnia and hypoxemia, which arise as a result of disorders ventilation and gas exchange in the lungs. When examining the function of external respiration in depending on the nature and severity of the pathology, there is a decrease in vital capacity (VC) up to 2400-1900 ml; maximum ventilation of the lungs (MVL) up to 50-30 liters and coefficient oxygen use (KI02) up to 30-28 ml; an increase in the minute volume of respiration (MRV) up to 8-10 l and the value of oxygen absorption per minute (POg) up to 240-270 ml / min. Shortened the duration of breath holding (Stange-Gench test) up to 10-15 s. Partial pressure of carbon

acid (pCO2) rises to 50-60 mm Hg, the pH value shifts towards acidosis (up to 7.3). Arterial oxygen saturation (HbO2) gradually decreases (up to 80%), changing the level of standard bicarbonates (SB) and buffer bases (BB).

Deep metabolic disorders develop (the amount of fibrinogen increases, α - and γ-globulins and amino acids, lactic acid, ammonia, inorganic phosphorus, decreases the amount of ATP, phosphocreatine, etc.). With thrombosis and pulmonary embolism arteries, an important role belongs to the pronounced hemodynamic disorders that arise due to blockage of the pulmonary vessel and widespread vascular spasm. Following this arterial ischemia and venous plethora of the brain and spinal cord develop, the permeability of the vascular walls increases with the release of erythrocytes perdiapedesem into subarachnoid space and brain matter, which causes massive primary karyocytolysis with the formation of extensive foci of prolapse of neurons in the cortex.

The complexity of the pathogenesis of cerebral disorders (hypoxemia, a drop in blood pressure in a large circle circulation, vascular spasm, homeostasis disturbances) leads to greater types of hypoxia, the frequency of focal brain lesions, the variety of their nature (meningeal syndrome, encephalopathy with seizures, non-thrombotic softening, hemorrhage, etc.).

The appearance of local neurological symptoms (often in the absence of macroscopic focus in the brain) is explained by the phenomenon of capillary ischemia, which does not uniform character, with massive primary karyocytolysis and areas of cell prolapse in bark. Glia is more resistant to hypoxia, although it exhibits rough proliferative-dystrophic reaction, but basically retains its structure.

Clinic. Neurological disorders in the form of a mild headache, photophobia, general hyperesthesia, small vegetative-dystonic manifestations are usually included in the clinical picture of uncomplicated pneumonia and are in one way or another practically in all patients. More pronounced neurological symptoms, which should be regarded as neuropsychiatric complications are observed in about 6-8% of inpatients with pneumonic

monia. These complications are manifested by cerebral, meningeal, focal and vegetative symptoms, including a sharp headache, dizziness, psycho-agitation, soreness when moving the eyeballs, a sharp general hyperesthesia, epileptiform seizures, nystagmus, anisoreflexia, pathological symptoms, sensitivity disorders, changes in blood pressure, pulse, sweating, etc. Encephalopathic and meningeal syndromes develop more often in severe forms croupous pneumonia. Encephalopathic syndrome is manifested by polymorphic nervous mental disorders: intense headache, a feeling of heaviness in the head and congestion in the ears, nausea, psychomotor agitation, mild focal symptoms - nystagmus, revitalization of tendon reflexes, anisoreflexia, increased muscle tone, as well as manifestations of vegetative-vascular dystonia in the form instability of blood pressure, lability of the pulse, acrocyanosis, etc. Often on the side of the somatic pathology in the Zakharyin-Ged zones, hyperesthesia, hyperpathy or hypesthesia are determined. Meningeal syndrome is manifested by moderate headache, nausea, urge to vomit or vomiting, pain with eye movements, photophobia, general hyperesthesia, sometimes psychomotor agitation, tonic symptoms (stiffness of the muscles of the neck, ankylosing spondylitis, etc.). In the cerebrospinal fluid, usually there is only an increase in pressure with an unchanged content of cells and protein. Flow short-lived (3-5 days). The indicated symptom complex (shell syndrome with normal CSF) is called meningism. However, severe pneumonia can be complicated purulent meningitis due to dissemination of the introduction of pathogens (most often pneumococcal kov) into the subarachnoid space. In these cases, there is a sharp deterioration. patients - a new rise in temperature to high numbers and pronounced cerebral, meningeal and sometimes focal symptoms. In the cerebrospinal fluid are observed neutrophilic pleocytosis and hyperalbuminosis. The course is longer (2-4 weeks), prognosis, especially in the elderly, it is not always favorable.

The clinical picture of pulmonary embolism is extremely polymorphic. She can manifest itself in the following neurological syndromes - psychomotor agitation, meningeal, focal lesions of the brain, epileptiform. It should be remembered that thromboembolism can be the cause of the acute development of coma.

Psychomotor agitation syndrome is especially common. For thromboembolism the main trunk or large branches of the pulmonary artery is characterized by an acute development of pronounced psychomotor agitation: patients jump up, try to run, do not recognize their relatives, hallucinate, do not navigate in the environment. With prolonged forms thrombosis and infarction pneumonia, such attacks occur periodically, more often at night time. Usually paroxysms of excitement are replaced by adynamia, a decrease in emotional reactions, lethargy, drowsiness, lethargy. In some cases, periods of motor excitement is accompanied by the appearance of focal symptoms.

Meningeal syndrome is observed in subacute and acute forms of the disease. How the more severe pulmonary heart disease, the usually more meningeals symptoms are detected. Meningeal syndrome in patients with pulmonary artery thrombosis and infarction pneumonia appears with an increase in cerebral edema and serves as a bad a prognostic sign.

Focal brain damage is observed in patients with predominantly prolonged forms of the disease. Of the transient symptoms of nervous systems most often nystagmus or nystagmoid twitching of the eyeballs,

anisocoria, anisoreflexia, pathological reflexes, intentional tremor when performing coordination samples. Often, against the background of a worsening of the patient's condition, disorders occur
speech by the type of sensory and motor aphasia, paresis, paralysis, etc. General cerebral local symptoms in some cases appear several hours earlier than pronounced respiratory
and heart disorders. It should be noted that the favorable dynamics of pulmonary pathology accompanied by a rapid complete or partial regression of neurological symptoms. If
an increase in thrombosis in the pulmonary vessels is accompanied by a deepening of cerebral disorders, then in
in the case of a fatal outcome, it is difficult to identify the immediate cause of death (violation cerebral circulation or process in the pulmonary artery). Spinal cord injury
develops less frequently and mainly in patients with subacute and protracted forms of the disease.
Suppression or complete extinction of tendon reflexes on the lower extremities is noted (especially often knee) and segmental sensory disorders. Is characteristic
positive dynamics of symptoms with the normalization of the functions of the respiratory and cardiovascular
vascular system. Torpidity of the knee reflexes is an important symptom
indicating the severity of the pulmonary process even with a relatively satisfactory general the patient's condition. The progression of spinal disorders is poor
a prognostic sign, as it usually indicates an increase in thrombotic
process in the pulmonary arteries.
Epileptic syndrome is characterized by the development of generalized convulsive
seizure. It can also complicate the course of pulmonary embolism (acute and
subacute forms). Sometimes after an attack in patients, there is an appearance or increase focal neurological symptoms that simulate an acute impairment of the cerebral
blood circulation. It is important to remember that post-seizure symptoms are usually smoothed out in
over the next day.
Polyneuropathic syndrome is rarely observed in patients with prolonged
a form of pulmonary embolism. These disorders are transient and
regress relatively well under the influence of treatment aimed at normalization
blood gas composition and hemodynamics.
The syndrome of "decompensation of the old focus" develops in patients with subacute and prolonged
forms of pulmonary embolism, previous stroke or transient
violation of cerebral circulation. The defeat of the nervous system is fully compensated and up to the onset of pulmonary disease does not appear. Sudden development of focal neurological symptoms (paresis, disorders of sensitivity, speech, etc.) simulates repeated disturbance cerebral circulation. Differential diagnosis based on history data
and clinic (thrombophlebitis, shortness of breath, cyanosis, tachycardia, etc.) and the presence of symptoms,
indicating the localization of the lesion in the basin of the same vessel. Confirms the diagnosis decompensation of the blood supply to the brain against the background of the presence of a cyst in the brain from a previously transferred
stroke, rapid favorable dynamics of neurological symptoms during relief
lethal failure. In patients with atherosclerosis or hypertensive
disease, increasing pulmonary insufficiency can result in a violation of the cerebral circulation - ischemic softening or hemorrhage.

Chronic encephalopathy syndrome develops in patients with COPD with prolonged

pulmonary insufficiency and is characterized by diffuse dull headache, especially intense in the morning, with physical exertion, coughing, increased fatigue, irritability, irascibility and small scattered focal symptoms (hyperreflexia, anisoreflexia, ataxia, trembling fingers of outstretched arms). Sometimes syncope and paroxysms are observed as a cough-fainting syndrome (betolepsy).

Inflammatory diseases of the lungs with the presence of a purulent focus (empyema, bronchiectasis, etc.) can lead to the formation in the brain of a metastatic abscess (single or multiple), which is expressed by an increase in cerebral and focal symptoms, epileptic seizures, etc.

Pulmonary tuberculosis is usually manifested by symptoms of general intoxication and autonomic dysfunction: headache, weakness, sweating, tachycardia, unstable STU HELL. Tuberculous intoxication can lead to meningism (more sharp headache, photophobia, nausea). In case of generalization of tuberculosis infection possible development of tuberculous meningitis, tuberculoma of the brain or spinal cord, tuberculous spondylitis.

Diagnosis of the diseases under consideration presents well-known difficulties, especially in the initial stages of the disease or its atypical course. The reaction of the nervous system in some may be advanced, for example, with prolonged pulmonary thrombosis

In some cases, neuropsychiatric disorders (headache) may come to the fore in some cases. pain, nausea, vomiting, psychomotor agitation, meningeal symptoms, paresis), and pulmonary cardiac disorders (shortness of breath, cyanosis, tachycardia, decreased blood pressure) become pronounced in more advanced stage of the disease.

The appearance of cerebral and focal disorders against the background of pulmonary insufficiency after behind the syndrome of psychomotor agitation (a typical sign of pulmonary embolism or infarction pneumonia), instability of symptoms and their dependence on the dynamics of pulmonary heart failure are the main diagnostic signs of hypoxic encephalopathy. When examining cerebrospinal fluid, only an increase in cerebrospinal fluid pressure (up to 200-300 mm water column and more) without cytosis and hyperalbuminosis.

Treatment of neuropsychiatric disorders in patients with pulmonary embolism is inextricably linked with the therapy of the underlying disease. Extremely severe trunk thromboembolism and the main branches of the pulmonary artery are treated promptly (emergency embolectomy). Availability neuropsychiatric disorders are not a contraindication to surgery, as it usually contributes to their regression. Severe pulmonary embolism as well as embolism intermediate and lobar branches are indications for thrombolytic therapy with simultaneous correction of the hemostasis system (fibrinolysin or activators of endogenous fibrinolysis, antiplatelet agents, thrombolytics, anticoagulants).

Conservative treatment can be combined with surgical prevention of recurrent pulmonary embolism (installation of a filter in the inferior vena cava). Along with drugs, directed at the treatment of pulmonary disease, it is necessary to recommend and means, normalizing functions of the nervous system (metabolism, vitamins, vasotropic drugs).

For the relief of psychomotor agitation, antipsychotics are most often used. Symptomatic treatment of other cerebral disorders - vomiting (droperidol, triftazine), hiccups (metoclopramide, torecan, ethaperazine), pain syndromes (analgesics or lytic mixtures consisting of chlorpromazine or tizercin, diphenhydramine or pipolfen and promedola).

Prediction of neuropsychiatric disorders in pulmonary embolism and
heart attack pneumonia is always serious.
Only favorable dynamics of the underlying disease prevents the transition
transient disorders in persistent organic brain damage. Neuropsychic
disorders in chronic pneumonia, pulmonary emphysema, bronchitis, pulmonary tuberculosis
are usually expressed moderately and with systematic treatment and adherence to
recommendations for
the regime, most of the patients remain able to work.
Liver disease. Diseases of the liver and biliary tract are often complicated by nervous
mental disorders. The clinical manifestations of the latter are determined by the form,

the severity and duration of the underlying disease - cholecystitis, gallstone disease,
cholangitis, cholepancreatitis, obstructive jaundice, liver cirrhosis, etc.
Asthenic manifestations are most often observed I disseminated head injury
and the spinal cord, and sometimes individual peripheral nerves, i.e., encephalopathy syndromes,
encephalomyelopathy and polyradiculoneuropathy.
Liver disease can be one of the causes of optic neuritis.
The pathogenesis of damage to the nervous system is caused by disorders of the detoxification
liver function and various types of metabolism - protein, lipid, carbohydrate, water
electrolyte, vitamin. Hyperglobulinemia develops, education is disrupted
fibrinogen, prothrombin, vitamin metabolism (in particular, vitamins C and K), which causes
change in the coagulating properties of blood and the development of hemorrhagic
syndrome. Acid-
the main state of the blood shifts towards acidosis, the alkaline reserve of blood decreases.
Water-salt metabolism and the course of all redox processes in
nervous system. In jaundice, the toxic effect is exerted by the accumulation of bilirubin in the
blood and
bile acids, hepatoportal disorders - ammonia. One of the links in pathogenesis can
be a violation of the function of other organs, which is confirmed by the frequent development
hepatocardial, hepatolienal, or hepatorenal syndromes.
Neurasthenic syndrome occurs in two variants - hypersthenic
(irritability, increased excitability, emotional lability, incontinence,
anger, excessive mobility) and asthenic (physical and mental fatigue,
resentment, suspiciousness). The mood is unstable. The dream is superficial, disturbing. Usually
complaints of headache, feeling of heaviness in the head, dizziness, as well as unpleasant
sensations in the region of the heart, palpitations, disorders of the genital area (impotence,
violation
menstrual cycle, etc.). Some patients with sensitive character traits with
the disease of obstructive or parenchymal jaundice experience severe itching and feeling
burning sensation in any part of the body, causing the desire to “take everything off yourself,”
sensations
the passage of electric current and hot waves through the body, “twitching and gurgling in the
area
abdomen ”,“ vibration in the spine ”, etc.
Hepathogenic encephalopathy is manifested by headache, dizziness, sometimes
nausea and slight diffuse symptoms (nystagmus, cranial nerve paresis,
coordination disorders, hyperkinesis, anisoreflexia, pathological reflexes). Far away
advanced stage of liver cirrhosis (ascites, splenomegaly), more severe forms are observed
portal encephalopathy with impaired consciousness. Due to the formation of anastomoses
between the system of the vena cava and portal veins, ammonia and other toxic products from
gastrointestinal tract (normally they pass through the hepatic filter and undergo

detoxification). Against the background of polymorphic neurological symptoms, there are often psychomotor agitation, as well as hyperkinesis (more often of the type of choreoathetosis or fluttering
tremor). Psychomotor agitation can turn into stunning, stupor and then comatose condition. With an acute increase in the difficulty of blood flow from the portal vein to the liver, the likelihood
the appearance of neurological disorders is increasing. Ammonia formation is proportional to the content of proteins in the intestine, therefore, with bleeding from varicose veins Esophageal portal encephalopathy develops especially often.
The cause of the development of acute toxic-discirculatory encephalopathy (ETSE) is often is obstructive jaundice. In milder cases, ETS is manifested by severe apathy, adynamia, headache, diffuse decrease in muscle tone. With an average OTDE severity is joined by focal neurological symptoms (anisoreflexia, pathological reflexes, impaired cranial innervation, reflexes of oral automatism, meningeal signs), and in severe OTDE, in addition to an increase in organic cerebral symptoms, signs of spinal cord injury appear (paresis of the legs, muscle tone, plantar and Achilles reflexes). There is a certain correspondence between the severity and duration of autointoxication with bilirubin, with one hand, and the depth of neurological disorders - on the other.
Symptoms of polyradiculoneuropathies associated with liver pathology have the following features: at the onset of the disease, typically limited lesions of one or two roots or one nerve, and only after a few weeks or months the process spreads to other nerves and gradually captures all limbs - arises polyneuropathic syndrome. Depending on the clinical manifestations, they are distinguished

sensitive, motor and mixed forms.
The most common sensitive (sensory) form, which is characterized by pain, paresthesia, disorder of superficial sensitivity in the distal regions extremities and vegetative-vascular disorders (cold hands and feet, hyperkeratosis, hyperhidrosis, discoloration of the skin). The degree of movement disorders varies from slight weakness to relatively deep paresis. On the hands there is a predominant damage to the radial, on the legs - peroneal nerves.
Sometimes, with diseases of the liver and biliary tract, various visceral disorders - hepatocholecystocardial and hepatorenal syndromes. More often in total, Botkin's cholecystocoronary syndrome is observed, which manifests itself recurrent cardialgia against the background of a violation of the diet, dyspeptic symptoms, jaundice, etc.
e. In elderly patients suffering from atherosclerosis involving cerebral vessels and heart, liver and biliary tract pathology can be a risk factor for stroke or myocardial infarction.
Diagnostics is based on clinic data and additional research methods, among which are the most important EEG indicators (the appearance of three-phase waves is one of the earliest
signs of the transition of hepatogenic encephalopathy into a coma) and EMG (decrease in myopotential volumes, rare fibrillations, changes in the speed of conduction along the nerves are detected in subclinical forms of hepatogenic polyneuropathies). In the spinal an increase in pressure is detected. To assess the state of the brain and liver CT and MRI are essential.
Treatment. Prescribe detoxification, dehydration, lipotropic and fortifying agents - reopolyglucin, diacarb, cerebrolysin, retabolil, glutamic acid, methionine, pancreatin, intravenous glucose with insulin, isotonic

sodium chloride solution, blood, plasma or blood substitutes (polyglucin, etc.), as well as a diet with restriction of salt and proteins. To reduce the activity of the intestinal bacterial flora, under by the action of which ammonia is produced, sometimes short courses of antibiotics are prescribed
or sulfa drugs. Excitement and restlessness can be
stopped by the introduction of antipsychotics. When bleeding occurs, askorutin is prescribed, vicasol, calcium gluconate, dicinone, etc. A number of diseases of the liver and biliary tract are treated
promptly (cholecystitis, cholelithiasis, liver cirrhosis, etc.). The appearance of a nervous mental disorders, as a rule, should not be a contraindication to surgical
intervention.
The prognosis is more favorable with the development of neuropsychiatric disorders on the background
cholecystitis, cholangitis and gallstone disease, less - against the background of liver cirrhosis , sportocaval anastomosis, splenomegaly, ascites, bleeding from the veins of the esophagus.
Diseases of the pancreas. In hyperglycemic conditions due to
diabetes mellitus, various symptoms are observed: headache, dizziness, general
weakness, memory loss, pruritus, sensitivity disorders, movement disorders
spheres. The following syndromes are distinguished: neurasthenic, encephalopathic, polyneuropathic, autonomic polyneuropathy, neuralgia and neuropathy of individual nerves, most often facial, as well as hyperglycemic (diabetic) coma.
Diabetic encephalopathy is characterized by headache, memory loss, and
attention, nystagmus, impaired pupillary reactions to light and convergence, paresis
facial and oculomotor nerves, etc. Severe forms of diabetes mellitus, especially in individuals elderly, may be complicated by a stroke. Pathological studies
suggest that an important role in the pathogenesis of encephalopathy and stroke in diabetes belongs to the macroangiopathy inherent in diabetes mellitus, i.e., damage to arterioles, precapillaries vascularizing the cortex, subcortical formations and the brain stem.
It should be emphasized the frequency of non-thrombotic softening, which is explained by the excessive
accumulation of carbon dioxide. The latter, by expanding the cerebral vessels, causes a regional fall
Blood pressure, that in the presence of an increased demand for oxygen in the brain tissue of diabetic patients and
leads to softening without the formation of a blood clot in the cerebral vessel.
Diagnosis of strokes occurring against the background of diabetes mellitus has certain difficulties. The neurological picture is masked by adynamia accompanying diabetes, impaired sensitivity of the peripheral type, anisoreflexia, areflexia, etc.

in many patients in the acute period of a stroke, the course of diabetes also worsens:
blood sugar levels to unusual values for this patient, acetonuria appears and
etc. The coma in these patients is characterized by a long duration (from 1 to 3-5-8 and more than 20 days).
Disorder
cerebral
circulation
maybe
complicate
development
hyperglycemic coma. All this in cases of stroke with loss of consciousness, stupor, deafening

makes it difficult
differential
diagnostics
with
diabetic
(hyperglycemic) coma.
Hemorrhagic stroke in patients with diabetes mellitus develops more often when combined with hypertension or as a complication of diabetic coma, apparently as a result
toxic effects on cerebral vessels of products of impaired metabolism, in particular
ketone bodies.
Patients with diabetes mellitus often have polyneuropathic disorders that
proceed with a predominance of sensitive, autonomic and motor symptoms.
The sensitive form is manifested by paresthesias, pain and a slight decrease
superficial sensitivity, impaired coordination of movements, motor - not rough
flaccid paresis of the limbs and muscle atrophy, more pronounced in the proximal
departments. For diabetic polyneuropathies, peripheral
vegetative failure. The most common manifestation of PVI is orthostatic
hypotension, fixed tachycardia, nocturnal diarrhea, pelvic disorders. Also meet
neuropathy and neuralgia of individual nerves, especially often of the facial.
Hyperglycemic diabetic coma often develops gradually - over
several hours or days. Headache, dizziness, thirst, and polyuria appear.
Patients become lethargic, drowsy, apathetic, indifferent. Dry skin is noted with
traces of scratching. If untreated, the precomatose state turns into a coma: consciousness
is completely lost, blood pressure falls, the pulse becomes weak, frequent, there is an odor
acetone from the mouth. Pupils narrow, corneal, abdominal and tendon reflexes gradually
decline. With coma of 1-11 degrees, pathological reflexes are usually determined.
Treatment. In the acute period of stroke against the background of diabetes mellitus,
insulin: by normalizing carbohydrate metabolism and reducing hypoxia, it improves brain
nutrition
govy tissue. To avoid hypoglycemia, insulin is best administered in divided doses. Dysfunction
coagulation and anticoagulation system of the blood, the frequency of non-thrombotic softening
and
the presence of foci combined by the nature of the pathological process requires caution when
prescribing anticoagulants to patients with diabetes mellitus. In cases of diabetic coma
shows immediate intravenous administration of insulin, hypoglycemic - glucose.
Surgical interventions are used for purulent pancreatitis, pancreatic necrosis, tumors
pancreas (insulinoma).
The course of the syndromes of diabetic polyneuropathy, diabetic and hypoglycemic
encephalopathy is often recurrent with improvement under the influence of complex treatment.
Kidney disease. Acute renal failure (acute glomerulonephritis,
post-abortion sepsis, poisoning, trauma, etc.) and long-term kidney disease
(chronic glomerulonephritis, pyelonephritis, urolithiasis) in the stage of subcompensation and
especially decompensation can cause a variety of neuropsychiatric disorders -
polyneuropathy, encephalopathy, dyskalemic paralysis, uremic coma, etc.
Pathomorphologically, in the brain, a typical picture of toxic
encephalopathy with a combination of vascular and parenchymal cell changes (edema,
angionecrosis, diapedetic hemorrhages, degenerative changes in cells, etc.).
Pathogenesis of neurological disorders in kidney disease in the stage of decompensation
due mainly to intoxication caused by azotemia.
However, syndromes such as asthenic, renovisceral, moderate
encephalopathy, may complicate subcompensated kidney failure, when the phenomena
there is no azotemia yet. Loss of sodium and chloride, mild hypo- and hyperkalemia and

hypoalbuminemia lead to a decrease in the colloid-osmotic pressure of the blood, an increase vascular permeability with the development of edema in the brain, spinal cord and peripheral nerves,
diapedetic hemorrhages and plasmorrhages, and later on to changes in nerve cells, conductors, nerve plexuses and peripheral nerves, as well as impaired contractile

muscle function.
Toxic damage or mechanical compression of the branches of the vegetative plexus of the kidneys can lead to pathological irritation of the pain impulse in spinal nodes and segmental apparatus of the spinal cord and the appearance of pain and hyperesthesia
in the renal zones of Zakharyin-Ged, pain in the heart (renocardial syndrome), abdomen (renovisceral syndrome) or exacerbation of lumbosacral sciatica. Far away the advanced stage of renal failure, the combined
toxic effects of azotemia and the whole complex of metabolites of average molecular weight, metabolic acidosis, impaired protein and water-electrolyte balance, especially hyperkalemia and hypercreatininemia, as well as arterial hypertension.
Neurasthenic syndrome in the initial period of kidney disease is manifested symptoms of hypersthenia (irritability, irascibility, instability of mood, sleep disorders), later (stage of subcompensation and decompensation) begin symptoms of hyposthenia predominate (increased fatigue, absent-mindedness, resentment, tearfulness). All disorders usually develop against the background of back pain, edema, dysuric disorders, etc.
Algic syndrome is characterized by pain that is localized in the lower back at the level segments Tshh-L I on one (renal colic) or on two (nephritis) sides, wear permanent or paroxysmal character, do not always subside in the supine position and can spread to inner thigh and groin fold.
In the study of sensitivity, in the area of the affected segments it is determined more often total hyperesthesia or hyperpathy. The severity of symptoms of tension of the nerve trunks insignificant. Loss symptoms in the motor and reflex areas are usually absent.
It should be borne in mind that kidney pathology can cause an exacerbation of lumboischialgic syndrome in patients suffering from spondylosis deformans and osteochondrosis of the spine, which accordingly changes the clinical picture of the disease.
With polyneuropathic syndrome, moderate sensory, autonomic and reflex disorders: pain, burning, numbness, acrocyanosis, hypoesthesia or hyperesthesia in distal parts of the arms and legs (mainly in the feet), sometimes a decrease in Achilles reflexes. Severe forms with paralysis and paresis of the limbs are currently due to improved treatment for renal failure is rarely seen.
Renocardial syndrome is characterized by prolonged aching pain in the left side of the chest, which are combined with pain in the lower back and roglycerin. Electrocardiographic study does not reveal significant abnormalities from the norm. The pains regress with the relief of renal failure. However, patients with ischemic heart disease, paroxysm of renal pain can provoke attacks of angina pectoris.
Reno-abdominal syndrome develops at the height of an attack of urolithiasis and manifested by pain in the epigastrium, nausea, belching, heartburn (not associated with food), hiccups, loss of appetite and other dyspeptic disorders. Maybe mimic diseases such as cholecystitis, appendicitis, pancreatitis, gastritis, ulcerative disease.
Acute encephalopathic disorders usually occur against the background of a sharp increase in

renal failure. Patients develop cerebral (headache,
dizziness, apathy, or, conversely, agitation), as well as meningeal and small
focal symptoms (anisocoria, horizontal nystagmus, muscle hypotension, increased
reflexes, etc.). The most severe disorders are observed in the oligoanuric stage.
illness, when a sharp psychomotor agitation can be replaced by somnolence, and in
further - soporous or coma. Expansion is observed on the fundus
veins or even congested nipples. In the cerebrospinal fluid, an increase in pressure is noted (up to 250-300 mm water column) with normal composition or slight pleocytosis and hyperalbuminosis.
With a significant increase in hypo- or hyperkalemia, they often develop
dyskalemic paralysis - weakness of the muscles of the arms, legs and trunk, which can reach
degree of complete immobility, as well as respiratory and cardiac disorders
(shortness of breath, bradycardia, arterial hypotension, etc.). Tendon reflexes and muscle
the tone is reduced. Hypokalemic paralysis is more pronounced in the proximal arms and

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legs, rarely capture the muscles of the face, hyperkalemic - usually spread to
muscles of the face, pharynx and larynx.
Clinical manifestations of acute encephalopathy and dyskalemic paralysis are usually
gradually disappear as the kidney failure is compensated. But in cases of prolonged and
severe course of renal disease, neuropsychiatric disorders (headache, general
weakness, decreased memory and attention, increased reflexes of oral automatism, revitalization
tendon reflexes, pathological reflexes, etc.) become persistent, that is, it develops
chronic dysmetabolic encephalopathy. Sometimes it can proceed according to the type
pseudotumorous syndrome (headache, nausea, epileptic seizures, spontaneity,
workload).
Cerebral circulation disorders (crises, transient disorders, strokes) more often
in total are observed in chronic nephritis complicated by arterial hypertension.
Uremic coma is characterized by itching, scratching of the skin, ammonia odor
from the mouth, hiccups, vomiting, myoclonus, and often convulsive paroxysms. In a shallow
coma stage, all tendon reflexes are revitalized, and corneal and pharyngeal usually narrower
reduced. Bilateral pathological pyramidal reflexes are caused.
It should be borne in mind that the course of chronic renal failure (CRF) for
the last two decades have changed somewhat due to the development of effective methods
treatment. This is largely related to the terminal stage of chronic renal failure -
uremia. Hemodialysis and
kidney transplantation can prolong the life of patients for many years.
Neurological disorders in CRF should be considered as part of the overall response
the body for impaired renal function. The influence of a complex of factors of intoxication, including
anemia, can manifest itself at different times depending on the sensitivity of certain
departments of the nervous system. This underlies a certain staging in development.
neurological disorders in the terminal stage of chronic renal failure. The clinical dynamics are as follows.
Stem symptoms initially appear, then tendon symptoms gradually decrease
reflexes and strength of leg muscles; tendon reflexes remain on the hands for some time
elevated, sometimes with the presence of pathological signs; followed by weakness and
suppression of tendon reflexes on the hands against a background of even greater damage to the lower
extremities - the appearance of deep lower atrophic paresis and paralysis with the absence
tendon reflexes and distal sensory disorders. This process
occurs against the background of progressive encephalopathy, as evidenced by the increasing

changes in the emotional and mental sphere, asterixis, multiple myoclonus.
It is known that phylogenetically younger parts of the nervous system are more sensitive to both hypoxia and intoxication. Therefore, with a mild degree of intoxication first of all, the cortical level suffers, and the functional state also changes limbic-reticular complex. In this case, the symptoms of encephalopathy are revealed. At increased intoxication, the initial excitement is replaced by a sharp weakness, increased fatigue, lethargy, lethargy, forgetfulness, drowsiness. Further an increase in intoxication, spinal symptoms are more and more revealed - a decrease muscle strength, muscle tone and tendon reflexes and sensory disorders in legs. These symptoms may depend on both the violation of the descending influences of the reticular formation, and from the direct effect of intoxication on the spinal cord and peripheral nerves, which leads to a decrease in pyramidal symptoms and an increase in atrophic paresis.
The widespread introduction of chronic dialysis has led to the identification of a new form neurological pathology - dialysis encephalopathy, the leading manifestation of which is dementia Until now, the pathogenesis of brain damage in such cases; an excess of aluminum in the water used to make dialysis. The presence of a permanent shunt sometimes leads to the development of median tunnel neuropathy.
a dinosaur nerve in the carpal tunnel.
Regularity of regression of symptoms of focal lesions of the nervous system after successful transplantation of the kidney in the opposite direction to the dynamics of their growth: the symptoms initially disappear lesions of peripheral nerves and spinal cord, then there is a regression of symptoms renal encephalopathy. The duration of the recovery process is up to 2-3 years. Remaining by the expiration of this period, the symptoms are hardly reversible and they have to be attributed to persistent dual violations. It should be remembered that patients with a transplanted kidney often have cytomegalovirus infection.

Treatment. Therapy should be carried out taking into account the form and stage of renal disease, clinical manifestations and pathogenesis. Usually it is aimed primarily at compensation renal failure. Uremia with hypercreatininemia and hyperkalemia and an increase in cerebral, meningeal and general focal symptoms requires mandatory use hemodialysis (peritoneal dialysis) or hemosorption (which are often a preparation to kidney transplant). With urolithiasis, pyelonephritis, hydronephrosis, kidney injuries indications (ineffectiveness of conservative therapy, etc.), an operative intervention, after which there is a complete or partial regression of neuropsychic violations. Treatment of emotional and mental disorders in the structure of encephalopathic the syndrome is carried out using tranquilizers, antidepressants, nootropics, fortifying drugs, etc. Prescription of any drugs should carried out only after consultation with a nephrologist, given that some patients have severe renal failure.
The prognosis is determined by the form and severity of the underlying disease and partly by the characteristics complications. Stable compensation of neurological symptoms can be achieved only with eliminating renal failure.
Nevra-walled

polyneuropathic,
renocardial, renovisceral, encephalopathic disorders can completely
regress if they are due to acute and relatively mild kidney disease
or long-term ongoing diseases in the stage of subcompensation or compensation.
The increasing or recurrent course of these syndromes is observed in patients with chronic glomerulonephritis or pyelonephritis in the stage of decompensation.
Connective tissue lesions. Lupus erythematosus, polymyositis, dermatomyositis,
periarteritis nodosa, scleroderma, temporal arteritis, thromboangiitis obliterans
often accompanied by neuropsychiatric disorders - encephalopathic syndromes,
polyneuropathic, myasthenic, myopathic and some others.
The pathogenesis of these disorders is due to autoimmune degenerative inflammatory
changes in the membranes of the brain and spinal cord and blood vessels.
Rheumatism can be complicated by cerebral disorders with predominant
defeat of the subcortical nodes - small chorea. Essentially rheumatic brain injury
exhausted by small chorea. The previously existing concept of "cerebral rheumatic vasculitis" as everyday causes of damage to the nervous system proved to be untenable. Defeat
cerebral vessels with rheumatism - a rarity.
Among the causes of cerebral strokes, both primary vasculitis and
and vasculitis in connective tissue diseases. Syndrome attracts special attention
Snedonna associated with antiphospholipid factor is perhaps the most common cause
ischemic strokes in young patients.
Temporal arteritis (Horton's disease) is characterized by sharp local pain in
temporal region, which may be accompanied by trismus. Pathological basis
diseases - giant cell arteritis of the temporal artery. Palpation reveals
thickened and painful temporal artery. Sometimes it is already visible upon examination. Often in
the process on the side of the lesion involves the optic nerve (decreased visual acuity; on the eye-day - a picture of ischemic neuritis). Characterized by a sharp increase in ESR.
Neurolupus is a neurological manifestation of systemic lupus erythematosus. Most often on
the background of general malaise, fever, headache, dizziness is observed
damage to the peripheral nervous system (neuropathy, polyneuropathy, etc.), but sometimes
other levels of the nervous system are also involved with the development of encephalopathy syndrome,
myelopathy, encephalomyelopathy.
Periarteritis nodosa is pathomorphologically characterized by the defeat of small
arteries with the development of dense nodules. Since the process may involve blood vessels
almost all organs and tissues, including the nervous system, the clinical manifestations
diseases are extremely varied. It occurs at any age, but somewhat more often in men
30-50 years old. At the onset of the disease, fever, diffuse pain and polymorphic
rashes on the skin. In the course of the vessels, dense, painful on palpation are felt
nodules. Already at an early stage of the disease, internal organs are involved - the spleen, liver, kidneys,
gastrointestinal tract, which is manifested by abdominal pain, hematuria, intestinal
bleeding and other symptoms. Over time, patients acquire a characteristic

appearance - salty-pale skin color against the background of general exhaustion. Almost in
in all cases, various neurological disorders are observed - neuropathies,
polyneuropathies, encephalopathy, myelopathy, acute vascular disorders (subarachnoid,
subdural, parenchymal hemorrhages). Most often, the peripheral nervous
system in the form of multiple mononeuropathies.
Diagnosis is based on clinical presentation (combination of fever, skin disorders,

lesions of the kidneys and peripheral nerves) and data from additional studies (hypergammaglobulinemia, leukocytosis with a shift to the left, high ESR).
With polymyositis, edema, lymphoid cell clusters are detected in muscle tissue, destruction of fibers, etc.
The clinical picture is characterized by the appearance of diffuse or limited pain in muscles, mainly in the proximal extremities, subfebrile condition, general fatigue. The muscles are a little swollen, painful on palpation. Develop gradually changes in internal organs (heart, lungs, gastrointestinal tract), skin (depigmentation, swelling), the nervous system (peripheral nerves, membranes, spinal cord, brain stem, etc.) and muscles. Therefore, the disease can occur with syndromes polyneuropathy, radiculoneuropathy, myelopathy, encephalopathy, myopathy, myasthenia gravis. V
blood - leukocytosis, hyperglobulinemia, increased ESR, as well as the activity of aminotransferases and
aldolases.
Differential diagnosis is most often carried out with other forms polyneuropathies and myopathy. In unclear cases, the diagnosis is aided by the data muscle biopsy.
Treatment. All forms of collagenosis are treated with long repeated courses anti-inflammatory (indomethacin, voltaren, brufen, reopirin, delagil), antihistamines (suprastin, pipolfen, diazolin) and hormonal
(prednisone, urbazone, dexamethasone)
drugs in various combinations. The inclusion of corticosteroids is mandatory in severe the course of the disease.

EPILEPSY.
According to the definition of WHO experts, epilepsy is a chronic disease of the head brain of various etiology, which is characterized by repeated epileptic seizures, resulting from excessive neural discharges, and is accompanied by a variety of clinical and paraclinical symptoms.
It is necessary to strictly distinguish between an epileptic seizure and epilepsy as a disease. Single, or, according to the terminological dictionary of epilepsy, occasional epileptic seizures or epileptic reaction, in the terminology of domestic researchers, having arisen in a certain situation, they are not repeated in the future. An example is some cases of febrile seizures in children. Epilepsy should not include repetitive epileptic seizures in acute cerebral diseases, such as disorders
cerebral circulation, meningitis, encephalitis. At the suggestion of S.N.Davidenkov, in in such cases, it is advisable to use the term "epileptic syndrome".
Etiology. For the development of epilepsy, a persistent focus of epileptic activity due to organic brain damage. In the same time
epileptization of neurons, that is, a special state of neurons that determines "convulsive readiness"
the brain in the foci of its organic lesion and the degree of epileptic influence of these foci on brain structures, depends on the premorbid characteristics of the body and, in particular, on the epi-
leptic predisposition of a genetic or acquired nature, which determines a greater likelihood of an epileptic seizure in the patient with brain damage.
The significance of the genetic factor is most clearly seen in typical absences. (short-term loss of consciousness followed by amnesia) inherited in an autosomal dominant type with incomplete gene penetrance, with primary generalized epilepsy, starting in childhood; the role of the genetic factor in partial seizures is less pronounced,

however, as noted, in this case, among the close relatives of patients, seizures
are more common than the population average.
Exogenous factors affecting the development of the disease include perinatal
and postnatal neuroinfections, neurotoxicosis and traumatic brain injury, having
highest value. This does not exclude the role of other factors - intrauterine, vascular,
toxic. As for perinatal pathology (from the 27th week of fetal life to the 7th day of life
newborn), then traumatic factors play the greatest role (inadequacy
the size of the fetal head and pelvis, the use of obstetric aids, etc.) and anoxic (asphyxia
fetus during prolonged childbirth, entanglement of the fetal neck with the umbilical cord, etc.).
Pathogenesis. In the pathogenesis of epilepsy, they are important as changes in functional
the state of some neurons in the area of epileptogenic lesion (epileptogenic focus),
the combination of which makes up the epileptic focus, and the interaction features
populations of epileptic neurons. Electrical activity of epileptic neurons
characterized by the occurrence of paroxysmal depolarization shift (PDS)
membrane potential, followed by a phase of hyperpolarization. In the same time
the neurons surrounding the epileptic focus are in a state of constant hyperpolarization,
which prevents the spread of epileptic activity from the focus.
The most important pathophysiological mechanism of epilepsy is hypersynchronization
activity of neurons, i.e., simultaneous coverage of a large number of
epileptic and adjacent shoot neurons. At the heart of hypersynchronization, which determines not
only the formation of an epileptic focus, but also the effect of the latter on the brain, can lie
various
mechanisms
-
the rise
synaptic
conductivity,
epaptic
(extrasynaptic) effect of the electric field of an epileptic neuron on neighboring cells,
in-phase discharges, etc.
There are three concepts to explain the epileptization of neurons: 1) membrane disruption
neuron or its metabolism; 2) changes in the environment surrounding the neuron; 3) pathological
changes in the population of neurons associated with a deficiency of inhibition processes. All
violations
are detected at the metabolic and neurotransmitter levels, as well as at the structural level.
In the epileptic focus, the loss of dendritic spines by neurons, reduction
dendritic endings, their varicose veins, glial proliferation, selective prolapse
GABAergic terminals. These changes are considered as morphological manifestations.

partial neural; afferentation, which may explain the increase in spontaneous neural
activity and hypersensitivity of postsynaptic receptors.
The variability of the membrane potential of epileptic neurons and its rhythmic
fluctuations can be caused by a violation of maintaining an adequate concentration gradient
ions K, Na, Ca, Mg on both sides of the nerve cell membrane, as well as a change in the
distribution
ions inside the cell (in particular, the accumulation of K ions in mitochondria). Also identified
violations of energy mechanisms: a decrease in the activity of cytochrome oxidase, which leads
to
decrease in the formation of ATP, disruption of the functioning of the Krebs cycle.
Currently, the role of biogenic amines in the onset of epilepsy has been established.

A decrease in the content of DOPA, dopamine and norepinephrine in epileptogenic foci was revealed, and
also a weakening of the reactivity of the DOPA - dopamine - norepinephrine system, which can have
influence on the epileptic activity of the brain, causing system failure
antiepileptic protection. A violation of
serotonin metabolism, in particular a decrease in the content of 5-hydroxyindoleacetic acid. except
In addition, in the focus of epileptic activity, a decrease in the content of those involved in the cycle
Krebs amino acids - glutamic and gamma-aminobutyric (GABA). The latter is known to be has a pronounced anticonvulsant effect. Apparently seizures like
the component of hypovitaminosis B6 (pyridoxine) is associated precisely with a lack of GABA, since it
formed with the participation of pyridoxine phosphate from glutamic acid under the action decarboxylase. Pyridoxine deficiency epilepsy is an example of biochemical disorders, which may be due to genetic factors or exogenous lesions, and
increase the epileptic readiness of the brain. There is also evidence of a possible role excitatory neurotransmitter glutamate in the mechanism of triggering epileptic seizures.
The inhibitory mediators taurine and glycine may also have a certain effect, the content which in the epileptic focus is reduced.
The spread of the influence of the epileptic focus on the brain can be promoted and extrafocal factors. In case of epilepsy, they form a special functional state of the brain, which is referred to as increased epileptic readiness, "convulsive reactivity"
brain, etc. It is the increased epileptic readiness of the brain that contributes to the formation epileptic focus and the spread of its influence on other parts of the brain.
According to P.M.Sarajishvili, an epileptic focus cannot cause an epileptic
seizure, not covering the functional system, in which, apparently, by all means are involved specific and nonspecific nuclei of the optic tubercle. In the mechanism of generalization paroxysmal activity from a primary cortical epileptic focus
have “generalizing formations of the subcortex,” which include the structures of the limbic brain, medial thalamus, subthalamus and reticular formation of the midbrain.
Structures and mechanisms that ensure the spread of an epileptic discharge from places of origin, form the epileptic system.
At the same time, the brain contains structures that inhibit epileptogenesis and counteracting the spread of the influence of the epileptic focus on the brain. To such formations include the caudate nucleus, the caudal nucleus of the pons, the lateral nucleus of the hypothalamus,
orbitofrontal cortex, cerebellum, area of the solitary bundle. Their influence is carried out as in in the form of a direct inhibitory effect, and in the form of return inhibition, which develops under the influence of an epileptic focus. This system is also described as a system of negative feedback, working through the population of intercalary neurons. Intercalary neuron, possible awakened by impulses from an epileptic focus, can interrupt the flow of these impulses by hyperpolarizing an epileptic neuron.
Recurrent
inhibition of neurons located on the periphery of the epileptic focus. Structures and the mechanisms that prevent epileptogenesis are referred to as the antiepileptic system.
The facts revealed in recent years confirm the assumption that
desynchronizing devices of the brain have an antiepileptic effect, and synchronizing emitters can promote the activation of epileptic foci. At least,
electro-polygraphic studies of night sleep in humans have revealed that ac-

the activation of the epileptic focus in this case occurs in the second stage of "slow" sleep, that is, in

the carotid spindle stage, during which thalamocortical recruitment is facilitated and accordingly, the spread of epileptic activity is facilitated. And vice versa, REM sleep suppresses the generalization of epileptic activity, which to a large extent also applies to delta sleep.

Schematically, the sequence of "events" in the development of generalized tonic clonic seizure can be represented as follows. Under certain conditions the amplitude and frequency of epileptic neuron discharges begins to increase. After the intensity of charges will exceed a certain threshold, epileptic activity overcomes inhibitory effects of surrounding neurons and spreads to nearby areas of the cortex subcortical structures (basal ganglia, thalamic nuclei, stem reticular formation). The activity of the latter spreads both in the rostral direction, additionally exciting neurons in the epileptic focus and other areas of the cortex, and in caudal (along the corticospinal and reticulospinal tracts) to spinal neurons.

The spread of excitation to the subcortical, thalamic and stem nuclei corresponds tonic phase of the seizure, accompanied by loss of consciousness, pronounced vegetative manifestations (hypersalivation, mydriasis, tachycardia, arterial hypertension, sometimes short-term respiratory arrest), characteristic high-amplitude discharges (peaks) on EEG over the surface of the entire cortex. However, the subsequent activation of the diencephalic the cortical inhibitory system periodically interrupts the spread of epileptic discharge, which corresponds to the transition of the tonic phase to the clonic; while the EEG shows rhythmic discharges are transformed into “peak-wave” complexes. Clonic twitches and causing them discharges become less pronounced and more rare and, finally, disappear, which reflects "Depletion" of neurons of the epileptic focus, manifested in some cases also Todd's post-attack palsy. At the same time, diffuse slow waves.

If the epileptic focus cannot be stably blocked, which usually manifests itself manifestation of epileptic seizures, a number of new pathophysiological mechanisms of formation and development of epilepsy as a disease. The most important of these is the emergence secondary epileptic foci. In their genesis, the mechanism of secondary recurrent generalization, i.e. excitement under the influence of a cortical epileptic focus generalizing apparatus of the subcortex, followed by a secondary "reflection" of excitation in bark. This "reflection" most often occurs in the symmetrical point of the cortex of a "healthy" hemisphere. Transcallosal influences also play a certain role. Secondary epilepsy a tic focus, in its formation, passes the stage of a dependent focus, and later becomes independent of the influence of the primary focus and plays the role of an independent epileptogenic factor.

Another important mechanism of “epileptization” of the brain is a violation of the information function neurons that undergo significant restructuring. As a result, they recode afferent stimuli in a specific epileptic manner.

The mechanism of occurrence of primary generalized epilepsy is not clear enough, with which fails to establish an epileptogenic lesion, seizures do not have a focal onset, and epileptic activity is characterized by generalized synchronous and symmetrical discharges. The significance of deficiency of activating factors in the genesis of this form of epilepsy

influences of the brain stem, increasing the excitability of the cortex and increasing the tendency to generate
undamped vibrations. However, the question of the pacemaker of epileptic activity and the role of the organ-
of brain damage in primary generalized epilepsy remains open.
The presence of dysontogenesis in this form of epilepsy (impaired development of the body during
prenatal period or early childhood) in the form of ectopia of nerve cells; this is
indicates that primary generalized epilepsy is based on structural
changes in the brain, and its mechanism, apparently, is reduced to denervation hypersensitivity
ectopic neurons acquiring epileptic properties.
Clinic. The main clinical symptom of the disease is epileptic
seizures. In the International Classification of Epilepsy, generalized and
partial (focal) epileptic seizures. Generalized seizures are accompanied by
loss of consciousness, vegetative manifestations, the severity of which depends on

whether they are accompanied by convulsions or not. Distinguish between convulsive and non-convulsive forms.
Generalized epileptic seizures on the EEG are characterized by bilateral
symmetric and synchronous epileptic discharges.
Convulsive forms of generalized epileptic seizures include a large
epileptic seizure and its varieties. Large epileptic seizure ("large
disease ", grandmal) is characterized not only by loss of consciousness and autonomic disorders (mydriasis, hyperhidrosis, tachycardia, etc.), but also convulsions involving both sides of the body
simultaneously. First, tonic and then clonic convulsions appear. In more rare
cases of convulsions can be only tonic or only clonic (the so-called
non-deployed convulsive seizure). As a rule, due to the involvement in the process of respiratory muscle apnea occurs, patients bite their tongue, involuntary urination is observed
(if at the time of the seizure there was urine in the bladder), when falling, patients often receive traumatic injury. The seizure ends in an epileptic coma, turning into sleep,
on exit from which amnesia is noted; patients experience a feeling of weakness, pain in muscles, weakness. In other cases, after a seizure, psychomotor agitation may develop,
twilight state of consciousness and other mental disorders.
A large epileptic seizure on the EEG is characterized by the appearance of rhythmic
discharges with a frequency of 8-14 per second of low amplitude, followed by an increase in the last
(recruitment) up to 100-200 μV (tonic stage of seizure) and transition to discharges of the type of peak
wave and polypeak wave (clonic stage).
The second type of generalized epileptic seizure is absence. He
characterized by switching off consciousness without seizures and without falling of the patient and vegetative
manifestations (paleness or redness of the face, mydriasis, salivation, etc.). EEG -
paroxysms of discharges, having the structure of peak-wave complexes with a discharge frequency of 3 per second.
The described type of seizure is called "simple absence," and its electroencephalographic
Tina qualifies as "typical absence". This type of seizure is more common
observed in persons with a pronounced hereditary burden, it is easily provoked
flickering light and hyperventilation, has a relatively good prognosis. With the so-called

complex absences, switching off consciousness and autonomic disturbances are accompanied by various
motor phenomena such as myoclonic twitching (myoclonic abscess),
involuntary contractions of the muscles of the face, rolling the eyeballs, turning off
postural tone, as a result of which the patient falls (atonic absence). Difficult absence for
EEG is manifested by rhythmic peak-wave complexes with a frequency of 3 discharges per second (usually
this is myoclonic abscess), but more often the indicated discharges have a frequency of 1.5-2.5 per second (so
called atypical absence). This form of absence is usually observed in children 2-8 years old with pronounced organic symptoms and delayed mental and physical development and
has a poor prognosis (Lennox-Gastaut syndrome).
Often absences are called minor seizures ("minor illness", petitmal). However, these
the terms are not identical, since small seizures sometimes denote some forms
partial epileptic seizures occurring with loss of consciousness without convulsions and falling patient, for example temporal pseudoabsances.
It is assumed that the epileptic focus in generalized seizures is located in
oral parts of the brain stem. However, these seizures may be due to
pathological foci in the mediobasal parts of the cerebral hemispheres with instant generalization.

CLASSIFICATION OF EPILEPTIC SEASTS INTERNATIONAL ANTIEPILEPTIC LEAGUE (1981)

I. Partial (focal) seizures.
Partial simple
1. Propulsion with the march (Jacksonian); without march - adversive, postural, speech (vocalization or speech arrest).
2. Sensory (somatosensory, visual, auditory, olfactory, gustatory, seizures
dizziness of an epileptic nature).
3. Vegetative.

4.With psychopathological manifestations (dysphasic, dysmnestic, cognitive,
affective, illusions and hallucinations).
Partial complex
1. Beginning as simple with subsequent impairment of consciousness (with and without automatisms
automatisms).
2. Beginning with a violation of consciousness (with and without automatisms).
Partial seizures with secondary generalization
P. Generalized seizures
Absances
1. Simple
2. Complex (clonic, tonic, atonic, with vegetative manifestations, with
automatisms)
Seizures
Tonic-clonic
Tonic
Clonic
Myoclonic (localized myoclonus)
Atonic (akinetic).
Unclassified seizures.
The second group of epileptic seizures is partial (focal). With partial

seizures, the epileptic focus is always located in the cerebral cortex, only
part of the brain, while, as established, the participation of subcortical structures is mandatory, partial
seizures are divided into simple (without impaired consciousness) and complex (with impaired consciousness).
In addition, partial seizures with secondary generalization are distinguished. Among the partial simple distinguish seizures with motor, sensitive, autonomic,
psychopathological manifestations.
Among the partial movement seizures, the most conspicuous is
Jacksonian, or somatomotor seizure, arising from the location of the epileptic
focus in the projection motor cortex. It is characterized by clonic seizures with
involvement in the process of the muscles of the face, hand, foot, etc. Convulsions can be localized or
common, depending on the characteristics of the cortical somatotopic localization
motor functions (Jackson's march). Consciousness is preserved. In some cases, seizures
spread to the entire half of the body, and sometimes generalized, which is accompanied by loss consciousness.
An oculomotor epileptic seizure is manifested by tonic abduction of the ocular
apples, adversive - turning the eyes and head in the opposite direction, and a seizure
epileptic rotation - by turning the trunk to the side as well. These seizures are caused
epileptic foci in the premotor cortex.
A special type of partial movement seizure occurs in epilepsy.
Kozhevnikova - constant myoclonus in a limited muscle group, periodically turning into generalized seizures.
Partial sensory seizures include primarily sensory Jacksonian,
or somatosensory seizures. These are attacks of paresthesias of limited localization, it is possible
Jackson's march, consciousness preserved. They are found when foci appear in the projection
sensory cortex. A somatosensory seizure often turns into a somatomotor (so
called a sensorimotor seizure). Sensory seizures also include visual,
cold, olfactory, gustatory seizures, manifested in the form of irritation phenomena
the corresponding projection cortex (photopsies, false sensations of taste, smell, etc.).
With epileptic seizures, psychopathological manifestations rarely proceed without
disturbances of consciousness and therefore are more often observed in the framework of partial complex seizures.
A change in consciousness in partial complex seizures is manifested by a lack of response to external stimuli or a violation of awareness of what is happening.
In case of impaired perception caused by foci in the projection-associative cortex,
illusory or hallucinatory experiences arise - vivid visual scenes, complex

melodies. These seizures are psychosensory. The latter also includes seizures
with cognitive and dysmnestic manifestations, accompanied by impaired sensory
synthesis by the type of derealization or depersonalization, usually proceeding with impairments consciousness. These are the syndromes dejavu - "already seen", dejaentendu - "already heard", dejavecu - "already
experienced ", jamaisvu -“ never seen ”, jamaisentendu -“ never heard ”,
jamaisvecu - “never experienced”. In all these cases, the visible, audible, experienced
to the sick it seems to him already previously visible, audible, experienced. Or, conversely, situations and
the phenomena that are repeatedly encountered, experienced by the patient, seem to him completely new,
never seen before, never experienced. With these paroxysms, there is one or another

the degree of impairment of consciousness. In severe cases, there are, in Jackson's terminology, "Dreamy states" (dreamystates) experienced by the patient as if in a dream. Similar paroxysms can include pharyngo-oral symptoms (swallowing, sucking, smacking, etc.), combine with olfactory and gustatory hallucinations, violent memories.
More rare seizures, defined as attacks of impaired memory and ideation seizures, usually manifest as interruption of thoughts, often an intrusive memory something, etc. and are caused by epileptic discharges in the temporal or frontal lobe of the head brain.
Partial seizures include vegetative-visceral paroxysms. They diverse, caused by epileptic discharges in the orbital-insulotemporal area and, possibly in the rostral part of the trunk. Tachycardia is clinically observed, increased blood pressure, shortness of breath, mydriasis, sweating. The most studied digestive epileptic seizures caused by discharges in the pararinal region - pharyngo-oral, epigastric, abdominal. Pharyngo-oral epileptic seizures are manifested hypersalivation, often combined with movement of the lips, tongue, licking, swallowing, chewing, and
etc. Abdominal epileptic seizure is characterized by various sensations in epigastric region (epigastric seizure), often with rumbling in the abdomen, vomiting, etc., often accompanied by a change in consciousness.
Vegetative-visceral seizures are characterized by the same features as others epileptic seizures: short duration, stereotyped manifestations in one and the other the same patient, they are often accompanied by changes in consciousness.
The literature describes the so-called diencephalic, or hypothalamic epilepsy, characterized by vegetative crises or vegetative-visceral paroxysms, lasting from several tens of minutes to several hours or more. In this case, short duration of loss of consciousness and seizures, developing only at the height of the seizure. Seizures are clinically different from epileptic seizures. Electrographically, they do not it is possible to register the phenomena characteristic of an epileptic seizure. Hypothalamic paroxysms do not respond to antiepileptic drugs.
Therefore, in the Terminological Dictionary of Epilepsy and in the works of a number of Russian Researchers questioned the validity of diencephalic epilepsy isolation.
With epilepsy, psychomotor seizures, or, according to the Terminological Dictionary, epileptic seizures of automatism, which are characterized by paroxysmal disturbances of consciousness and motor activity in form of automatisms and, therefore, also refer to complex partial seizures.
The behavior of patients is distinguished by externally ordered actions, which, however, inadequate to this situation. Darkening is observed during psychomotor seizures consciousness followed by amnesia.
All types of psychomotor seizures are most often caused by epileptic discharges in the anterior sections of the temporal lobe. The International Classification provides for the allocation affective seizures - various paroxysmal mood disorders, usually unmotivated feelings of fear, less often - fits of laughter (hololepsy), a state of bliss and etc. Usually such paroxysms are accompanied by a change in consciousness. They are caused by discharges in the anteromedial part of the temporal lobe.
Partial (focal) seizures with both simple and complex symptoms can go into generalized seizures; in these cases, they speak of secondary generalized seizures. Secondary generalized seizures should also include generalized

seizures with a preceding aura ("symptom-signal"). Aura (Latin aura - "breath")

- a term denoting pathological sensations of a different nature that arise in patients at the onset of a seizure (in addition to secondary generalized, it can also be observed with partial complex seizures).
Usually, the same patient has an aura with a recurrence of a large seizure. stereotyped. Its nature is determined by the location of the epileptic focus. During the aura the patient may sense, for example, any smell (olfactory aura), taste (gustatory aura), see various images or whole pictures (visual aura), etc.
There are the following types of aura: sensory, sensitive, motor, speech, vegetative and mental. Visual and auditory auras are examples of sensory auras. Sensitive aura manifests itself in the form of various senestopathies (dry mouth, feeling of numbness, distorted perception of your body, etc.). Motor aura is a stereotyped movement performed by a patient before
the occurrence of seizures. Speech aura is manifested by interruption of speech (inability to speak)
or, conversely, the violent involuntary pronunciation of certain words. In addition to motorn speech aura, maybe a sensory speech aura - the patients either do not understand addressed to them, or they hear words that no one really says.
The vegetative aura is perhaps the most diverse. 9to and various unpleasant sensations in areas of the heart (cardiac aura), abdominal organs (abdominal aura), stopping sensations breathing or shortness of breath, hunger, etc.
The psychic aura is especially peculiar, during which the patient experiences an unusual emotional state, sometimes incredible bliss. An example of a similar aura can be gleaned from the excellent description of f. M. Dostoevsky has a seizure in Prince Myshkin.
The writer himself suffered from epilepsy and, apparently, experienced similar sensations.
The aura lasts for a moment and is the only, often very vivid memory, which subsequently persists in the patient about the seizure. Often the aura can be the only one clinical manifestation of an epileptic seizure. However, usually following the aura, loss of consciousness and seizures develop. The aura always indicates the focal onset of the seizure.
As already noted, epilepsy is characterized by paroxysmal manifestations and non-paroxysmal chronic changes in the patient's personality. Epilepsy is characterized by slowness and stiffness of mental processes, pathological thoroughness thinking. Patients in a conversation are verbose, but they cannot highlight the main thing, express the main point, to give a short answer, get stuck on the secondary details.
Excessive punctuality, pedantry, petty touchiness, impatience develop, foolishness, importunity and at the same time timidity, obsequiousness, flattering, shyness, sweetness, exaggerated deference, gentleness in handling, desire to please the interlocutor (the so-called defensivity). The circle of interests gradually narrows, worsens memory, egocentrism grows, features of epileptic dementia are revealed.
These changes are based on many factors: organic brain damage, impairment normal functional activity of the brain under the influence of epileptic discharges, chronic stressful conditions due to recurrent seizures and related disease, family and social difficulties, hereditary predisposition and adverse effects of long-term antiepileptic therapy.
In addition to chronic changes in the patient's personality and mental manifestations during seizure (mnestic, ideatorial, affective symptoms), with epilepsy often occur periodic mental disorders in the form of dysphoria or psychoses of varying duration.
In addition to the International Classification of Epileptic Seizures, there is International classification of epilepsy, according to which the forms of epilepsy are distinguished by two
main criteria: etiology and type of seizures. With an established etiological factor and

localization of epileptogenic lesions, epilepsy is classified as symptomatic. If etiology cannot be established, and organic brain damage is obvious, epilepsy called cryptogenic. In the absence of data for organic brain damage and unknown etiology of epilepsy is idiopathic. By the type of seizures, focal and generalized epilepsy. The forms of focal epilepsy are: benign rolandic and occipital epilepsy, reading epilepsy (idiopathic forms); Kozhevnikovskaya epilepsy and epilepsy with specific forms of provocation (symptoms

matic forms). Generalized forms include: benign neonatal convulsions, benign myoclonus-epilepsy of infancy, pycnolepsy, juvenile myoclonus-epilepsy, epilepsy with generalized tonic-clonic seizures awakening; West and Lennox-Gastaut syndromes, epilepsy with myoclonic-astatic seizures, epilepsy with myoclonic seizures (cryptogenic and symptomatic forms); early myoclonic encephalopathy and other symptomatic forms.
In addition, undifferentiated epilepsy with generalized and focal seizures (neonatal, myoclonus-epilepsy of infancy, acquired epilepsy-aphasia, etc.) and special forms, in particular, situational seizures (febrile, dismetabolic).
Diagnostics. Anamnesis must be collected both from the words of the patient and his immediate relatives. Indications of perinatal pathology, early cerebral processes give reason to suspect residual brain damage, which may result in epilepsy.
Often in the anamnesis there are indications of the presence of epileptic seizures in relatives. Particular attention should be paid to paroxysmal episodes in childhood - convulsions in the neonatal period, the so-called spasmophilia, which is often mistaken for epileptic seizures, convulsive seizures during febrile states (febrile convulsions). These should also include the so-called abdominal crises - paroxysms short-term abdominal pain, occurring regardless of food intake, accompanied by vegetative disorders (paleness, nausea, pulse changes, etc.).
The role of some paroxysmal conditions that occur during sleep has been less studied - night fears, myoclonus, tonic spasms, etc., which are often observed in children.
Epilepsy is characterized by stereotype and regularity of seizures, relative the independence of the latter from external influences, often confined to a certain time of day (night seizures - "sleep epilepsy"; morning - "epilepsy awakenings ", daytime -" wakefulness epilepsy ", etc.), the presence of appropriate changes in character and intelligence.
When formulating a diagnosis, it is necessary to indicate the form of epilepsy, the nature of the seizures, their frequency, features of their distribution in the cycle "wakefulness - sleep", the presence or absence mental changes, such as epilepsy with frequent polymorphic seizures (complex psi hosensory, secondary generalized) sleep and wakefulness, dysphoric states and pronounced personality changes.
Early manifestations of epilepsy have certain characteristics: incomplete, rudimentary, abortive, partial forms of paroxysms, high frequency of sleepwalking and dreaming, muscle twitching. At this time, seizures may still be episodic. character, are provoked by various external influences, for example, fear, which is especially often observed in children, overwork, etc. However, gradually characteristic for each patient the type and rhythm of seizures. As the disease worsens, gradually new symptoms come to light: increased frequency of seizures, often a tendency to serial manifestation or the development of status epilepticus. At this time, mental changes may increase. So

Thus, epilepsy has certain patterns of course, the study and analysis of which are important for the diagnosis and prognosis of the disease.
Epilepsy is characterized by certain changes in the EEG. Similar changes may be found in epileptic syndromes, but in these cases they are combined with EEG changes due to the main process.
The most characteristic of epilepsy are the so-called epileptic signs - peaks, acute waves and peak-wave complexes. With epilepsy, EEG also records paroxysmal rhythms - rhythmic discharges of increased voltage with a frequency of 8-12. 14-16, 20-30 per second.
However, electrographic epileptic signs are by no means always detected with ordinary recording conditions - about 1/3 of the time. Therefore, various methods of provocation are used: rhythmic light stimulation (light flashes in a rhythm of 4 to 50 per second), hyperventilation (deep breathing for 3 minutes), in some cases - introduction pharmacological agents of convulsive action (corazole, bemegrid, etc.). Moreover, the percentage
detected on the EEG of epileptic phenomena increases. The most powerful activator epileptic activity is sleep, namely the stage of the carotid spindles associated with the mechanism

thalamocortical recruitment. However, electrographic registration during natural or pharmacological sleep can only be applied in special institutions. A widely available and highly effective method of provoking epileptic activity is 24-hour sleep deprivation followed by a routine EEG recording. Repeated EEG recording at short intervals also increases the likelihood of detecting pathological changes in epilepsy. The nature of the electrographic phenomena detected with epilepsy, largely depends on the form of epilepsy, type of seizure, localization epileptic focus. The most characteristic changes occur with generalized forms of epilepsy, primary generalized seizures.
Partial seizures, which are usually of cortical origin characterized by the appearance on the EEG of multiple peaks, sharp waves. However, others partial seizures - olfactory, auditory, visual, attacks of dizziness, psychomotor and psychosensory - may have a different electrographic characteristic, not, however, clearly defined.
So, bilateral, often asymmetric, more or less pronounced acute and slow waves. Paroxysmal rhythms are very often observed with a discharge frequency of 4-7 V second or with a different frequency, recorded in the temporal or frontal leads, or generalized.
With secondary generalized seizures, the described EEG changes usually turn into electrographic picture of a large seizure. Apart from the above changes, characteristic of epilepsy, other EEG changes are usually found, with focal, unilateral or generalized character (hypersynchronous α-rhythm, deformity of normal rhythms, the appearance of slow oscillations - δ- and ε-waves), etc.
With unfavorably current epilepsy, so-called hypsarrhythmia is often observed. (from the Greek. hypsos - "high"), the presence of slow waves and rhythmic discharges in the l- and e-range
waves of increased amplitude in combination with epileptic phenomena in the absence of normal rhythms. Hypsarrhythmia is characteristic of infantile spasm.
Of great importance for the diagnosis of epilepsy are such research methods as CT and MRI, which allow visualization of brain structures at various levels and identify even minor areas of brain tissue atrophy and other changes.
In recent years, video and tele-electroencephalographic monitoring has been used.

patients, which can significantly improve the accuracy of diagnosis.
Changes in the body during ontogenesis leave an imprint not
only on the features of its functioning at different age periods, but also on
clinical manifestations of various pathological conditions.
The paroxysmal readiness of the child's brain is much higher than that of adults. Causes
of this are varied: high hydrophilicity of the brain tissue, other cortical-subcortical
relationships, immaturity of brake systems, lability of homeostasis, etc.
Late epilepsy (late-onset epilepsy) is characterized by a predominance of large
convulsive seizures and psychomotor paroxysms. Convulsive seizures proceed as
as a rule, with pronounced vegetative manifestations (fluctuations in blood pressure, breathing disorders).
Post-attack coma is more prolonged. The aura is more often speech, affective, migraine-like.
The seizures, as a rule, are monomorphic, there is a tendency to a serial course.
Epileptic activity on the EEG is detected less often and is more localized. Flow
disease (in idiopathic epilepsy) usually benign, epileptic dementia
rarely develops. It should be emphasized, however, that idiopathic epilepsy rarely manifests itself.
stays in adulthood and old age, therefore, a particularly careful examination of such
patients to exclude focal pathology (primarily a brain tumor or other volumetric
process).
Differential diagnosis of epilepsy. If the patient has epileptic
seizures, it is necessary first of all to exclude the current cerebral process. Moreover, in addition to
symptoms characteristic of certain brain diseases (tumors, encephalitis, etc.) should be
take into account the peculiarities of the manifestation and course of the epileptic seizures themselves.
So, Jacksonian seizures in epilepsy are rare and, conversely, they are often
are the first manifestation of tumors of the Roland region of the cerebral hemispheres.

The dynamics of seizures in tumors is characterized by a number of features. More often the first seizures
are generalized, and the subsequent ones are of a partial nature, often begin with a series
seizures. It is characteristic that as the hypertensive syndrome appears and increases, seizures
become more rare and limited, and then completely disappear.
Epileptic seizures in acute disorders of cerebral circulation have, as
as a rule, convulsive in nature, the features of their manifestations correspond to the zone of violation
cerebral circulation. So, with discirculation in the vertebrobasilar basin,
generalized, and in the carotid - Jacksonian and secondary generalized seizures. V
subsequent seizures are repeated due to repeated acute cerebral disturbances
circulation, as well as decompensation of blood circulation in the area of old foci.
In some patients, seizures can occur under the influence of certain external
stimuli (the so-called reflex epilepsy): most often visual (flashes of light,
some images, different color sensations, especially red), less often auditory
(unexpected noise or certain sounds, voices, music), somatosensory (unexpected
touching or prolonged stimulation of a specific part of the body); besides, they sometimes
occur when reading and eating. Seizures can be partial (occurring in the
the area of the brain awakened by the stimulus), however, they are more often primary
generalized. They usually begin in childhood and adolescence and with
usually become less pronounced with age. More often seizures occur while watching
television programs, in a disco with flickering light, as well as when moving on

escalator. Preventing light-stimulated seizures sometimes
you can cover one eye. It is necessary to use anticonvulsants only when it is not possible
avoid the action of provoking factors, but these funds often turn out to be
ineffective. In rare cases, with reflex epilepsy, any
focal brain damage, however, in most patients, the reasons for such a selective increase
convulsive readiness remain unknown.
Sometimes in women, the frequency of seizures increases just before menstruation and
during them (the so-called menstrual epilepsy), which may be associated with a delay in
the body of water and / or with cyclical changes in hormonal levels. Treatment in this case
is carried out only on the eve of and during the "dangerous" period and includes the use of
diacarb,
oral contraceptives (consultation with a gynecologist is required), as well as anticonvulsants
(it is obviously preferable to use benzodiazepines - clonazepam or nitrazepam, however
other drugs can be used).
Often, seizures occur in patients with alcoholism when they suddenly stop taking
alcohol; 90% of these seizures occur in the first 7-48 hours of withdrawal. More often there is a
series of
2-6 seizures (over 4-12 hours), after which the seizures usually do not recur. Usually,
generalized tonic-clonic seizures are observed.
Focal seizures are more likely to indicate concomitant focal brain damage.
(for example, head injury). Alcohol can also trigger seizures when
existing epilepsy.
Seizures can occur with sudden withdrawal of phenobarbital, as well as a number of hypnotics
drugs, as a rule, against the background of other manifestations of withdrawal symptoms.
It should be remembered that seizures occurring in the 1st year of life are more often due to
congenital malformations, birth trauma, infections, anoxia, metabolic
disorders (hypocalcemia, hypoglycemia, vitamin B6 deficiency, phenylketonuria) or
represent an infantile spasm. Seizures that occur during childhood are usually
caused by perinatal anoxia or birth trauma, infections, cerebral thrombosis
arteries and veins are either manifestations of idiopathic epilepsy. Seizures starting
in adolescence are usually associated with idiopathic epilepsy (especially with its
genetically determined varieties) or with trauma. Starting at a young
age (18-25 years), they can also be caused by idiopathic epilepsy, trauma,
tumors, alcohol withdrawal syndrome. Seizures occurring in middle age (35-60 years)
are more often caused by injuries, tumors, vascular diseases, chronic al-
coholism. In old age, the most common causes of seizures are vascular
diseases and tumors, less often degenerative diseases and injuries.

Treatment. During a generalized tonic-clonic seizure, help is reduced to
to protect the patient from possible damage (injury) as much as possible. His
laid on a bed or on the floor, if possible on its side, remove all surrounding objects,
capable of causing damage. Do not try to prevent tongue bite by placing
any object between the patient's jaws, since broken teeth are undoubtedly
a more unpleasant consequence of a seizure than a bitten tongue.
In the case of symptomatic epilepsy, it is necessary first of all to treat the main
disease. However, sometimes even after successful treatment (for example, after removal of the
tumor
or abscess) the need for anticonvulsant therapy remains.
The organization of a rational regime of work and rest is of great importance, as well as
psychotherapy. The patient should, if possible, lead an active lifestyle. Moderate
physical exercise. Activity is a seizure antagonist. At the same time, it follows

avoid excessive emotional and physical stress, prolonged stay at
sun, alcohol intake. Experience, however, shows that in patients with a favorable course
illness, occasional intake of small doses of alcohol, as a rule, does not provoke
the occurrence of seizures. It is necessary to establish regular good sleep. Should
overcome the feeling of inferiority, inferiority that often arises in patients, which often,
especially in children, it is associated with overprotection in the family. On the other hand, it
should be borne in mind that
sufferers often suffer from social isolation. Since most people with epilepsy
either mentally sound, or have only minor characterological features,
children can and should attend school, adults can continue their professional
activity, if it is not related to driving a car, working with moving mechanisms,
at a height, with fire, etc. It is necessary to identify provoking factors in each patient in order to
in order to exclude their effect as much as possible (some patients are unusually sensitive to
stress, hyperventilation, sleep disturbances, alcohol or medication).
The mainstay of treatment is the use of anticonvulsants. Successful therapy
contribute to the correct choice of the drug and the selection of its dose, continuity and duration
treatment, good contact between the patient and the doctor.
The choice of drug is based on the type of seizure. In the case of generalized and
simple partial seizures, 4 main drugs are used: phenobarbital, diphenin,
carbamazepine, sodium valproate (Table 7), while their effectiveness is theoretically
approximately
is equal, and the choice of a specific drug is carried out taking into account individual sensitivity
the patient (by trial and error), side effects, as well as the availability of the drug.
In practice, phenobarbital is used more often in adults at a dose of 2-4 mg / (kg * day), in
children 3-8
mg / (kg * day), however, in case of sleep epilepsy, it is advisable to avoid it, since it suppresses
the phase
REM sleep, which has a depressing effect on epileptic activity, shortened in
patients with sleep epilepsy. In children, the drug can cause paradoxical hyperactivity,
irritability and interfere with the learning process. Drowsiness (main side effect
drug), as a rule, decreases after a few days or weeks from the start of administration
drug; if this does not happen, it sometimes has to be canceled. However, usually
phenobarbital is well tolerated and can be effective in all types of epilepsy.
Diphenin with prolonged use can cause hypertrichosis, coarseness of facial features,
gingival hyperplasia, impaired concentration. These side effects limit it
use in children and young women. Chronic diphenin intoxication sometimes
accompanied by the appearance of neuropathy and cerebellar degeneration. The drug has
adverse effects on folate and vitamin K metabolism.
When using carbamazepine (finlepsin), emotional lability may occur,
drowsiness and difficulty falling asleep, impaired concentration, loss of appetite, vomiting,
headache, allergic reactions, leukopenia, thrombocytopenia. However, as it develops
effect of autoinduction, these phenomena, as a rule, pass. The most favorable side
the effects of the drug are associated with the suppression of bone marrow function and the
damaging effect on
liver. Therefore, patients regularly (at the beginning of treatment - once a month) need to carry
out
clinical blood test and liver function tests. With signs of bone marrow suppression
or liver dysfunctions, the drug must be canceled. In most cases, these
the phenomena are reversible, although occasionally irreversible aplastic anemia with fatal

outcome. However, it should be emphasized that these complications are rare. At that

the same time due to the fact that carbamazepia to a lesser extent than other anticonvulsants, inhibits cognitive functions, according to some epileptologists, it is a drug for boron for generalized convulsive seizures (especially with sleep epilepsy); with him lack of effectiveness should be simultaneously prescribed phenobarbital or hexamidine.
For the treatment of partial movement seizures other than those listed above drugs, you can prescribe benzonal (benzobarbital) at a dose of 5-10 mg / kg. For the treatment of partial complex seizures, the following drugs are used in order of importance: carbamazepine, 15 20 mg / (kg * day), diphenin 5-10 mg / (kg * day). hexamidine at 10-15 mg / (kg * day). It should be noted that with this type of seizure, anticonvulsants are less effective than with generalized tonic-clonic seizures.
For the treatment of absences, ethosuximide is primarily used at a dose of 10-25 mg / (kg day). The drug can provoke the occurrence of large seizures requiring the use of phenobarbital. If it is ineffective, sodium valproate, diacarb are used. At in atypical absences, sodium valproate is the drug of choice, if there is no effect sodium valproate should be used with ethosuximide or lamictal.
For the treatment of myoclonic seizures, sodium valproate is used at 10-50 mg / (kg day), benzodiazepine derivatives - clonazepam at 0.05-0.1 mg / (kg day) and sibazone (2-4 mg / day in children, 5-10 mg / day in adults, followed by a slow increase in dose). If they are ineffective, you can prescribe diphenin and phenobarbital. The use of valproic acid preparations that increase the content of GABA in the brain, as well as lamiktal, significantly improved the prospects treating a number of severe forms of epilepsy, including infantile spasm. For the treatment of the latter use GABA drugs, for example, synacthen, which is administered intramuscularly at a dose of 1 mg per a year of life (usually 12-15 injections per course); in case of recurrence of the disease, a second course is carried out treatment. Use corticosteroids, lamictal, nitrazepam (2-3 mg 1-3 times a day), ketogenic diet or diacarb (fonurite).
It should be remembered that with prolonged use of clonazepam in patients with different the period of treatment develops a tolerance to the drug, which requires a "medicinal vacation".
Diacarb (fonurite), which is a carbonic anhydrase inhibitor, is prescribed to enhance anticonvulsant therapy also for epileptic seizures occurring against the background hydrocephalus, at menstrual epilepsy, at seizures provoked hyperventilation. It has potassium-depleting properties, which requires appropriate correction; when using it, paresthesia, anorexia, drowsiness may occur, rarely - agranulocytosis. Doses of the drug - from 250 mg 2 times a day to 0.5 g 3 times a day. Since to Diacarbu quickly develops tolerance, intermittent treatment is optimal.
Treatment should always start with one drug, gradually increasing its dose from initial to moderate, then if seizures continue and toxic effects do not occur - up to the maximum. If the frequency of seizures has not changed, then the drug should be canceled, at the same time prescribing another drug. In cases where only partial therapeutic effect, especially if the seizures are of a mixed nature, and

if none of the anticonvulsants in isolation has had the proper effect, to
the second drug is added to the first drug, or a combination of drugs is prescribed accordingly.
It should be remembered that with a combination of drugs, the likelihood of developing side effects increases.
reactions as a result of their interaction. However, a combination of drugs sometimes allows, by reducing
the dose of one of them, to reduce the phenomena of intoxication, while maintaining the therapeutic effect.
Treatment for epilepsy should be continuous and long-term. Cancellation question
antiepileptic drugs can be supplied in those patients in whom drug
remission lasts at least 3 years. Cancellation of drugs is made gradually within 1-2
years by reducing doses with careful clinical and electroencephalographic control.
Discontinuation of treatment should not be carried out during puberty. Insofar as
antiepileptic treatment is carried out for a long time, you should pay special attention to
possible side effects of drugs. Signs of their intolerance appear in the form
drowsiness, weakness, nausea, ataxia, changes in blood counts and other symptoms,
therefore, first of all, it is necessary to regularly examine the blood. In addition, one should consider
features of the side effects of individual drugs.

Low efficacy of anticonvulsant therapy can be expected in the presence of
pronounced mental changes in adult patients, in children - with mental retardation
development, the presence of malformations, pronounced focal neurological symptoms, with
prolonged existence of seizures, the presence of partial seizures (especially partial
complex), with significant changes in the EEG. At the same time, factors such as the number of seizures
prior to treatment, family history does not affect treatment success.
It should be remembered that in some cases, mental changes in patients (including
apparent dementia) may be associated with inadequate treatment: chronic intoxication
drugs (including those with a deficiency that often occurs with their prolonged use
folic acid), with insufficient control of seizures (especially partial complex
seizures); a combination of both reasons is often observed. Correction of therapy in these cases
can cause significant improvement in the patient's condition.
In the absence of improvement from conservative treatment, it should be timely, that is, before
the occurrence of severe mental changes and secondary epileptic foci, put
the question of surgical treatment. Currently, many methods are used with success.
surgical treatment of epilepsy aimed at removing epileptic foci
(hypokampectomy, anterior temporal lobectomy, etc.), interruption of the pathways
epileptic discharge (fornicotomy, commissurotomy, etc.) and activation of inhibitory structures
(stimulation of the dentate nucleus of the cerebellum, etc.). Methods for bioelectric
management of epileptic seizures.

EPILEPTIC STATUS

According to the definition of WHO experts, status epilepticus is a fixed
epileptic condition resulting from a prolonged epileptic seizure
or seizures that recur at short intervals. The second is usually found
a type of status epilepticus due to recurrent seizures, and
extremely rarely, status epilepticus manifests itself as one continuous
seizure. There are as many options for status epilepticus as there are types
epileptic seizures. However, epileptic
the status of seizures, as it is very difficult and is an immediate
a threat to the patient's life. 15 years ago, mortality from status epilepticus,

developed as a result of convulsive seizures, was 16-33%; she is not currently
exceeds 5%.
The most common cause of status epilepticus in epilepsy is
feverish conditions, somatic diseases, a sudden interruption in the treatment of epilepsy,
alcoholism, sleep disturbance, etc. At the same time, there is a status-like form of epilepsy, with
which epileptic seizures occur, as a rule, in the form of status epilepticus.
Often, status epilepticus develops in other diseases - brain tumors,
cysticercosis, neurotoxicosis in children, traumatic brain injury, etc.
varying frequency, however, it is characteristic that each subsequent attack occurs before
clouding of consciousness, breathing disorders and cardiovascular activity disappear,
called a previous seizure.
Disturbances of consciousness in the pauses between seizures - stupor, and then coma
accompany
seizure status epilepticus. For status epilepticus Jacksonian seizures
consciousness can remain intact for a long time.
As a rule, status epilepticus is accompanied by severe disorders
breathing: apnea during the tonic phase of an attack with the development of intense cyanosis,
dyspnea in
the clonic phase of the seizure, and after the end of the seizure - compensatory
hyperpnea. Alternation
asphyxiation and hyperventilation due to recurrent seizures is an important factor
self-maintenance of status epilepticus. Breathing disorders are common
pharyngeal type - stenosis and occlusion of the upper respiratory tract by secretion products and
aspiration. In severe cases, breathing disorders of the periodic type are observed -
wavelike shortness of breath, Cheyne-Stokes breathing.
With status epilepticus, patients usually have increased blood pressure and

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tachycardia develops, often reaching extreme degrees. Considering the heavy load on
cardiovascular system due to repetitive seizures, accompanied by a violation
breathing, then frequent deaths in status epilepticus become clear. This
also contribute to significant shifts in the internal environment of the body -
metabolic acidosis, arterial hypoxemia and venous hyperoxia, increased and then
a decrease in the content of 17-hydroxycorticosteroids in the blood, almost complete
disappearance in the blood
free adrenaline, myoglobinuria, etc.
Treatment. In status epilepticus, treatment is urgent, staged and complex.
character, that is, it starts on the spot, continues in the ambulance and in the hospital and
is aimed at eliminating seizures, eliminating respiratory and cardiovascular disorders,
normalization of homeostasis.
On the spot (on the street, at home, etc.), it is necessary to free the oral cavity from foreign
items, enter the duct. Sibazone 20 mg diluted in 20 ml is slowly injected intravenously
40% glucose solution. Intramuscular injection of the drug is ineffective. In the ambulance
help, if necessary, drain the upper respiratory tract, if repeated
seizures are re-injected intravenously sibazone or intramuscularly thiopental sodium or
hexenal at the rate of 1 ml of a 10% solution per 10 mg of the patient's body
weight. Simultaneously with
symptomatic status epilepticus begins treatment of the underlying disease - stroke,
meningitis, traumatic brain injury, etc. Patients are delivered to the intensive care unit.
Here, the final diagnosis of the disease is carried out (it should be remembered that
status epilepticus can occur in case of violations of water-mineral metabolism -
hyponatremia, hypocalcemia, which requires appropriate research). Held

lumbar puncture.
If seizures continue, an intravenous drip of Sibazone is attempted.
at the rate of 100 mg of the drug per 500 ml of 5% glucose solution at a rate of 40 ml / h, that. guarantees
maintaining the concentration of the drug in the blood at a therapeutic level. For adults sick together with glucose, 2 ml of thiamine should be introduced to prevent the possibility of development
encephalopathy Wernicke-Korsakov. When hyponatremia is detected, intravenously slowly inject
hypertonic (3%) sodium chloride solution; overly rapid correction of this disorder
can lead to the development of central pontine myelinolysis. If the seizures are not completely are eliminated, prolonged dosed anesthesia is performed. The seizures stop completely
upon reaching the I or II stage of the surgical stage of anesthesia, the duration of which should be 1.5-2 hours; then the anesthesia continues in a more superficial stage for several more hours.
In case of intractable status epilepticus, it is necessary to apply for several days
oxygen-nitrous-nitric anesthesia on muscle relaxants and controlled breathing with
deepening of anesthesia with other anesthetics (fluorothane, barbiturates, viadril, etc.). Held drainage of the upper respiratory tract, cardiac remedies are used to eliminate
metabolic acidosis - intravenous drip infusion of 8.4% sodium bicarbonate solution
under the control of the acid-base balance of the blood.
To improve metabolic processes in tissues, the use of inhibitors is shown
proteolytic enzymes, for example 25000-50000 IU of trasilol (counterkal, tzalol) in 300-500 ml of isotonic sodium chloride solution intravenously (antikinin action). At
hyperthermia of the patient is covered with ice bubbles, lytic cocktails are injected intravenously.
Since with status epilepticus in all cases, disseminated syndrome develops
intravascular coagulation (DIC), it is necessary to inject heparin under the skin of the area abdomen 5000 IU several times a day (under the control of blood clotting) (chime, persantine, anginal).
With symptomatic status epilepticus, treatment is fully carried out
the underlying disease. In some cases, you have to resort to surgical
intervention with the removal of the epileptogenic focus or its local cooling, which accompanied by the restoration of the effect of anticonvulsants, which, when
status epilepticus often lose their effect.
After the patient's exit from status epilepticus, treatment should be directed to
prevention of recurrence of the disease, elimination of asthenic syndrome and elimination respiratory complications - tracheobronchitis and pneumonia.

PROGRESSIVE DISEASES OF THE NERVOUS SYSTEM
SYRINGOMIELIA
Syringomyelia is a chronic disease characterized by the presence of longitudinal
cavities that are localized in the central part of the spinal cord and often in the oblong brain (syringobulbia). For the first time the term "syringomyelia" (from the Greek syrinx - a tube) suggested
Ollivier in 1824
Etiology and pathogenesis. More often, typical changes are found in the lower cervical and upper thoracic spinal cord. The spread of pathological
process in the medulla oblongata; higher localization is also possible - bridge, inner capsule (syringoencephaly). Cavities in the thoracic region are regularly found. Against,

lumbosacral syringomyelia - casuistry. The affected spinal cord is enlarged. V
in some cases, an increase in transverse dimensions leads to erosion of bones that form
spinal canal. On a transverse section of the spinal cord, a cavity is visible, surrounded by
translucent gelatinous tissue in which glial cells are histologically determined.
There are two types of syringomyelic cavities - "communicating" and
"Non-communicating". The term "communicating" syringomyelia (hydromyelia) suggests the
presence of
direct communication of the IV ventricle with an abnormally dilated central canal
spinal cord. The contents of the cavity are identical to the cerebrospinal fluid.
Both types of syringomyelia are similar in pathomorphological features, and
the differences lie in the nature of the pathological processes leading to education. If
“Communicating” syringomyelia is associated with a defect in the anlage of the primary cerebral
tube or suture,
then "noncommunicating" is usually symptomatic and associated with a spinal injury
brain, arachnoiditis (as a consequence of purulent or tuberculous meningitis, complications
spinal
anesthesia,
carried over
subarachnoid
hemorrhage
or
neurosurgical intervention) or, finally, the cavity develops into. intramedullary
glioma or ependymoma. In general, both "communicating" and "non-communicating" cavities
syringomyelias are usually located in the cervicothoracic region. Only intramedullary cysts
tumors can be localized in any part of the spinal cord.
With traumatic paraplegia (tetraplegia) or arachnoiditis, the cavity is usually
extending upward from the site of damage. In the formation of a cavity, in addition to
hydrodynamic
chemical factors, venous obstruction, protein exudation, ischemia and edema play a role.
"Communicating" syringomyelia is much more common. T. Gardner first pointed out
on the connection of cavities of this type with congenital anomalies in the region of the greater
occipital
holes. This includes Chiari I anomaly (congenital prolapse of the cerebellar tonsils below
edges' foramenmagnum), anomalies in the development of the craniovertebral joint,
hydrocephalus,
basal arachnoiditis (15% of cases), Dandy-Walker syndrome. The indicated anomalies occur
in more than 50% of patients. Syringomyelia is especially often combined with Chiari I anomaly.
Gardner believes that syringomyelia occurs due to the occurrence of an obstacle to
pathways for the outflow of cerebrospinal fluid from the IV ventricle into the subarachnoid space
spinal cord, whereby under the pressure of a pulsating downward wave
Cerebrospinal fluid dilates the central canal of the spinal cord (hydromyelia).
The ruptures of the walls of the central channel lead to the formation of cavities located in the
parallel to the central channel. Gardner's concept is the basis of modern ideas about
“Communicating” syringomyelia, although there are different opinions about the role of
hydrodynamic
mechanisms. It is assumed that perinatal trauma can cause displacement of the tonsils
cerebellum or contribute to the development of syringomyelia in the presence of a congenital
anomaly.
However, it is also known that primary ectopia of the cerebellar tonsils may not lead to the
development of
syringomyelia, but is manifested by other progressive neurological symptoms
(occlusive hydrocephalus, cerebellar syndrome, paresis).

The prevalence of syringomyelia is 8-9 per 100,000 population.
Clinic. The disease is sometimes familial. In addition, family members may
other malformations are observed. The disease affects more often men, it can occur in any
ages from 10 to 60 years, mainly in the period of 25-40 years.
The onset of the disease is usually gradual. Sometimes the manifestation of the first symptoms
provoke coughing, sneezing, physical activity. The earliest changes include
weight loss, weakness of small muscles of the hand and loss of sensitivity in it. Less commonly
the first symptom

are pain or trophic disorders.
Sensitivity disorders. At the earliest stage in the central gray matter
the spinal cord has a longitudinal cavity, the length of which corresponds to several
segments of the lower cervical and upper thoracic regions. First, the cavity is located
predominantly
on the one hand, destroying the posterior horns and interrupting the fibers of the spinothalamic
pathway,
starting in the corresponding segments. There is a loss of pain and temperature
sensitivity, while other modalities remain intact. Such segmental
dissociated sensory impairment was first described by Charcot. If the cavity
located centrally or during the course of the disease spreads to the other half,
the dissociated loss of sensitivity turns out to be bilateral, and the "half jacket"
transforms into a "jacket".
Frequent atypical variants of loss of sensitivity in the form of stripes, "spots",
Collar. Involvement of the spinal nucleus of the trigeminal nerve in the process leads to prolapse
pain and temperature sensitivity in the outer segments of the face, the area of the tip of the nose
and
the upper lip is affected by the latter. If the cavity is primarily located in the medulla oblongata,
then the first violations of sensitivity are found on the face. Further expansion
cavity causes compression of the lateral spinothalamic tracts on one or both sides,
which is manifested by a violation of pain and temperature sensitivity in the lower parts of the
body.
Sometimes between the zones of anesthesia in the upper half of the body and the lower
extremities is determined
area (abdomen) with normal sensitivity. If the spinothalamic tract
is compressed at the level of the medulla oblongata, then pain and temperature sensitivity
disturbed or lost throughout the contralateral half of the body. The back pillars are amazed
usually the last, and in the later stages of the disease, violations of the deep,
vibration and tactile sensitivity. Thus, dissociation is lost
(splitting) sensory disturbances.
Analgesia is responsible for the frequency of injury, especially burns to the fingers, which at first
ignored. A very common symptom is spontaneous pain, which can be burning,
sharp or shooting. Unilateral pain in the face or arm may be the first
manifestation of the disease. Along with pain, paresthesias and hyperpathies are observed.
Movement disorders. The earliest movement disorders include weakness.
and muscle atrophy due to compression or destruction of anterior horn cells. Insofar as
the formation of cavities begins in the cervicothoracic spinal cord, the first amyotrophies
found in the small muscles of the hand; in this case, the process can be two-way from the very
start or develop sequentially in each upper limb. Further it is noted
weight loss of the muscles of the forearm, shoulder, shoulder girdle, upper intercostal spaces.
Atrophies usually do not reach the severity that is characteristic of motor disease
neuron. Fasciculations are rare. The proliferation of cavities in the dorsolateral parts of the

the long brain leads to damage to the item ambiguus with the development of paresis of the soft palate, pharynx,
vocal cords. Paralysis of the larynx is occasionally complicated by stridor, which may require tracheostomy.
Much less often there are motor disturbances from other cranial
nerves. Paralysis of mimic and masticatory muscles, external rectus muscle of the eye, typically asymmetric tongue involvement. Very often nystagmus is observed, as horizontal, and vertical. The destruction of the sympathetic centers in the spinal cord is accompanied by the appearance of Horner's syndrome on one or both sides. Constricted pupil response to light saved. In the case of compression of the pyramidal pathways, lower spastic paraparesis occurs. Tendon reflexes in the lower extremities increase, in the upper ones, they are decreased or absent. However, very rarely and on the hands, an increase in reflexes can be observed. Exactly at
This situation is mistakenly assumed to be amyotrophic lateral sclerosis. Pelvic functions organs are rarely violated. The ascending nature of paresis, both central and peripheral, like the ascending type of sensory impairment, with a high probability indicates non-communicating cavity growing upward. This very important clinical symptom does not apply to
lesions of the upper cervical localization.
Trophic disorders. True hypertrophy of all tissues can be observed on one
limbs (for example, cheiromegaly), or half of the body, or even in the tongue. Anhidrosis occurs usually in the face or upper extremities. An increase in sweating is also possible, which occurs spontaneously or reflexively when eating hot or spicy food. IN 20%

cases of neuroosteoarthropathy (Charcot's joints) are noted. The most commonly affected shoulder
and elbow joints, less often - the joints of the hand, temporomandibular, sternoclavicular and clavicular-acromial. X-ray shows atrophy and decalcification
bones that form the joint, erosion of the articular surfaces and the subsequent destruction of bone fabrics. Typical painlessness with gross osteoarticular changes. Stricken
the joint is often enlarged, movements in it are accompanied by loud crepitus.
Fragility of long tubular bones is noted. Trophic skin changes include cyanosis, hyperkeratosis, thickening of the subcutaneous tissue, especially on the hands; swollen fingers accept
a kind of "banana bunch". As mentioned, loss of pain sensitivity results in
exceptional susceptibility to repeated injury; healing is slow.
Purulent inflammation of the soft tissue of the distal phalanges, bone necrosis are often observed. Sequestration of the affected phalanges, when accompanied by the discharge of bone fragments (philistine - “attacks”). This picture resembles the symptom complex described in 1883. Morvan. However, the detailed picture of Morvan's syndrome, including mutation of the terminal
phalanges and other manifestations of acroosteolysis in the hands and feet, typical of leprosy and inherited
natural sensory neuropathy. Scars are usually found on the palmar surface of the fingers from previous burns. Frequent development of panaritiums, usually painless,
- the reason for the assumption of syringomyelia. Severe deep burns occur in
proximal extremities and on the trunk. Typical everyday situations - burns during sleep from steam heating batteries or heating pads.
Siringobulbia. The medulla oblongata can be involved in the pathological process when it spreads upward from the spinal cord or may serve as a site of primary localization defeat; in the latter case, the onset of the disease can be sudden or gradual.

Clinic. The clinical manifestations are trigeminal pain, dizziness,
atrophy of the tongue, paralysis of the soft palate, pharynx, larynx, nystagmus.
Syringomyelia patients have a variety of abnormalities, including
most important are kyphoscoliosis, sometimes with a rib hump, disproportionately long
in relation to the trunk of the hand, curvature of the fingers, ear anomalies, cervical rib,
spinabifida,
basilar impression, fusion (concrescence) of the cervical vertebrae (short neck syndrome) and
other craniovertebral anomalies, hydrocephalus, hollow foot. Specified congenital
deviations constitute the dysraphic status, studied in detail by Bremer. It is impossible not to
notice
that kyphoscoliosis may not be congenital, but is formed throughout life
due to asymmetric denervation of the paraspinal muscles.
When examining the cerebrospinal fluid, abnormalities, as a rule, are not detected;
blockage of the outflow by an expanding cavity is accompanied by an increase in protein
content.
Local EMG with relative constancy reveals damage to the cells of the anterior horns
in the cervical spinal cord at normal speed of conduction along the nerves, including
sensitive in areas of analgesia.
X-rays of the cervical spine can reveal congenital bone
abnormalities (eg, cervical concrescence, occipital atlas, coarctation
upper cervical segments of the spinal canal, high position of the axis tooth, cervical
ribs) or an increase in the anteroposterior size of the spinal canal.
If Gardner's concept, without solving all the problems of the formation of syringomyelia,
was the first pathogenetic construct of this disease to stand the test of time,
then the introduction of MRI was revolutionary in the diagnosis of syringomyelia. This method
first gave
the ability to analyze in detail the substrate of the disease - the cavity. First of all, it was revealed
unreliability of pathomorphological findings, the spectrum of which turned out to be
incomparably poorer than
intravital imaging of the spinal cord. MRI detects one-, two- and multi-site
Lost variants of syringomyelia. The cavities can be uniform along the entire length, widened
at the top, in the form of "beads". The width of the cavity varies, averaging 4-10 mm. By
data of N.N. Yakhno et al., in 46 examined patients with communicating syringomyelia
MRI cavities were localized in the cervical, thoracic, cervicothoracic, or bulbar regions. By
the diameter of the spinal cord, cavities could occupy the central (59% of cases), central
front and center-back position. Cervicothoracic localization was found in 81% of cases.
cavities, single cavities were present in 80% of patients. The volume of the cavities was naturally
larger
with a long (13-31 years) course of the disease. Among 12 patients with bulbar disorders
cavities in the medulla oblongata were found in 4 people. No strong match was found between

the length of sensitive drops and the length of the cavity.
Glial (without cavities) forms of syringomyelia found on MRI in some cases
do not allow differentiation with glial tumors without dynamic observation.
Diagnostics. In the advanced stages of the disease, the diagnosis is not difficult, since
a typical combination of segmental-dissociated disorders soon appears
sensitivity, muscle loss and trophic disorders of the hand, lower spastic
paraparesis, as well as dysraphic stigma, especially kyphoscoliosis.
The reason for the differential diagnosis of syringomyelia is most often
carpal and cubital canal syndromes, motor neuron disease and other diseases,
manifested by amyotrophy of the hands, in particular, upper chest aperture syndrome,

Pancost's syndrome (a tumor of the apex of the lung). A special place is occupied by hereditary sensory
and autonomic neuropathies: it was these diseases that for many years gave rise to erroneous diagnostics of the lumbosacral form of syringomyelia. Meanwhile, awareness of this relatively recently isolated nosological form, often proceeding with the roughest trophic disorders (perforating foot ulcer, acroosteolysis), allows even with first examination to distinguish hereditary neuropathy from syringomyelia, lumbosacral a variant of which, as indicated above, is extremely rare. Investigation of somatosensory evoked potentials confirms the spinal genesis of sensitivity disorders. In all cases when the lumbar cavity occurs in the absence of spinabifida (sometimes combined with hydromyelia) and in
absence of long-standing traumatic paraplegia, intraspinal tumor should be assumed.
Hematomyelia is characterized by acute development of symptoms. You just have to keep in mind
the possibility (very rare!) of hemorrhage in the syringomyelic cavity. Trophic disorders in Raynaud's disease may resemble those in syringomyelia, but in the first case there is no dissociated loss of sensitivity, and blanching of the fingers, typical for Raynaud's disease, with syringomyelia is not observed. Diagnose syringobulbia in typical cases (with a clinical picture of syringomyelia with lesions cervical spine) is not difficult. With primary lesion of the medulla oblongata, syringobulbia it is necessary to differentiate with other pathological processes of this localization. For tumors of the medulla oblongata are characterized by a more rapid development of symptoms, frequent
spread into the area of the bridge.
The course of syringomyelia is chronic, slowly progressive. Sudden increase symptoms are the result of coughing, physical exertion, trauma, or occurs when hemorrhage in the syringomyelic cavity; in exceptional cases, spinal cord sprain the brain is so pronounced that its complete transverse compression occurs and the lower paraplegia. Patients, as a rule, retain their ability to work for a long time. Death is coming from the consequences of bulbar paralysis (bronchopneumonia) or intercurrent infections.
Treatment consists of protecting non-sensitive skin areas and treating early commonplace injuries to speed up the healing process. With prolonged and difficult to stop pain requires the use of analgesics in combination with antidepressants and antipsychotics. Sometimes they resort to medullary tractotomy, stereotaxic thalamotomy.
In the presence of non-communicating cavities that have arisen as a result of a spinal tumor or arachnoiditis, in some cases, laminectomy with complete or partial removal of the tumor, decompression, drainage of the arachnoid cysts itself syringomyelic cavities, dissection of fibrous cords, compressing the substance of the dorsal brain. Decompression is indicated for hydromyelia and developmental abnormalities (Chiari malformation)
upper cervical spinal cord and lower medulla oblongata.
Syringoperitoneal and other forms of cavity shunting are currently the most commonly used surgical treatment options for hydromyelia. Indications for operations are determined by the severity and dynamics of the clinical picture and MRI data. Surgical intervention can help relieve pain, gradually
restoration of lost sensitivity and normalization of reflexes; full recovery rarely observed.

LATERAL AMYOTROPHIC SCLEROSIS

Amyotrophic lateral sclerosis (ALS) - a chronic progressive disease
nervous system due to selective damage to the spinal cord motor neurons and

the brain stem ("lower", or peripheral, motor neurons) and cortical ("upper", or central) motor neurons, clinically manifested by paresis, atrophy and pyramidal syndrome in various combinations.

ALS is the most common form of so-called motor neuron disease. V depending on the localization of the lesion, there are 4 main forms of this disease. At an isolated lesion of the brainstem develops progressive bulbar palsy (PBP), constituting about 10% of all cases of motor neuron disease and is not prognostically different from ALS. If the lesion is limited to motoneurons of the anterior horns of the dorsal brain, then spinal muscular atrophy (SMA) occurs - 7% of cases. With a rare (2% of cases) an isolated lesion of the corticospinal tracts develops the so-called primary lateral sclerosis (PBS). ALS accounts for more than 80% of cases of motor neuron.

Pathomorphology. Macroscopic changes in the spinal cord are minimal, and the brain the brain is practically absent. Microscopic examination in the spinal cord determines significant degeneration of the cells of the anterior horns, usually widespread, but the most expressed at the level of the cervical thickening. The total number of peripheral motoneurons reduced, predominantly α-motoneurons die. For unclear reasons, relatively the sacral motor neurons (Onuf's nucleus) remain intact, which innervate the external sphincters of the pelvic organs. This explains the ability of patients to control pelvic functions up to the last stages of the disease (the phenomenon of sparing sacral segments). Cellular degeneration is usually accompanied by secondary gliosis, less often - lymphocytic infiltration. The same changes are found in the motor nuclei of the lower part of the trunk. They are most pronounced in the nucleus of the hypoglossal nerve, the dorsal nucleus of the vagus nerve, the double nucleus of the IX-X nerves and the motor nucleus of the trigeminal nerve. The nucleus of the facial nerve less affected. The nuclei of the oculomotor, trochlear and abducens nerves are usually saved. In the cerebral cortex, degeneration of neurons of the precentral gyrus and adjacent sections of the frontal lobe in the third and fifth layers.

The described changes in the bodies of motor neurons are accompanied by damage to the white matter the anterior and lateral columns of the spinal cord, as well as the inner capsule. Moreover, the most significant lesions of the corticospinal tract, however, degeneration is also subject to spinocerebellar, rubrospinal, vestibulospinal and tectospinal pathways, and in the trunk brain - the lower legs of the cerebellum, medial longitudinal bundle, medial and lateral loops, reticular formation. Although sensory disturbances are uncommon in ALS, degeneration the posterior cords are often observed. The defeat of the fibers of the white matter of the hemispheres for the first time described by A. Ya. Kozhevnikov, who thus, according to Charcot, filled a significant lacuna in the pathomorphology of ALS.

In peripheral nerves, axonal degeneration with secondary demyelination. Reinnervation of denervated muscles is carried out due to the growth collaterals from the currently preserved motor axons. In the muscles is observed atrophy of denervated fibers. Moreover, the larger the group of atrophied fibers, the the disease progresses faster.

Epidemiology, etiology. The disease usually debuts at the age of 50-70 (average age patients about 57 years old), although an earlier and later onset is possible. In most cases the disease is sporadic and occurs in different geographic areas with a frequency 1.2-5 cases per 100,000 population. Men get sick more often than women (approximately 1.5: 1). Familial cases of ALS are known (5-10%), often with atypical manifestations: beginning in

childhood, isolated bulbar palsy, benign proximal paresis
extremities, etc. Pathomorphological changes in the nervous system in sporadic and
familial cases appear to be identical. There are several regions where ALS occurs
unusually often; in these cases, as a rule, atypical clinical
manifestations of the disease. So, on the island of Guam, in Japan on the Kii peninsula, as well as in the west
In New Guinea, there are familial cases of ALS, combined with parkinsonism and dementia.
The viral or genetic etiology of these ALS variants has not been proven. It is most likely that in

hitherto unknown environmental factors play a role in their origin.
In the classification of ALS, proposed in 1983 by M.A.Morariu, there are three options
diseases - sporadic classic ALS, familial and ALS complex - parkinsonism - dementia.
In turn, among family forms, there are “pure” cases of ALS and 6 variants of the disease, with
which the clinical picture is complemented by one or two syndromes: dementia, disorders
sensitivity, extrapyramidal signs, extrapyramidal and cerebellar
disorders, extrapyramidal and sensory defects, peripheral neuropathy. Type of
inheritance of family forms of the disease is autosomal dominant or autosomal recessive.
The etiology of sporadic ALS cases remains as mysterious as 100
years ago when Charcot described it. Hypotheses about the role of genetic, infectious,
immunological, toxic factors in the development of the disease have not been proven. Pointed to
connection of ALS with the HLA-A3 complex, especially in the case of a severe course of the
disease. Relatively
it is not uncommon to observe when spastic-atrophic paresis debuts in an injured hand
or operation. The opinion has been repeatedly expressed about the possibility of paraneoplastic
the origin of ALS, at least in some cases. However, it has now been shown that
detection of a malignant neoplasm in a patient with ALS indicates only
he has two independent diseases. Paraneoplastic origin may be
only the syndrome of spinal amyotrophy, while the lesion of the central motor neuron for
paraneoplastic conditions are uncommon. In recent years, a variant of ALS has been described,
in which
detect multiple blocks of conduction along peripheral nerves and antibodies to
neuronal gangliosides; in these cases, glucocorticoids are effective. Detection at 75%
patients with ALS antibodies to neuromuscular synapses indicates a possible role of autoimmune
factor in the genesis of the disease. This assumption is consistent with the fact that Charcot's
disease,
like myasthenia gravis and polymyositis, it is observed more often (compared with the
population) in patients with
Sjogren's syndrome (“dry syndrome”) - a caste rheumatologic autoimmune disease.
Clinic. There are 4 main forms of ALS: high, bulbar, cervicothoracic and
lumbosacral. Patients with a high form of the disease are most often observed
symptoms of damage to the corticospinal and corticonuclear tracts - spastic tetraparesis
and pseudobulbar syndrome, which are usually accompanied by mild anterior
violations. With the bulbar form, the bulbar and
pseudobulbar syndromes. The cervicothoracic form is characterized by atrophic and spastic
atrophic paresis of the hands and spastic paresis of the legs. In the lumbosacral form,
atrophic paresis of the legs with mild pyramidal symptoms.
The course of the disease is usually chronic, less often subacute. Its first symptoms may
to be weak in the distal parts of the hands, awkwardness when performing fine movements with
the fingers,
significant weight loss of limbs and fasciculations. Less often in the opening, weakness is noted
in

proximal arms and shoulder girdle, leg muscle atrophy and lower spastic paraparesis. In the bulbar form, the disease begins with dysarthria and dysphagia. Often ALS harbingers are cramps (in 30% of patients), often 3-6 months ahead of the main clinical manifestations.

Degeneration of peripheral neurons is clinically manifested by the development of weakness, atrophy, fasciculations. Fasciculations are common or are observed in limited muscle group, often capturing externally intact muscles. Only in rare cases of weakness and atrophy are not accompanied by fasciculations. In typical cases, weight loss starts asymmetrically with the thenar muscles of one of the hands, then muscles are involved in the process forearms, finger movements become awkward, the hand becomes "clawed". Later atrophy of the other hand develops for several months.

Sometimes, however, the disease begins symmetrically. Atrophy, spreading segment by segment, gradually captures the muscles of the shoulder and shoulder girdle. Developing in parallel damage to the bulbar muscles, fasciculations occur in the tongue, it quickly atrophies, the function of the soft palate, muscles of the larynx and pharynx is disrupted. Expression muscles, excluding the circular muscles of the mouth are affected to a lesser extent and much later than other muscle groups.

The same applies to the chewing muscles. As the disease progresses, become impossible to pull the lips into a tube and stick out the tongue. Due to paresis of the pharyngeal muscles, larynx, tongue, lips, the patient's speech is blurry, illegible, dysphonic, dispropodic (from Greek prosodia - melody). Swallowing is significantly difficult, fluid regurgitation often occurs. coy food through the nose. Patients can more easily swallow semi-liquid, rather than solid or liquid

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food. Oculomotor disorders due to damage to the nuclei of the III, IV, VI cranial nerves uncharacteristic and are extremely rare even in cases where the patient's life in for several years it is artificially supported with the help of mechanical ventilation. Sometimes in the early stages the disease develops weakness of the muscles - the extensors of the neck.

With lesions of the lower extremities, the disease begins in 25% of patients. Usually the first the process involves the anterior and lateral muscle groups of the lower leg, which is manifested by “hanging foot ”and steppage (pseudo-polyneuropathic variant of Patricos-Marie). Much less often the disease debuts with damage to the proximal muscles, which leads to myopathic manifestations. With any variant of the onset of ALS, atrophy generalizes over time. In development respiratory disorders in the late stages of ALS, the leading role is played by diaphragm paralysis.

Signs of damage to the pyramidal system appear already at an early stage of ALS; at first revitalization of reflexes is noted, followed by spastic paraparesis lower limbs. In the hands, the pyramidal component is usually manifested by an increase in reflexes, "Paradoxical" against the background of massive atrophy. This unusual combination is almost the most important clinical sign for suspecting ALS. E.K.Sepp drew attention to the special agitation of tendon reflexes inherent only in ALS. In general, the state of reflexes depends on what is most affected - the upper or lower motoneuron, and varies from a sharp increase to almost complete disappearance. As

degeneration of the pyramidal tract superficial abdominal reflexes persisting in ALS
they disappear incomparably longer than with multiple sclerosis. At the same time, deep
abdominal reflexes. An almost constant feature is pathological pyramidal signs. More often
reveal flexion foot signs (symptoms of Rossolimo, Bekhterev, Zhukovsky) than
extensor (symptoms of Babinsky, Oppenheim, Gordon). With the defeat of corticonuclear
pathways, pseudobulbar syndrome develops, manifested primarily by dysphagia and
dysarthria; the mandibular and pharyngeal reflexes are revived, reflexes of the oral
automatism, violent laughter or crying may occur. Pseudobulbar
the syndrome is often combined with bulbar, in this situation, the pharyngeal and mandibular
reflexes may decrease or disappear altogether. Revitalization of the mandibular reflex sometimes
is determined 5-6 months before the development of bulbar symptoms and thus is, along with
fasciculations in the language are the most important cerebral sign in the spinal stage of the
disease.
As already noted, the defeat of the sphincters is uncommon for ALS, but with far
the entered process can sometimes be noted incontinence or urinary retention. At the same time
sometimes
impotence develops early enough. There are no sensory disturbances. Occasionally
due to degeneration of the cells of the lateral horns of the spinal cord, Horner's syndrome occurs.
Often patients are worried about pain, especially at night, which may be associated with
crampi, joint stiffness (especially shoulder joints), prolonged immobilization,
hypoventilation, flexor and extensor spasms due to spasticity, depression,
reflex sympathetic dystrophy and other reasons.
Subcutaneous adipose tissue usually disappears in parallel with the development of muscle
atrophy.
In ALS, changes in the structure of collagen fibers of the skin were found, which explains
paradoxical absence of bedsores in bedridden cachectic patients. Intelligence
patients, as a rule, are not disturbed. However, the combination of ALS with frontal lobe is very
rare.
dementia and non-familial cases
Poliomyelitis-like syndrome (subacute progressive poliomyelitis of Aran-
Duchenne) - one of the variants of ALS, in which there are no signs of damage to the pyramidal
paths. The disease is more common in men (male to female ratio 3.6: 1).
In about half of the patients, the small muscles of the hands are symmetrically affected, gradually
in
the process involves the proximal parts. Much less often, the disease begins with a lesion
distal muscles of the legs or proximal extremities. N.V. Konovalov dedicated
subacute poliomyelitis Duchenne special monograph.
The nosological affiliation of the so-called
progressive form of tick-borne encephalitis, regularly mentioned in the domestic
See the literature when describing the differential diagnosis of the poliomyelitis-like variant of
ALS.
Bulbar form (progressive bulbar palsy) - a variant of motor disease
neuron, the first and main manifestation of which is a violation of bulbar functions,
most often due to a combination of bulbar and pseudobulbar syndrome. About
25% of ALS patients begin with bulbar disorders, which is considered unfavorable

a prognostic sign, since in these cases the disease is malignant and
leads to death within 1-3 years. Bulbar paralysis rarely occurs in isolation: during
Several months after the onset of the disease, certain ALS symptoms join it.
V.V.Mikheev observed 4 patients in whom ALS developed after removal
convexital meningioma.

An uncommon form of motor neuron disease is post-polio syndrome.
("Postpolio"), which occurs after 30-40 years in persons who have had acute poliomyelitis, and manifested by progressive muscle atrophy of those limbs that were the most affected in the acute stage of the disease. It is assumed that the "postpolio" -syndrome is based on aging process of reinnervated enlarged motor units. S. N. Davidenkov drew attention to the fact that the described syndrome was already known to Charcot.

Diagnostics. EMG is very important to record widespread damage to the cells of the anterior horns; while, as a rule, in two or more extremities show signs of denervation, fibrillation potentials, a decrease in the number motor units with the appearance of gigantic potentials. Somatosensory evoked the potentials and speed of conduction through sensitive fibers are usually normal. The speed of the guidance along motor fibers is reduced; the decrease may be insignificant (to the lower normal limits) or pronounced, especially against the background of massive atrophy of the affected muscles.

In this situation, special attention should be paid to the temperature of the examined limb. Have in healthy people, a decrease in temperature leads to a slowdown in the conduction of motor nerves. In ALS patients, the temperature of an atrophic and almost completely paralyzed limb decreases so much that it undoubtedly affects the speed of conduction. Generally conduction velocity along motor and sensory fibers does not change (if not positional neuropathy occurs due to compression). However, the identification of a decrease conduction velocity along motor fibers does not contradict the ALS diagnosis. it the circumstance is of particular practical importance in the case of the pseudopolyneuropathic form of ALS.

Sometimes with rhythmic stimulation, a myasthenic-like decrease in amplitude may occur motor responses.

When examining cerebrospinal fluid, a slight increase in protein levels. The content of CPK in plasma may be slightly (2-3 times) increased. How mentioned, antibodies to acetylcholine receptors are often found. CT scan not informative, MRI reveals little significant abnormalities.

Differential diagnostics. ALS needs to be differentiated from others diseases that cause damage to the upper and lower motor neurons and are accompanied by severe atrophy and bulbar disorders. With syringomyelia, in contrast to ALS reveal segmental sensitivity disorders and often nystagmus. Tumors of the cervical part of the spinal cord, manifested by flaccid paresis of the upper limbs and spastic paraparesis of the lower, often accompanied by severe radicular pain and conduction disorders of sensitivity. The introduction of CT and MRI has greatly simplified diagnosis of syringomyelia, spinal cord tumors and, to a certain extent, spondylogenic cervical myelopathy. If CT and MRI are not available, myelography is sometimes necessary. Before the emergence of CT and MRI great difficulties were caused by timely diagnosis craniovertebral tumors, in particular meningiomas of the foramen magnum.

Perhaps the most common reason for differential diagnosis with ALS is spondylogenic cervical myelopathy. For her, however, in contrast to ALS, disorders are characteristic deep sensitivity and lack of supraspinal signs. Sometimes differential diagnostic difficulties arise with pathological processes in the area of the upper aperture chest (in particular, cervical rib), trunk glioma, myasthenia gravis, polyneuropathies, polymyositis, Charcot-Geoffroy syphilitic amyotrophy, cervical "trimeningitis" (progressive compression of the cervical thickening in the inflammatory process, designated

previously as pachymeningitis), hyperparathyroidism, thyrotoxicosis, accompanied by myelopathy or
myopathy. It is pertinent to recall that thyrotoxic myopathy may be accompanied by fasciculations. Massive amyotrophy of the hand with tunnel neuropathy of the ulnar nerve and in advanced cases of compression of the median nerve in the carpal tunnel - perhaps the most a common situation when ALS is suspected. Locality of amyotrophy, absence of pyramidal signs and sensory defects (especially Tinel's symptom) allow a diagnosis to be made prior to use EMG. Obviously, the greatest difficulties are caused by the timely diagnosis of the lumbar

sacral form of ALS. Meanwhile, upon close examination for progressively weakening leg, along with massive amyotrophies, show the safety or even revitalization of the lazy and / or Achilles reflex. When benign fasciculations occur
(myokymia) in strong young people who do not have any other symptoms like as a rule, the patient and the doctor have an assumption about the onset of a fatal disease. Awareness of such a rare symptom complex allows you to confidently
overcome the reactive depression of the patient. Due to the lack of awareness of Kennedy's disease
it is usually misinterpreted as ALS.
The cornerstone of the differential diagnosis of ALS in doubtful cases
serves to identify symptoms of damage to the brain stem and segmental defects (atrophy, fasciculations) below the cervical thickening.
Forecast. 80-90% of patients die, usually from respiratory complications, within 3-5 years, the remaining 10% have a more benign course of ALS. They live longer than others patients with lumbosacral form. Life expectancy is shorter in patients with more old age and with involvement of bulbar muscles in the pathological process.
Treatment. There is currently no effective treatment for the disease.
The effectiveness of neuropeptides (thyroid-stimulating hormone releasing factor, dalargin) and amino acid blends have not been proven. The main one is symptomatic therapy, the purpose of which is -
to at least to some extent make life easier for the patient. Apply various orthopedic accessories: special collar to support the head, splints, gripping devices
items. Excessive exertion should be avoided while maintaining the strength for daily activities. In the presence of cramps, diphenin is prescribed at 300 mg / day, Relanium at a dose of 10-130 mg / day. When
severe spasticity, muscle relaxants are used. To “mitigate” bulbar disorders
Kalimin can be used at 30-60 mg 3 times a day. Equally in violent
crying sometimes helps amitriptyline. Significant inconvenience is caused by the accumulating due to impaired swallowing of saliva. In this case, it is recommended to apply anticholinergics, it is also important to keep the head in a position that makes it easier to swallow.
In the event of attacks of suffocation, in particular due to the ingress of saliva into the respiratory ways, patients are taught to tilt the body forward and cleanse the oral cavity, if possible use special portable suction devices.
The effectiveness of massage is problematic. The role of psychological
support of the patient, every day more and more aware of the hopelessness of his condition. Often in patients due to depression, daytime sleepiness, hypersalivation, and hypoventilation disturbances in nighttime sleep occur. In these cases, psychotropic drugs can help, in particular tricyclic antidepressants with anticholinergic properties. Important also find a comfortable position for the patient in bed. Diet Matters: Food
should be nutritious enough and at the same time not cause significant difficulties in swallowing, it is easiest for patients to swallow semi-liquid, jelly-like food. In some patients

there is a need to use a nasogastric tube or gastrostomy.
Treatment of constipation is important; in these cases it is preferable to use
glycerin suppositories and enemas, not regular laxatives. In case of severe pain, prescribe
non-steroidal anti-inflammatory drugs, and in the later stages, use is permissible
narcotic analgesics.
Based on the unique neuroresuscitation experience of the Institute of Neurology of the Russian Academy of Medical Sciences, L.M.
Popova believes that artificial ventilation of the lungs with amyotrophic lateral sclerosis impractical.

DISEASES OF THE VEGETATIVE NERVOUS SYSTEM

Vegetative and neuroendocrine disorders

Anatomical and physiological data. The purpose of the autonomic nervous system can be systematize in two directions: 1) retention of all functional parameters
activity of various systems within the boundaries of homeostasis, i.e. maintaining the constancy of internal
Wednesday; 2) vegetative provision of various forms of mental and physical activity, adaptation to changing external environmental conditions.
It should immediately emphasize the indissoluble unity of the autonomic and somatic nervous system, the coordinated activity of which ensures the maintenance of the physiological state of the body.
Based on the general plan of the organization of the nervous system, taking into account the segmental nature
the construction of all vertebrates, as well as the anatomical and functional features of the vegetative
nervous system, two levels of autonomic organization can be distinguished: segmental (peripheral) and suprasegmental (central). True vegetative apparatuses make up segmental autonomic system, they differ in the localization of nuclear-neuronal groups ferments, the speed of impulse conduction, the presence of efferent neural formations for outside the central nervous system, two-neuronal efferent part. Segmental autonomic nervous system
divided into sympathetic and parasympathetic.
The suprasegmental level of autonomic regulation is located in the brain and is an integral part of the integrative systems of the brain. It is impossible to single out specific vegetative centers, there are no morpho-functional features characteristic of autonomic nervous system. When these systems are irritated, autonomic responses are always arise in conjunction with the activation of other functional systems in the picture of a holistic a statement of conduct or a fragment thereof. Within this level, by the nature of the functional response
allocate ergotropic and trophotropic systems. Each brings together for the organization purposeful behavior activities of motor, sensory, emotional, endocrine and vegetative apparatus. With the help of suprasegmental integrated systems, segmental the vegetative system is included in the provision of various forms of activity, while it is perfectly coordinated with other systems.
Segmental autonomic nervous system. Sympathetic neurons are located in lateral horns and the intermediate zone of the thoracic and upper lumbar segments of the spinal cord.
Their axons with anterior roots leave the spinal canal and approach the sympathetic trunk (preganglionic with pronounced myelin sheath). Sympathetic trunk located on both sides of the spine and contains 20-22 paired nodes (3 cervical, 10-12

pectoral, 3-4 abdominal and 4 pelvic). There are three types of cells in the ganglia, which differ in their
size {large, medium, small). Preganglionic fibers are partially interrupted in
neurons of the nodes, partly go, without interruption, to the prevertebral ganglia. Vegetative fibers after switching in the ganglia are defined as postganglionic and differ
with a smaller thickness of the myelin sheath, and therefore have a lower speed
conducted pulse. Another station for switching them is the prevertebral nodes, or plexuses, containing mostly medium-sized neurons. The largest are the heart,
pulmonary, hypogastric and the largest is the solar plexus. Cervical sympathetic nerves innervate the head area, thoracic - mainly the trunk and visceral organs, and
lumbar - lower limbs. In the prevertebral plexuses, sympathetic
fibers that did not have contact with the neurons of the sympathetic chain, they also contain rasympathetic neurons. After passing through the plexuses, the vegetative fibers are already directly to the tissues of the innervated organs or approach the ganglia that take place in
the organs themselves (heart, gastrointestinal tract).
Parasympathetic neurons are located in the brainstem and lateral horns
of the spinal cord at the sacral level, at the level of the trunk these are the autonomic nuclei of the III nerve, the axons of it
go with fibers of the III nerve, interrupting in the ciliary node. Postganglionic fibers cause constriction of the pupil. The nuclei of the parasympathetic nerves that regulate lacrimation and salivation,
located in the pons of the brain. Preganglionic fibers are part of the VII nerve,
are interrupted in the pterygopalatine, submandibular and sublingual ganglia. Postganglionic neurons

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provide lacrimation, secretion of mucus in the nasal passages and salivation. The core of the lower
salivary nucleus are located in the bulbar part of the brain stem, their preganglionic
fibers leave the skull with the IX nerve, interrupting in the parotid ganglion. Postganglionic vo-
the locks are directed to the parotid gland.
In the X nerve, autonomic nuclear formations dominate over sensorimotor,
controlling the activity of organs located in the chest and abdominal cavities. Pre-
ganglionic fibers leave the skull through the posterior laceration and are included in neurovascular cervical bundle. In its course, the vagus nerve gives off fibers at the level neck, chest, abdominal cavity, which are interrupted in the plexuses located
near the working bodies. The left vagus nerve primarily innervates the stomach,
pancreas and hepatic-vesical system, right - intestines.
Pelvic parasympathetic nerves originate from neurons lying in the lateral horns
sacral segments. Their preganglionic axons terminate in ganglia located
close to the pelvic organs. There are observations that parasympathetic preganglionic fibers leave the spinal cord as part of not only the anterior (this is typical for sympathetic fibers), but also the posterior roots.
The afferent part of the autonomic nervous system is less well understood. There are two types sensory autonomic receptors: 1) receptors that respond to pressure and stretch (bodies Vatera-Pacini); 2) chemoreceptors. The fibers coming from the receptors have little
myelin sheath and go along with efferent vegetative fibers. Their neurons
located in the spinal ganglia. Further afferentation goes along the spinothalamic tract
with conductors of deep sensitivity. At the level of the spinal cord, differentiate
sensory animal and vegetative fibers fail. Experiments with irritation
internal organs indicate that evoked potentials can be registered in
different areas of the cortex. There is evidence of the role of premotor fields. Pain-carrying

vegetative fibers are not found in the vagus nerve, so vegetative pain
are rightly designated as sympathetic.
By their morphological structure, sympathetic and parasympathetic neurons and their axons are indistinguishable. They differ in the grouping of centers along the central nervous system (thoracic region -
sympathetic, stem-sacral localization - parasympathetic), different remoteness
ganglia from the working organ (sympathetic in the distance, parasympathetic - in the immediate proximity), which determines the greater diffuseness and generalization of sympathetic influences.
Finally, mediator differences are very significant. Acetylcholine is secreted in all intermediate ganglia of the autonomic nervous system and in postganglionic fibers its parasympathetic link. In postganglionic sympathetic fibers (with the exception of sweating) norepinephrine and adrenaline are released. It is important that the functional effect is determined by the interaction of the mediator with the receptor. Two types of adrenergic receptors have been identified -
alpha and beta. Norepinephrine affects α-receptors, and adrenaline affects α- and β-receptors.
Suprasegmental vegetative formations. Essentially on irritation of any department of the brain, you can get autonomic responses, which are part of the polysystem reactions. However, to distinguish specialized autonomic centers in the brain, in addition to described as part of the segmental vegetative system is impossible. As part of the integrative formations of the brain, three formations are of greatest importance: the brain stem brain, hypothalamus and rhinencephalon.
In the brain stem, there are three types of formations involved in the regulation vegetative functions: 1) parasympathetic nuclei, the axons of which go from III, VII, IX and X cranial nerves belonging to the true segmental autonomic nervous system; 2) semi-specialized formations, which include the vasomotor center, which has its composition depressor, pressor, accelerator and inhibitory centers that regulate blood pressure and heart rate, and the respiratory center with expiratory and inspiratory neural formations. Semi-specialized these formations
indicated because, together with autonomic cardiovascular and respiratory shifts the facilitating and inhibiting supraspinal influences on the spinal apparatus change.
It is obvious that these devices carry out the integration of vegetative-motor functions; 3) reticular
formation of the brain. Participation of the latter in the regulation of shift and maintenance of sleep and

wakefulness are obvious, and these processes are well coupled with vegetative accompaniment different levels of wakefulness, phases and stages of sleep. The reticular formation carries out communication and
coordination of ascending (level of wakefulness) and descending (autonomic and motor) influences.
An important link in the regulation of the autonomic nervous system is the hypothalamus, traditionally but misidentified as the supreme vegetative center. Inside the hypothalamus allocate specific and nonspecific departments. The first includes groups of neurons, projecting onto the pituitary gland, the effect of irritation or destruction of which is specific, and a distinctive feature of neurons is the ability to neurocrinia.
In this case, either the triple hormones themselves are formed (antidiuretic hormone - ADH), or factors that stimulate or inhibit the secretion of triple hormones in the adenohypophysis. TO specific divisions also include chemoreceptors that receive signals from the internal environment (glycoreceptors, osmoreceptors, etc.).

Disruption of the functioning of these departments is manifested in the clinic as distinct and characteristic neuroendocrine metabolic syndromes. Another part of the hypothalamus is continuation of the reticular formation of the brainstem and can be indicated nonspecific, since its irritation leads to a complex of psychophysiological shifts. Everything this led to the idea that the hypothalamus is an important integrative part of the brain, the center neurohumoral coordination, a link that includes hormonal systems in a holistic organization of behavior, ensuring the interaction of vegetative, endocrine and emotional components of the body.

Interest in rhinencephalon is determined by the presence in it of such structures as amygdala complex, hippocampus, medial-basal cortex of the temporal lobe, which are formations that are part of the functional limbic system. The latter is determined and as the “emotional brain” (Konorsky), and as the “visceral brain” (McLean). Were received data on the role of these formations in the organization of motivational, mnestic and emotional processes. The role of these systems in emotional-vegetative integration is revealed, vegetative provision of already holistic forms of behavior.

In general, all these formations make up the limbic-reticular complex: integrative apparatus of the brain, providing adaptive goal-directed behavior. Wherein ergotropic systems provide active physical and mental activity, adapt to changing environmental conditions, provide reactions to stress, contribute to the course of catabolic processes. The degree of complex (sensorimotor, endocrine and autonomic) reactions are determined by the importance, significance, novelty of the situation, the level of motivational stress. In the vegetative sphere, at the same time, pre- property sympathetic-adrenal link.

Trophotropic systems carry out anabolic, nutritional, endophylactic reactions during rest, relaxation, phases slow sleep, under external circumstances that do not require overcoming, struggle with them. All the activity is aimed at maintaining homeostatic balance. The same complex polysystemic reactions behave in the opposite way (muscle relaxation, decreased sensory tension and secretion of "stress" hormones), mainly vagoinsular orientation of vegetative shifts.

It is important to emphasize that the pathology of suprasegmental formations causes not only vegetative shifts, and always a complex of polysystemic reactions, where vegetative disorders are not are deterministic (neither in form nor in localization).

Pathogenesis of vegetative disorders. The division of the autonomic nervous system into the segmental and suprasegmental departments have not only theoretical, but also important practical pathogenetic significance. The pathogenesis of segmental disorders is determined by processes irritation and loss of certain links and their localization. Syndromes are described lesions of cerebral autonomic centers, preganglionic fibers, sympathetic chain, peripheral plexuses, damage to autonomic nerves and fibers in mixed nerves (autonomic neuropathy).

At the heart of vegetative disorders of suprasegmental genesis is a violation integrative activity, defined as "disintegration syndrome". The manifestation of it is a mismatch in the activity of various brain systems (sensorimotor, emotional, vegetative), and vegetative dysfunction reflects a violation of the adaptive, adaptive

activities.

SYNDROME OF VEGETATIVE DYSTONIA

The clinical doctrine of the pathology of the autonomic nervous system is associated with the names
Eppinger and Hess, who created the concept of vagotonia syndrome. Subject to separation
the autonomic nervous system into the sympathetic and parasympathetic divisions soon appeared
description of the second generalized autonomic syndrome - sympathicotonia.
The doctrine of sympathicotonia and vagotonia was often criticized, the basis of which
formed an idea of the absence in real practice of such pure syndromes.
Indeed, more often one has to deal with mixed sympathetic and
parasympathetic manifestations, however, it is often possible to identify the predominant direction
laziness in certain functional systems (for example, sympathetic activation in
cardiovascular and parasympathetic in the gastrointestinal systems).
The second principle of separation is associated with the permanence and paroxysmality of vegetative
violations. If the latter are intense, outlined in time
"Vegetative storms", then the designation of the remaining violations as permanent to a certain extent
conditionally. All vegetative symptoms are labile, mobile. This also applies to hyperhidrosis, and
heart rate, and blood pressure. Thus, permanent violations are not
absolutely stable vegetative indicators, but they fluctuate within limits that do not reach
the degree and intensity of vegetative crises.
Sympathetic-adrenal crises are characterized by unpleasant sensations in the area
chest and head, tachycardia, rise in blood pressure, mydriasis, chill-like hyperkinesis,
pronounced feelings of fear, anxiety. The attack ends with polyuria with light urine.
Vagoinsular (parasympathetic) crises are manifested by dizziness, nausea,
decrease in blood pressure, sometimes bradycardia, extrasystole, difficulty breathing, gastrointestinal
intestinal dyskinesias.
More often crises are mixed, when signs of sympathetic and
parasympathetic activations occur simultaneously or follow one another. In for-
In borderline studies, these conditions are referred to as "panic attacks."
Vegetative disorders can be generalized, systemic, or local.
The former manifest themselves simultaneously in all visceral systems, and include cutaneous
vegetative disorders, and thermoregulation disorders. Often vegetative manifestations
predominantly capture any one system, most often cardiovascular, the most
dynamic and psychologically significant for the patient. In Anglo-Saxon literature, this
the syndrome is referred to as “neurocirculatory asthenia”. There are other synonyms for the name
this syndrome: "soldier", or "irritable heart", "De Costa syndrome", "syndrome
efforts ”,“ instability of the heart ”,“ tachycardic neurosis ”,“ vasoregulatory asthenia ”.
Palpitations, pain in the left side of the chest, asthenia, irritability,
sleep disturbances, headache, dizziness, paresthesia, belching. Distinct somatic
violations can not be detected.
In recent years, hyperventilation syndrome has been widely covered and studied.
also described by terms such as "hyperventilated anxiety", "nervous
respiratory syndrome "," cardiorespiratory tethane-like syndrome ", etc. Classical
its sign is a triad - increased breathing, paresthesia and tetany, and the most typical and
a vivid manifestation - a hyperventilation crisis, characterized by anxiety, sensations
shortness of breath, chest tightness, shortness of breath, lump in the throat, other

vegetative-visceral disorders. The most pronounced are respiratory
disorders. There is dissatisfaction with inhalation, despite increased breathing, patients
they do not tolerate a stuffy room, they constantly strive for fresh air. All these phenomena
sharply exacerbated in situations of anxiety (exam, public speaking, transport,
especially metro, height).
Respiratory disturbances are accompanied by palpitations, a feeling of constriction and chest pain,
are combined with migraine and fainting, playing a role in their genesis. Often
increased peristalsis, belching, aerophagia, and abdominal distention are found. A special place in
the structure of hyperventilation syndrome is occupied by muscle-tonic manifestations in the form
normocalcemic variant of latent neurogenic tetany; the latter is manifested

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carpopedic spasms in the picture of vegetative crises and is detected using clinical
signs of increased neuromuscular excitability (Khvostek's symptoms, Trousseau's symptom - Bonsdorf), as well as during electromyographic research.
Hyperventilation syndrome leads to hypocapnia and alkalosis, which plays
an essential role in the formation of symptoms in autonomic disorders. Similar
increased neuromuscular excitability (latent tetany) plays. Described and
"Neurodigestive asthenia", or "neurogastric dystonia", where in the foreground
subjective complaints from the digestive tract appear, but objectively there is
dyskinetic syndrome.
Autonomic disorders can predominantly manifest themselves in the thermoregulatory environment:
prolonged non-infectious subfebrile conditions, febrile crises. Local vegetative
disorders can occur in one half of the head, distal extremities,
mainly in the form of lateralized manifestations on the trunk and limbs.
Sympathetic, parasympathetic and mixed symptom complexes (permanent and
paroxysmal), wearing generalized, mainly systemic or local
character, are combined into a syndrome of autonomic dystonia.
Autonomic dystonia syndrome is not a nosological form and is only syndromic
reflects the presence of constitutional or acquired autonomic dysfunction. Diagnostics
it consists of two stages:
1) in the presence of characteristic complaints and certain objective symptoms of a disorder
functions of various body systems, it is necessary to exclude organic pathology
certain visceral systems. Thus, the diagnosis is based on a positive analysis.
existing manifestations of the disease and the exclusion of somatic organic disease. How
as a rule, this stage of diagnostics does not cause difficulties;
2) more complex is nosological and topical (determining the level of damage)
analysis of the syndrome of vegetative dystonia. However, it is necessary from both theoretical and practical
technical positions. Sufficient resistance of vegetative disturbances is well known, difficult
their curability. All this is often the result of attempts to treat directly
autonomic disorders without regard to their nature.
Recently, there has been a tendency to view neurocirculatory dystonia as
an independent disease. This point of view is wrong. Neurocirculatory
dystonia is an integral part of the general syndrome of autonomic dystonia, usually
combined with a violation of autonomic regulation and other visceral systems, as well as vessels,
has a nature in common with all forms of vegetative dystonia.
With a certain degree of schematicity, 8 factors can be distinguished that cause vegetative

violations.
1. Syndrome of vegetative dystonia of constitutional nature. It usually manifests itself with early childhood and is characterized by instability, lability of vegetative parameters. Fast discoloration of the skin, sweating, fluctuations in heart rate and blood pressure, pain and dyskinesia in the gastrointestinal tract, tendency to subfebrile condition, nausea, bad tolerance of physical and mental stress, meteotropy. Eppinger and then Guillaume defined such persons as “disabled of the vegetative system,” not yet sick, but prone to the elimination of all these manifestations with the adverse effects of the external environment. Often these
disorders are family-hereditary. With age, these persons
correct tempering education achieve a certain compensation, although all their life remain vegetatively stigmatized. There are also very difficult constitutional vegetative disorders. We are talking about familial dysautonomy, Riley-Day syndrome, in which gross violations occur in the internal environment of the body, incompatible with life, and in the pathological process significantly involved the peripheral autonomic system.
2. Syndrome of autonomic dystonia arising against the background of endocrine rearrangements organism. It manifests itself during puberty and menopause. At puberty, there are two preconditions for the emergence of autonomic syndromes: the emergence of new endocrine vegetative interactions requiring the formation of other integrative patterns, and fast, often accelerated growth gain; this creates a gap between new physical parameters and capabilities of vascular support. Typical manifestations are vegetative disorders against the background of mild or severe endocrine disorders,

fluctuations in blood pressure, orthostatic syndromes with pre-syncope and fainting conditions, emotional instability, thermoregulation disorders.
Vegetative processes are exacerbated during menopause, which is associated with physiological endocrine and emotional accompaniments of this state. Vegetative disorders
are both permanent and paroxysmal in nature, and among the latter, in addition to the characteristic
hot flashes, feelings of heat, profuse sweating, vegetative-vascular crises may occur.
It should be emphasized that both menopause and puberty are characterized by significant psychological
restructuring. Considering this fact, we can assume that vegetative disorders are based on endocrine and psychological factors.
3. Syndrome of vegetative dystonia in primary lesions of the visceral organs. Speech is about diseases that do not have a leading neurogenic factor in their pathogenesis. TO these include, for example, gallstone disease, chronic pancreatitis, diaphragmatic hernia, chronic appendicitis, urolithiasis.
The mechanisms causing autonomic disturbances are reduced to irritation of autonomic receptors available in these organs, the involvement of the nearest vegetative formations, chronically existing algic syndrome. With chronic course
diseases occur first, reflex local, and then generalized
vegetative disorders. The cure of the underlying disease is often accompanied by or improvement, or disappearance of autonomic dysfunction.
4. Syndrome of vegetative dystonia in primary peripheral diseases
endocrine glands (thyroid, adrenal glands, ovaries). Decrease or increase in secretion these glands entail disturbances in the endocrine-vegetative balance. Release into blood active biological substances (thyroxine, catecholamines, steroids, insulin), closely interacting with autonomic systems, a decrease in their secretion are factors that contributing to the occurrence of vegetative disorders of a generalized nature.
5. Allergy is one of the causes of vegetative disorders. She happens to be

the result of many factors: mass vaccinations, environmental changes, use
preparations that are products of organic chemistry, contact with food in everyday life
chemical industry. The role of psychogenic factors, chronic stress
situations in the formation and course of allergies. The autonomic nervous system, on the one hand,
participates in the pathogenesis of the formation of vegetative disorders. Known role in this regard
insufficiency of sympathetic-adrenal influences. On the other hand, the formed
allergies are accompanied by distinct autonomic disorders, often of the nature
deployed sympathetic-adrenal crises.
6. Syndrome of autonomic dystonia in pathology of segmental autonomic nervous
systems. The latter consists of autonomic centers located in the central nervous system (autonomic nuclei
III, IX and X cranial nerves, lateral horns of the spinal cord), preganglionic and
postganglionic fibers, sympathetic chains and autonomic plexuses. Severe, often
vital disorders of the respiratory and cardiovascular systems are detected when
pathology of the bulbar parts of the brain stem. The clinical significance of autonomic disorders in
spinal cord damage (tumor process, syringomyelia) is relatively small, and they
overlapped by massive movement and sensory disorders.
More often than others, preganglionic fibers are involved in the process at the level
anterior roots of the spinal cord. As a rule, the cause of autonomic disorders is
is osteochondrosis of the spine. Emerging radicular disorders include and
sympathetic manifestations, and vegetative-vascular symptoms, which can be as
local, mainly in the affected area of the roots, and generalized. Especially
this refers to the complications of cervical osteochondrosis, which often occur
vegetative-vascular crises associated with the involvement of vegetative plexuses in the process
vertebral artery (posterior cervical sympathetic syndrome, cervical migraine, Barre syndrome).
Pathology of the anterior roots and vegetative fibers passing with them can
manifest itself in a number of pseudovisceral syndromes in which pain occurs
specific localization. The most studied syndrome of "cervical angina", or syndrome
"Anterior chest wall", manifested by pain in the left half of the chest with
irradiation to the left arm, scapula, sometimes the left half of the head. Clinically this syndrome
you can distinguish from true angina pectoris by the following signs: pains are long-term

character, aggravated by excitement and less associated with physical activity, localized not for
sternum, and in the apex of the heart, are tolerant to antispasmodic drugs, but can
be relieved by pain relievers; there are no changes on the ECG, there are
signs of cervical osteochondrosis, tension and soreness of the pectoral muscles.
Given these differences, it should be remembered that this syndrome is more common in humans.
middle and old age and can be combined with true coronary insufficiency. Can
painful sympathetic phenomena occur in the abdominal cavity, imitating diseases of the internal
renny organs. It should be noted that the organic pathology of the visceral systems has
a certain influence on the occurrence of lateralized root-sympathetic and
myofascial syndromes. It is known that the latter more often occur at the cervical level on the left.
Right-sided lesions are usually accompanied by pathologies of the liver and biliary tract.
Unilateral pulmonary processes, urolithiasis, chronic
appendicitis, ovarian pathology.
7. Syndrome of vegetative dystonia with organic brain damage.

Almost always, with any form of cerebral pathology, vegetative
disorders, however, they are most pronounced when the deep brain systems are affected
(brain stem, hypothalamus and rhinencephalon), which are important structural links
limbic-reticular complex.
With an interest in the caudal parts of the brain stem, in particular the vestibular
complex, vestibulo-vegetative disorders are most clearly manifested. In emerging
this crisis has two features. Often the onset of a crisis is dizziness, and in
the paroxysm itself is dominated by vagoinsular manifestations.
With the defeat of the mesencephalic structures, sympathetic-adrenal
paroxysmal and permanent disorders, close to those observed with
hypothalamic insufficiency. This is due not only to the topographic proximity, but also
close functional connection of the oral divisions of the trunk and the hypothalamus. Essential for
practitioners have hypothalamic dysfunction. Due to the current trend towards
overdiagnosis of hylothalamic syndromes, it became necessary to formulate
diagnostic criteria. They are the following: 1) neuroendocrine syndromes in
exclusion of primary damage to peripheral endocrine glands; 2) motivational
disturbances (hunger, thirst, libido changes); 3) neurogenic disorders of thermoregulation; 4)
some forms of pathological drowsiness. Each of the selected criteria becomes
pathognomonic with the exclusion of endocrine, visceral and neurotic disorders.
It is important to emphasize that even pronounced autonomic disorders in the form of vegetative
crises
(they are often and incorrectly called diencephalic) are not sufficient for diagnosis
hypothalamic pathology, but this does not deny the presence of hypothalamic syndrome
bright permanent and paroxysmal disorders, combined with the above
pathognomonic manifestations. Usually, sympathetic reactions predominate.
The defeat of the rhinencephalic region is manifested primarily by the temporal lobe syndrome
epilepsy. Unlike all autonomic disorders described so far,
non-epileptic nature, autonomic disorders in temporal lobe epilepsy can be included in
model of an epileptic seizure as its aura. The most typical are
abdominal (sharp pain in the epigastric region) or cardiovascular (unpleasant
sensations in the region of the heart, arrhythmias) manifestations. Permanent disorders are not
clearly expressed,
are often subjective. Vagoinsular reactions predominate.
There are combined rhinencephalic-hypothalamic lesions.
8. Neuroses and syndrome of vegetative dystonia. Neuroses are more often than other reasons
a factor causing autonomic disorders. This is due to the fact that neuroses
are the most common diseases, and autonomic reactions are
their obligate manifestations. A special connection between the vegetative and emotional spheres
is noted
long enough. Recently, this is reflected in the allocation of psychovegetative
syndrome. At the same time, the obligatory combination and the pathogenetic relationship of
these
violations. In neuroses, emotional disturbances are primary, and vegetative
follow them. The latter depend on the form and intensity of neurotic disorders. Practical
it is important to remember that vegetative dystonia is accompanied by asthenic, depressive,
phobic, hypochondriacal manifestations, sleep disturbances. Clinical manifestations of vegetative

disorders are described in the neuroses section. Only a few factors need to be emphasized. At
neuroses, there is a bright permanent and paroxysmal dysfunction, either
polysystemic, or predominantly monosystem in nature. The presenters are sympathetic
adrenal manifestations. An important place in the picture of vegetative disorders is occupied by

hyperventilation and tetanic disorders.
In recent years, the term
"panic attacks". The clinical picture of these attacks is characterized by a combination of the following
manifestations: shortness of breath, palpitations, discomfort or pain behind the breastbone, sensation
choking or shortness of breath, dizziness, unsteadiness in the upright position
or an unsteady "wobbly" gait, a feeling of unreality in the environment, and
what is happening, paresthesia, hot or cold flashes, hyperhidrosis, fainting
states, trembling or chill-like hyperkinesis, fear of death, fear of “going crazy”. How
it can be seen that there is a detailed picture of psychovegetative paroxysmal syndrome, similar to
clinic of vegetative crises.
It is considered that the diagnosis of "panic attacks" is reliable if there is a minimum of 4
of these symptoms, and they occur with a frequency of at least 3 times every 3 weeks.
In the American diagnostic classification, they are separated into a separate subgroup in
class of anxiety disorders, and in the International Classification of Diseases, Injuries and Causes
death of the 9th revision named three causes of "panic attacks": anxiety neurosis, depressive non-
vroz and affective psychoses. Thus, according to international experience, the role of
neurotic disorders in the genesis of autonomic disorders of a paroxysmal nature.
The connection between "panic attacks" and agoraphobia is indicated: "panic attacks" are detected
in about 50% of people with this form of phobias.
Vegetative-visceral disorders most often occur in the cardiovascular system and
are manifested by algic, dysrhythmic and dysdynamic disorders.
Cardiac syndrome manifests itself in a variety of unpleasant painful
sensations in the left side of the chest. Their feature is the duration
course, the occurrence of emotional, not physical stress, the lack of effect from
antispasmodic therapy. Patients are usually fixed on their feelings, often refer
to a doctor, they are afraid to leave for places where they will be deprived of medical care, they constantly carry with them
medicine. In severe cases, a pronounced cardiophobic syndrome develops. Objectively
the lability of blood pressure is determined, its pronounced fluctuations during the first and
subsequent measurements.
Sinus tachycardia is characteristic, bradycardia is less often noted. Rhythm disorders wear
the nature of the extrasystole; ECG changes in neuroses are minimal, determined
satisfactory exercise tolerance.
The respiratory system is characterized by hyperventilation syndrome, which occurs against the background
feelings of incomplete inhalation and lack of air. Canine attacks may also appear.
breathing that mimics an attack of bronchial asthma. The most demonstrative are paroxysmal
neurotic laryngospasm (spasm of the muscles of the larynx), often associated with the act of eating.
In the gastrointestinal tract, there may be increased salivation, muscle spasm
esophagus with difficulty in passing food and vivid subjective sensations, aerophagia -
belching as a result of swallowing air with food, neurotic regurgitation or vomiting, pain
sensations in the abdomen - gastralgia, or abdominalgia, intestinal disorders in
in the form of diarrhea, diarrhea, changes in peristalsis.
Violations of vascular permeability in the form of symmetric or
lateralized hemorrhages in the skin, erosion in the gastrointestinal tract. Rarely
there is a syndrome of "bloody sweat".
Moderate neuroendocrine disorders are determined: thyroid dysfunction,

genital, pancreas. Motivational disorders are characteristic - a change in appetite, up to anorexia, decreased libido.
Thus, when detecting vegetative dystonia syndrome, it is necessary to establish factors that play a leading role in its genesis. This analysis is of paramount importance. practical value, as it determines the therapeutic tactics of the doctor. Based on this, autonomic dystonia syndrome cannot appear as the main clinical diagnosis. Examples of correct wording are as follows: neurasthenia, autonomic syndrome dystonia; allergy, vestibulopathy, vegetative dystonia syndrome; hypothalamic

insufficiency, neuroendocrine form, vegetative dystonia syndrome, etc. etc.
Treatment includes therapy for the underlying disorders that cause autonomic disorders, and the effect on autonomic manifestations.
In constitutional insufficiency and pubertal disorders, the leading role assigned to general strengthening and hardening activities (exercise therapy, water procedures). If in
in these situations, this approach is decisive, then you should remember the need inclusion of hardening in complex therapy for all patients with autonomic dystonia.
Treatment of the underlying disease is necessary in case of damage to internal and endocrine organs, menopause, allergies. Given the psychovegetative nature of the disease, is given to the normalization of the emotional sphere. In the case of neurotic disorders, all forms of psychotherapy, elimination or deactualization of traumatic factors, timely spa treatment.
Psychopharmacological treatment is prominent. Widely used for the presence of anxiety, fears, increased irritability, tranquilizers, as well as small antipsychotics. Tranquilizers are especially effective for stopping paroxysm. Drugs of choice for eliminating "panic attacks" are antidepressants, clonazepam (antelepsin) - 1.5-Zmg / day, alprazolam - 1.5-3 mg / day, used as a course of treatment (<1-2 months).
The use of antidepressants should be differentiated: in the case of anxiety, agitated depression, amitriptyline is shown (tryptisol 50-75 mg / day), with asthenic forms of depression - imipramine (imizine, melipramine) 50-100 mg / day. With pronounced hypochondria
other tendencies are prescribed teralen up to 20-40 mg / day, melleril at 30-50 mg / day. Used by a combination of several drugs: most often tranquilizers and antidepressants. Should prescribe sufficient, individually adjusted doses; sometimes the effect is weak affected by the use of small doses.
The appointment of vegetotropic drugs is determined by the nature of the interictal symptoms and
type of paroxysm. More often, enhanced sympathetic activation is detected, which determines the importance of prescribing β-blockers (anaprilin 40-120 mg / day, etc.) and α-blockers (pyrroxane at 30-90 mg / day). The blocking effect on the periphery is also exerted by ganglion blockers (gangleron, pentamin). With enhanced vagoinsular activation, anticholinergics of central and peripheral action (amizil at 3-5 mg / day, cyclodol at 4-5 mg / day) and ganglion blockers. Complex preparations have not lost their significance: belloid, bellaspon, bellataminal, which have a normalizing effect on both parts of the vegetative nervous system. In the presence of hyperventilation disorders, it is necessary to conduct respiratory exercise therapy, breathing training with the inclusion of the abdominal cavity, the development of correct
the ratio between the duration of inhalation and exhalation (1: 2).
Special treatment is provided for neurological diseases that determine the occurrence of autonomic disorders - radicular manifestations of osteochondrosis,

vestibulopathy, temporal lobe epilepsy.
It should be emphasized that the leading role in the treatment of vegetative dystonia is played along with
causal therapy the use of rational psychotherapy, psychotropic and vegetotropic
drugs.

MIGRAINE

Migraine is a disease caused by hereditary dysfunction
vasomotor regulation, manifested mainly in the form of periodically repeating
headache attacks, often in one half of the head.
The frequency of migraine in the population is, according to various authors, from 1.7 to 6.3% and
more. The disease is observed mainly in women. The relevance of studying this
problems are caused not only by the significant prevalence of migraine at a young age, but and the severity of paroxysmal manifestations.
The description of migraine is found in the works of doctors of the XVI-XVIII centuries, although the most complete
information was presented in the second half of the last century. Hereditary
the nature of the migraine. The disease is inherited in an autosomal dominant manner. In implementation
disease appears to involve a large number of typical and paratypical
factors, which explains the significant clinical polymorphism of migraine with intrafamily

similarities and differences between families.
Pathogenesis. The pathogenesis of the disease is complex and has not yet been fully elucidated. but
it is undoubtedly that with migraine there is a special form of vascular dysfunction, manifested generalized disorders of vasomotor innervation, mainly in the form
instability of the tone of cerebral and peripheral vessels. The center of gravity of these disorders is located in the extra- and intracranial vessels of the head. Peak vasomotor disorders represented by a migraine attack, which is a kind of cranial vascular
crisis. Headache during a migraine attack is caused by vasodilation of the solid
the meninges, an increase in the amplitude of the pulse oscillations of the vascular wall. Proven phasicity in the development and course of a migraine attack.
During the first phase, vasospasm occurs, while there is also a decrease in
blood supply to the vascular walls themselves and they become especially sensitive to stretching.
In the second phase - dilatation - there is an expansion of arteries, arterioles, veins and venules, increases
the amplitude of the pulse oscillations of the vessel walls. The first phase is most clearly expressed in
intracerebral and retinal vessels, and the second - in the branches of the external carotid artery - meningeal, temporal, occipital. In the next, third, phase, vascular edema develops.
walls and periarterial tissues, which leads to the rigidity of the vessel walls. In the fourth phase the reverse development of these changes occurs. The actual pain sensations are associated mainly with the second (throbbing pain) and third (dull pain) phases of the attack, which found confirmation in the data of angiographic and radioisotope studies of patients during time of migraine attack.
In the genesis of an attack, another mechanism also plays a role - expansion
arteriovenous anastomoses with signs of shunting and “stealing” of the capillary network.
In the pathogenesis of migraine, a significant role is assigned to metabolic disorders of a number of biologically
active substances, especially serotonin, the excessive release of which from platelets

causes the first phase of migraine paroxysm. In the future, due to intensive
denial of serotonin by the kidneys, its content in the blood decreases, which is accompanied by a drop in tone
arteries and their expansion. The importance of serotonin in the pathogenesis of migraine is confirmed, firstly,
the provoking effect of the introduction of exogenous serotonin on a migraine attack and, secondly,
pronounced vasoconstrictor effect of drugs with antiserotonin
action, which is confirmed by angiography. Along with this, there is a hypothesis linking pathogenesis of migraine with impaired tyramine metabolism. Due to hereditary deficiency tyrosinase and monoamine oxidase, an excess of tyramine is formed, displacing norepinephrine from its
reserves. The release of norepinephrine leads to vasoconstriction, while a contributing factor is a functional failure of certain vascular areas of the brain. In the next
phase, suppression of the functions of the sympathetic system occurs and, in connection with this, excessive expansion
extracranial vessels. The pain mechanism includes the release of neurokinin - a substance P, which has a suppressive effect on the anti-algogenic system of endorphins. In the same time some researchers are considering an increase in blood norepinephrine levels before
migraine attack as a manifestation of dysfunction of the hypothalamic-pituitary-adrenal system. Perhaps the concept of denervation hypersensitivity is also applicable to migraine,
arising from constitutional chronic insufficiency of monoamines -
serotonin and norepinephrine. In this case, a migraine crisis can be considered as excessive response to a temporary increase in the content of these substances in the blood.
There are also indications of an increase in the content of histamine and acetylcholine in the blood during
time of migraine attack. An increase in the content of quinines in the walls of arteries and perivascular spaces, which is accompanied by increased vascular permeability.
Serotonin and histamine released at the onset of migraine are also thought to increase permeability of the vascular wall, at the same time increases the sensitivity to algogenic
the effect of plasmokinin with a decrease in the threshold of pain sensitivity of receptors of the vessel walls.
Some authors believe that in the development of the first phase of migraine (vasoconstriction) a certain
the role is played by prostaglandins.
Recently, a fundamentally different concept of migraine has been proposed, which has clinical and experimental confirmation that links the onset of an attack with
neurophysiological mechanism - Leo's spreading cortical depression. The last
arises due to the fact that during migraine, neuronal-glial exchange of glutamate is impaired,

the level of which rises in the blood during migraine, especially at the onset of the crisis.
Much attention has been drawn to studies that have shown that cerebral arteries
are innervated not only by vegetative fibers, but also by fibers of the trigeminal nerve. And although
the reasons for the stimulation of the trigeminovascular system in headaches are not yet clear, participation
this mechanism in the development of migraine attacks is undoubtedly.
Since migraine attacks in many patients are closely related to
menstrual cycle ("menstrual migraine"), in recent years, studies have been carried out
the content of progesterone and estradiol in the blood plasma of women during the entire menstrual period

cycle. The dependence of a migraine attack on a decrease in the amount in the blood was found progesterone and an increase in estradiol.

Clinic and diagnostics. The disease in most patients begins in puberty period. The main clinical manifestation of the disease is a migraine attack.

According to the International Classification of Headaches, Cranial Neuralgia and Facial pain (1981), the following main forms of migraine are distinguished:

migraine without aura (simple form)
migraine with aura (classic form of migraine)
migraine with typical aura
migraine with prolonged aura
familial hemiplegic migraine
basilar migraine
migraine with aura without headache
migraine with acute aura
ophthalmoplegic migraine
retinal migraine
recurrent childhood syndromes,
which may precede or accompany a migraine

Complications of migraine:

migraine status
migraine infarction

A group of childhood recurrent syndromes preceding or accompanying migraine its clinically, the least certain. It includes very heterogeneous disorders - pain in abdomen (abdominal migraine), intermittent vomiting, dizziness, transient hemiplegia with alternating development of paresis or paralysis of the extremities on the right or on the left. These movement disorders occur before the age of one and a half years and are accompanied by other neuropsychiatric disorders. In our opinion, their belonging to migraine is enough questionable. Complications of migraine are migraine status. This is a prolonged attack migraine, continuing, despite treatment, without interruption or with short interruptions more 72 hours, and migraine ischemic cerebral infarction. It manifests itself in one or another characteristic for the aura, symptoms that do not undergo a complete reverse development within 7 days. At this CT scan confirms the presence of ischemic cerebral infarction. According to the recommendations International Headache League, the diagnosis of migraine should be based on the presence of most of the following: unilateral headache, pulsating its nature, moderate or severe intensity of pain, its intensification under the influence of usual mental activity, nausea and (or) vomiting, photo and (or) phonophobia, lack of organic brain diseases according to clinical and paraclinical research methods. To that add the onset of the disease at prepubertal, pubertal or adolescent age, well-being in the pauses between attacks, an indication of a family-hereditary character diseases. The course of migraine in most cases is stable: attacks are repeated with a certain frequency - from 1-2 times a month to several a year, weakening and terminating with the onset of the involutionary period. In other cases, there may be a regredient course - migraine paroxysms, occurring in childhood (prepubertal) age, fade after the end of puberty. In some patients, there is a gradual increase in seizures, causing one or another degree of social maladjustment.

Migraine headache in the vast majority of cases has one-sided character (hemicrania), localized when seizures recur in the same half of the head. Much less often the whole head hurts (holocranium) or there is an alternation of the sides of localization

seizures. The pains are felt mainly in the temple area, have a pulsating, boring character, by the end of the attack they turn into stupid. The intensity of pain varies from moderate to very significant, difficult to carry. The pain increases with exertion. During painful attack, general hyperesthesia, intolerance to bright light, loud sounds, painful and tactile irritations. The sick seek to retire in a darkened room, avoid movement, lie with closed eyes. Often, some relief comes from the contraction. shaking the head with a handkerchief, towel. A headache attack is often accompanied by nausea, vomiting, cold extremities, pallor or redness of the face, less often - retrosternal pain or dyspeptic symptoms. Possible depressed mood, anxiety, fear, severe depression (dysphrenic migraine).

The onset of a migraine attack can be preceded by a number of clinical manifestations: depressed mood, apathy, decreased performance, drowsiness, less often - excitement. Migraines with aura are preceded by various sensory or movement disorders. Aura, as a rule, it is characterized by a significant constancy of the clinical picture in the same sick. Ophthalmic aura - homonymous visual disturbances - zigzags, sparks, spreading to the left or right visual field, combined with a flickering a spiral-like contour moving in the lateral direction, as well as with an absolute or a relative scotoma. Hemiparesthetic aura - paresthesias or a feeling of numbness, nicking locally and spreading slowly to more or less part of one sides of the body. Paralytic aura is characterized by one-sided weakness, aphatic - speech disorders. Ophthalmic-moplegic migraine is characterized by recurrent attacks migraine headache, combined with transient oculomotor disorders.

These symptoms last for an hour, rarely more (migraine with prolonged aura), reversible. There may be a migraine aura without headache (migraine equivalents, acephalgic migraine, “migraine without migraine”).

The development of the aura is associated with a decrease in regional cerebral blood flow in corresponding areas of the cerebral cortex or in the trunk. Retinal migraine is a form a disease in which repeated attacks of mononuclear scotoma or blindness occur, lasting less than an hour and combined with an attack of migraine headache. In these cases it is necessary to exclude ophthalmic diseases and transient ischemic attack, caused by embolism in the retinal artery. Some authors distinguish abdominal migraine, manifested by a combination of headaches with abdominal pain, sometimes accompanied by dyspeptic symptoms, as well as vestibular migraine, in which headache attacks are combined with dizziness, instability; gait can take on an atactic character.

A rare form of migraine is basilar migraine, which usually occurs in puberty in girls and manifested by transient symptoms of discirculation in basin of the main artery: bilateral visual disturbances, tinnitus, dizziness, ataxia, sometimes loss of consciousness up to coma.

It should be borne in mind that in some cases migraine paroxysms can be associated with the presence of organic damage to the nervous system (the so-called symptomatic migraine). Associated forms of migraine are especially suspicious in this respect, in in particular ophthalmoplegic and hemiplegic.

Prolonged attacks of acute pain in the frontal-orbital region in combination with ophthalmoplegia can be a manifestation of Tolosa-Hunt syndrome. This is one of the options so called "painful ophthalmoplegia." It is characterized by prolonged pain, localized mainly inside the orbit for several days or weeks; damage to the oculomotor, abducens, trochlear nerves (or one or two nerves),

the orbital branch of the trigeminal, sometimes the optic nerve is affected; resumption of seizures after spontaneous remission after several months or years; pronounced therapeutic effect when using glucocorticoids. At the heart of the disease is limited granulomatous arteritis internal carotid artery in the cavernous sinus. It should be emphasized that Tolosa-Hunt syndrome
- a rare disease, much more often it is a parasellar tumor. If you suspect the symptomatic nature of migraine paroxysms requires an in-depth examination patient, primarily CT, MRI and magnetic resonance angiography or angiography. With MRI sometimes penetration of inflammatory tissue into the orbit is found.

For a long time, migraine was included in the group of diseases of the "epileptic circle". At present
time has shown that there is no reason for this. Apparently, in some cases we are talking about a combination of two independent diseases in the same patient, and in others - about occurrence under the influence of repeated migraine attacks of ischemic foci with epileptogenic properties and in more rare cases - hemodynamic disorders under by the action of an epileptic discharge.
It is noted that both diseases have a common constitutional predisposing factor.
Treatment of a migraine attack. Drugs of 4 main classes are used: analgesics, sedatives and tranquilizers, antiemetics, ergotamine derivatives and others vasoconstrictors.
Analgesics. Most analgesics contain acetylsalicylic acid, paracetamol (acetamifen), or ibuprofen. Taking more than two tablets every 4 hours is rare gives an additional analgesic effect. Psychological addiction often develops large quantities of analgesics, which is a serious problem. Besides, excessive daily use of analgesics can increase pain, and withdrawal of them accompanied by pain relief.
Antiemetic. Nausea and vomiting are accompanied by a sharp deterioration in the suction the ability of the gastric mucosa, which blocks the action of drugs taken orally. Metoclopramide (cerucal, raglan) is an effective antiemetic drug, accelerating the evacuation of food from the stomach and, in addition, increasing absorption acetylsalicylic acid. Meterazine and others are also used to treat vomiting. phenothiazines.
If you anticipate a severe migraine attack, you should take 10 mg metoclopramide inside. If nausea and vomiting occur unexpectedly, metoclopramide or phenothiazines are given parenterally, if necessary - repeatedly.
Adverse reactions common to all dopamine receptor antagonists, rarely are manifested by acute extrapyramidal disorders (torticollis, muscle spasms of the trunk, face, pharynx, trismus, oculomotor crises), are more common in children and sick young age, as well as with simultaneous treatment with drugs of the phenothiazine series.
It is possible to use cinnarizine, torecan as antiemetics, phenobarbital, diphenin, antelepsin, belloid, atropine, seduxene.
Vasoconstrictors. Ergotamine has been used to treat headaches for almost century. By its action on the smooth muscles of the arteries, it is a vasoconstrictor that stimulates α-
adrenergic receptors and increasing their sensitivity to endogenous norepinephrine. Besides, it has antiserotonin and central emetic effects. Anesthetic effect ergotamine is so specific that the drug can be used in diagnostics migraine
Usually, ergotamine is taken orally in the form of a salt (ergotamine tartrate) in combination with

caffeine, which enhances absorption and is a vasoconstrictor. Some recipes
contain, in addition to caffeine, small amounts of phenobarbital and belladonna alkaloids,
necessary to calm and reduce intestinal motility. Ergotamine should be taken in
the very beginning of the attack. The average starting dose for an adult is 1-2 mg, taking the same
doses can be repeated after 1 hour if pain persists.
It should be remembered that with already developed vomiting and gastric stasis, absorption of the drug
broken. Therefore, the correct tactic in this situation is parenteral administration of 10
mg metoclopramide and the use of rectal suppositories (1-1 / 2 suppositories) with a repetition of the dose
the scheme described above.
In patients who have to take ergotamine frequently due to repeated
migraine attacks, the development of ergotamine intoxication is possible, about which patients should
be warned. The drug should not be used during pregnancy, coronary artery disease, anemia, fever,
liver and kidney diseases, thyrotoxicosis. Ergotism vasospasm (moderate) may
cause peripheral neuropathy; with strong and prolonged vasospasm, ischemia is possible
limbs. Coronary spasm is also likely; however, angina pectoris and heart attack are rare.
A spasm of cerebral arteries with impaired brain function has been described. Perhaps psychological
addiction to the drug. To avoid ergotism, it is not recommended to take more than 4 mg of ergot

mine with one attack or more than 12 mg of the drug per week. For the treatment of vasospasm with ergotism
use sodium nitroprusside and other vasodilators.
Less effective than ergotamine for treating migraine attacks
dihydroergotamine. It is administered intravenously or intramuscularly. The drug is administered intravenously
slowly, the initial dose is 0.5 mg.
Sedatives. It is advisable to use sedatives in cases where
the attack was triggered by emotional stress. They potentiate the action of analgesics.
Usually, in a severe attack, people are unable to perform daily functions, and the attack
it is advisable to interrupt migraines with sleep.
Usually relanium is used by mouth or intravenously; intramuscular injection
reduces the effectiveness of the drug.
Sumatriptan, oral
taking 1-2 tablets of which or a subcutaneous injection relieves the migraine attack in most patients for 10-15 minutes.
Migraine status. Sometimes the migraine attack does not respond to conventional therapy and the pain
relapses several hours after improvement. Such patients need
hospitalized with sleep maintenance for several hours or even days. Such treatment
almost always relieves pain.
Prevention of migraine. It is necessary to explain to the patient in what situations
headache is provoked or intensified. So, sometimes a migraine attack is associated with skipping normal meal times. In 10% of cases, pain is caused by the use of some
foods, especially cheese, red wine and chocolate. The duration of sleep should
be adequate, but not excessive. Some patients sleep after a hard week of work
too much on weekends and wake up with migraines.

The decision to use prophylactic drugs on a daily basis must be made
by the patient himself. Often, upon learning that he does not have a tumor or other serious brain disease,
the patient decides to take the medication only during the attack of the headache. If it is decided to start
preventive treatment, then the patient should know that it is not yet possible to predict which
the medication will suit him and whether a combination of drugs or monotherapy is required.
Taking medications for prophylactic purposes is carried out for several months (up to
of the year).
Beta-blockers. The most commonly used is anaprilin (propranolol, obzidan). it
non-selective β-blocker; other drugs in this group are ineffective. Antimigraine
the action is explained by the effect on pial vessels with β-adrenergic receptors; propranolol
prevents the spasm of these vessels. It penetrates well the blood-brain barrier,
accumulates in the brain tissue and thus can block central serotonin
receptors. The drug also has a clear psychotropic (anxiolytic) effect and
used to stop somatic anxiety attacks. Probably this effect of the drug
important. Propranolol reduces the frequency and severity of migraine attacks, and also reduces
the need for analgesics and other drugs. It is contraindicated in chronic bronchitis,
chronic obstructive pulmonary diseases, asthma, allergic rhinitis during the flowering period,
sinus bradycardia, heart block, circulatory failure.
Usually, treatment begins with the appointment of 10 mg 2 times a day and within 1-2 weeks reach
an average dose of 80-120 mg / day. The drug is taken 3-4 times a day. While increasing the daily
doses up to 180-240 mg are unusual in the prevention of migraine, even higher doses are used in
cardiology, and it remains unclear whether increasing the amount of the drug will help in cases
resistant to conventional treatment regimens.
Side effects are rare. Bradycardia and orthostatic hypotension are possible. A patient
should be aware of the absence of an increase in heart rate during physical
activity and refrain from heavy exertion.
In rare cases, the drug can cause drowsiness, depression, nightmares,
hypnagogic hallucinations, psychosis, impotence.
Amitriptyli. The positive effect of amitriptyline may confirm
common knowledge that tricyclic antidepressants (often in combination with
phenothiazines) are effective in treating various types of pain. Some researchers
believe that the effect of amitriptyliya in chronic headaches and tension pains

mediated through its antidepressant action. The drug, however, is effective in the absence of
signs of depression.
More reason to associate the effect of amitriptyline with its effect on
serotonergic neurotransmitter system involved in regulation
nociceptive functions. Treatment begins with 25 mg of the drug (at night), gradually increasing it
dose (at weekly intervals); amitriptyline is taken once a day; maximum su-
the exact dose is 100-150 mg. The therapeutic effect begins, as a rule, after 2-3 weeks.
Adverse reactions (drowsiness, lethargy) usually occur early and disappear with treatment.
Since this antidepressant has a sedative effect, it is especially helpful in patients with
insomnia or anxiety. The drug has an anticholinergic effect, therefore
can cause urinary retention in men and dry mouth. For some unknown reason, he sometimes
stimulates appetite, which leads to an increase in body weight. Very rarely, an overdose can
cause arrhythmia.
Calcium channel blockers. These drugs are used in the treatment of angina pectoris and

hypertension, recently firmly established and as a means for
prevention of migraine. Verapamil is usually used at a dose of 120-240 mg; effective also
flunarizine, nimodipine, nifedipine. Side effects of verapamil - nausea, constipation, hyperemia
face, hypotension, bradyarrhythmia. Concomitant use of β-blockers increases the risk
the occurrence of the latter complication.
Non-steroidal anti-inflammatory drugs. Preventive action is widely known
vie with migraine 1-2 tablets of acetylsalicylic acid per day. Has a good effect
naproxen in a daily dose of 500-1000 mg (in 2 divided doses). Clonidine helps some people.
(clonidine, gemiton). It is available in a special dosage form of 25 mcg. A drug
begin to take 50 mcg / day (2 tablets), increasing the dose to the maximum - 75 mcg / day (not
earlier than 2 weeks).
Metysergide. It is a derivative of ergotamine with chemical similarities to lysergic acid
properties. It has a good effect as a prophylactic agent in strong
migraine attacks; appoint 2 mg 3-4 times a day.
Adverse reactions can be avoided if treatment is started with a dose of 1 mg and gradually
increase over several weeks.
Apparently, methysergide acts not through the circulation system, but directly
blocks serotonin neurons. Due to the vasoconstrictor action of the drug, rarely
spasm of the coronary or peripheral arteries may occur. With constant intake
metisergid for 4 months or more, the development of subendocardial, pericardial,
pleural and retroperitoneal fibrosis with subsequent dysfunction of the corresponding
organs, in particular the development of ureteral obstruction. In this regard, it is necessary every
4 months interrupt treatment for 1 month. In most cases, the fibrous tissue resolves after
drug withdrawal.
The risk of side effects when taking metisergide does not allow it to be attributed
to first-line drugs.
Non-pharmacological prevention of migraine includes the following measures:
traditional and non-traditional psychotherapy, hypnosis, the use of autogenous training for
muscle relaxation, programmed relaxation, relaxation feedback
muscles of the neck and scalp and expansion of the superficial temporal artery and arteries of the
finger,
acupuncture.

DEMIELINIZING DISEASES OF THE NERVOUS SYSTEM
MULTIPLE SCLEROSIS

Scattered
sclerosis
(multiple
sclerosis,
disseminated
sclerosis)
-
demyelinating disease characterized by multifocal lesions of the nervous system
stem, remitting course, variability of neurological symptoms, predominant
defeat of young people. As a nosological form, Charcot was first identified and described in
1886 According to the prevalence, zones with high, moderate and low frequency are
distinguished
morbidity. The disease is especially common in North America and Northwest
Europe. In our country, multiple sclerosis is more common in the northwest regions
European part. The frequency of multiple sclerosis in different regions of our country fluctuates
in

within 2-70 per 100,000 people. In general, this indicator decreases from north to south and from west to
East; morbidity in cities is higher than in rural areas. In recent years, the disease
detected in those regions where it was not previously observed (Central Asia, Transcaucasia).
Etiology. The most generally accepted theory is the autoimmune
multiple sclerosis. The immediate cause leading to the disease, obviously, is
long-term latent, most likely viral, infection. Basis for Assuming Participation
viruses in the development of the disease are additional results of serological studies
blood and cerebrospinal fluid (increased titers of various antibodies against measles viruses,
herpes simplex, rubella, mumps), detection of viral-like particles with electronic
microscopy of biopsies of nerve tissue taken during stereotaxic surgery from areas
the brain outside the plaques; coincidence of the development of multiple sclerosis with viral infections. Exists
the belief that multiple sclerosis refers to "slow infections" in which the virus invades
into the nervous system, latently exists in it (persists) and manifests its effect only through
long incubation period. The mechanism of persistence is not well understood. Suggest
that there is the formation of defective forms of the virus or integrated forms of the genome of the virus and
host cells. However, the virus was not detected in the blood and cerebrospinal fluid of patients.
multiple sclerosis, antiviral antibodies are detected with approximately the same frequency when
other inflammatory diseases of the nervous system, increased titers of antiviral antibodies
in the blood and cerebrospinal fluid of patients with multiple sclerosis does not always correlate with
clinical response to the ingress of the virus. In addition, in the cerebrospinal fluid of patients
antibodies against certain bacterial antigens are also found. This data
testify against the role of any one virus as an etiological factor of diffuse
sclerosis. It has been hypothesized about the etiological role of various viruses and, possibly,
simultaneous persistence in the nervous system of several viruses. Probably a virus or something else
an infectious or toxic agent may act as a trigger mechanism at the onset
autoimmune process.
It is assumed that the processes of demyelination are mediated by immunopathological
mechanisms: the virus sensitizes the lymphocyte population, which leads to an autoimmune attack on
myelin; it is also possible that some viruses (e.g. measles) carry antigenic determinants,
similar to myelin basic protein, resulting in a cross-reaction with the antigen
the main protein of myelin. It is also possible that the possibility of the persistence of the virus in the nervous
the system is due to the inadequacy of the immune system of the patient's body, and for the implementation
the neurotoxic properties of the putative virus requires the action of a number of additional
factors that reveal this inferiority.
These additional factors include constitutional hereditary
predisposition to disease. In patients with multiple sclerosis more often than in general
population, there are antigens of the histocompatibility system HLA: A3, B7, Dw2. It is noted
a significant increase in the risk of disease when these antigens are found together; exists
the assumption that loci A3, B7 and Dw2 may be markers of a hitherto unknown gene,
determining sensitivity to multiple sclerosis. Geographic
factor. There is evidence that moving from climatic cold zones with high risk
the occurrence of multiple sclerosis in low-risk areas increases the incidence of the disease in these
zones.

Pathogenesis. The hypothesis of the existence of an exogenous damaging agent suggests that in the presence of certain risk factors (geographical features, hereditary predisposition, deficiency of the immune system) this agent, invading cells oligodendroglia of the myelin sheath, causes disintegration of myelin, alters the synthesis

nucleic acids by glial cells. In this case, first of all, they are affected phylogenetically younger structures (optic nerves, pyramidal tract, posterior cords of the spinal cord), most susceptible to both the effects of exogenous factors and fluctuations in the system homeostasis. New protein compounds are formed, against which specific antibodies, including myelinotoxic antibodies. Revealed anti-myelin serum antibodies in patients with multiple sclerosis are classified as class G immunoglobulins (IgG). Formed immune complexes (antigen-antibody complex), on the one hand, support destructive changes in nerve and glial elements, and on the other, their formation leads to the release biologically active substances that violate vascular permeability, including permeability of the blood-brain barrier. In addition, immune complexes are involved in immunoregulatory mechanisms, leading to a violation of the ratio of cell populations. Myelinotoxic and anti-brain antibodies along with the elimination of degraded derivatives myelin can act on intact nerve tissue and support the process demyelination. An unfolding autoallergic reaction leads not only to destruction myelin, but also to vascular-inflammatory and proliferative processes in the focus of demyelination, to
the formation of multiple sclerosis plaques.
Immune restructuring of the whole organism takes place. Secondary changes occur homeostasis, manifested by impaired metabolism of lipids, carbohydrates, proteins and microelements,
platelet aggregation and vascular permeability increase, which leads to perivascular swelling and effusion of fibrin in plaques of multiple sclerosis, the formation of microthrombi in them.
There is also a direct damaging effect of an infectious agent or cytotoxic anti-brain antibodies to immunocompetent cells. In the stage of exacerbation, it is violated differentiation of lymphocytes towards a decrease in the number of T- and an increase in the number of B-cells, change
the ratio of T-lymphocyte subpopulations due to an increase in the killer: suppressor ratio.
Patients with multiple sclerosis develop glucocorticoid insufficiency, which changes immunoreactivity towards increased allergic manifestations and promotes deepening of demyelination processes.
During the immunopathological process, a certain phasing is noted: at first stages of the disease, demyelination is accompanied by excessive antibody production and at the first
plan advocate the phenomenon of autoimmunization. In the later stages as it deepens pathological condition and depletion of all defense mechanisms, a deep restructuring occurs the functioning of the immune system, a perversion of the protective immune responses that manifests itself
persistent immunodeficiency state.
Demyelination of nerve fibers leads to a decrease in speed and a violation of strictly isolated conduction of excitation. There is a chaotic transition of excitation from some nervous structures to others. With this feature and also with great sensitivity demyelinated axons to any changes in homeostasis are associated with diversity neurological symptoms, variability, reversibility with an already existing organic damage to the nervous system.
Pathomorphology. Morphologically multiple sclerosis is characterized by numerous

foci of demyelination in the white matter of the brain and spinal cord. Fresh hearths, so called active plaques, pink, soft, microscopically they show a picture periaxial demyelination of nerve fibers. Axial cylinders at the onset of the disease change insignificantly, only some of them disintegrate. In the area of demyelination, the vessels dilated, stasis, venous stasis, microthrombi are observed, vessels are surrounded by infiltrates from
lymphoid and plasma cells. Proliferation of glial elements is expressed. Gradually cellular elements turn into phagocytes, carrying out products outside the nervous system breakdown of myelin. Growth of microglia, connective tissue occurs, glial scars replacing dead tissue. An old, “inactive” plaque forms. Given "age" plaques, neurohistologists believe that initial changes in the tissue of the nervous system can be detected 2-3 months before the first clinical symptoms appear. Most often affected optic nerves (II), brain stem, cerebellum, spinal cord; possible defeat of others as well cranial and spinal nerves, roots (peripheral forms of multiple sclerosis).

Clinic. Multiple sclerosis is characterized by great variety and variability neurological symptoms. The disease begins mainly at the age of 16-45 years. Known cases of the disease in childhood, as well as in persons of older age groups.

The onset of the disease is often slow, imperceptible, monosymptomatic, but sometimes it occurs acutely
and manifests itself immediately with multiple neurological symptoms. Most often the first

symptoms of the disease are signs of damage to the visual system: fuzziness images, transient blindness, decreased visual acuity, scotomas. The disease can manifest oculomotor disorders (strabismus, diplopia), unstable at the beginning process with pyramidal symptoms (central mono-, hemi- or paraparesis with high tendon and periosteal reflexes, clonuses of feet, pathological reflexes), cerebellar disorders (instability when walking, intentional tremor), disorders sensitivity in the limbs (numbness, paresthesia). Much less often the first signs diseases can be neurosis-like disorders, dysfunction of the pelvic organs (delay urination or urinary incontinence, urgency), vegetative-vascular disorders, paresis facial (VII) and trigeminal (V) nerves, bulbar group nerves. Like a reflection vegetative endocrine disorders in women there is a violation of the menstrual cycle, in men - impotence. A characteristic early, but not obligatory sign of the disease is a decrease in or disappearance of abdominal reflexes.

The progression of the disease leads to the emergence of new ones that do not "fit" into the initial focus of symptoms. In later stages, psychopathological changes in the form of emotional instability, euphoria or depression, as well as a decrease in intelligence of varying degrees.

Depending on the predominant neurological manifestations reflecting mainly the localization of the pathological process, Charcot and Marie in 1868 identified three the main forms of the disease: cerebrospinal, spinal and cerebral. Cerebrospinal the form is characterized by multifocal lesions already in the initial period. Determined symptoms of damage to the pyramidal system, cerebellum, visual, oculomotor, vestibular and other systems. The spinal form is characterized by a lesion of the white matter of the spinal cord on
various levels, more often at the chest. As a rule, the leading clinical syndrome is lower spastic paraparesis, varying degrees of severity, pelvic disorders and disorders sensitivity. Sometimes spinal symptoms are expressed in the form of Brown-Séquard syndrome. Allocate a pseudotabetic form with a predominant lesion of the posterior cords.

The cerebral form includes cerebellar, optical and stem forms. Arise
ataxia, intentional tremor of the upper and lower extremities, adiadochokinesis, dysmetria,

violation of handwriting, chanted speech, horizontal and vertical nystagmus. Far away in cases, intentional trembling becomes sharply expressed and makes it impossible arbitrary precise movements. Pronounced tremor is the basis for the isolation of these cases in the hyperkinetic form of multiple sclerosis.
In the optical form, the leading clinical syndrome is a decrease in visual acuity on one, less often on both eyes, which after a while on their own or under the influence of treatment
passes completely. Ophthalmoscopy reveals signs of retrobulbar neuritis, blanching of the optic nerve head, especially its temporal side, narrowing of the visual fields (first for red and green). Simultaneously with visual impairment, it can other focal neurological symptoms are found.
Certain forms include the stem form of multiple sclerosis, as well as cortical a form characterized by mental disorders and epileptic seizures; hemiplegic form associated with the localization of pathological foci in the area of the radiant crown.
Isolation of forms of multiple sclerosis is conditional, since against the background of the dominant syndrome it is often
you can find other focal symptoms, although they are much less pronounced.
A benign form of multiple sclerosis with relapsing the course of the disease. In this case, remissions and exacerbations are observed, but each exacerbation
ends with a fairly complete restoration of the impaired functions. After the first exacerbation complete recovery is almost always observed, usually by 4-8 weeks from the time of onset first symptoms. In the future, in almost 60% of patients, the disease turns into a progressive form, in 30% from the very beginning the disease is progressive. Quite long remissions are observed in optical form.
In addition to the remitting course, the following options are also distinguished: 1) severe aggravation, increasing immobility and early death; 2) slow, steady progression with periodic exacerbations; 3) many short attacks with a growing tendency by frequency, duration and severity; 4) slow steady progression without exacerbations; 5) acute onset followed by long-term remission; 6) exacerbations, decreasing in frequency and severity, with mild residual symptoms; 7) acute onset, significant exacerbation a year later, without residual symptoms.

The duration of the disease ranges from 2 to 30-40 years. Death comes (for except for cases of acute stem forms) from joining intercurrent diseases: pneumonia, urosepsis, sepsis caused by pressure sores.
Additional research methods. In the blood of patients with multiple sclerosis, the most leukopenia and lymphopenia are often found, lymphocytosis may occur in the acute stage and eosinophilia. There is a change in the rheological and coagulating properties of blood: an increase
platelet aggregation, a tendency to increase the level of fibrinogen and at the same time activation
fibrinolysis. The indicators characterizing the general blood coagulability change little, although according to
as the severity of the disease increases, there is a tendency to hypercoagulability. When researching
cerebrospinal fluid in some patients in the acute stage, there is a slight increase an increase in the amount of protein and moderate pleocytosis (15-20 cells per 1 mm).
According to the results of immunological studies of blood and cerebrospinal fluid, you can identify the predominance of an autoimmune or immunosuppressive component.

In multiple sclerosis, serum and cerebrospinal dyslipidemia is noted.
liquids. In the blood against the background of general hyperlipidemia, an increase in cholesterol fractions and
phospholipids, in the cerebrospinal fluid, mainly phospholipids, especially when
the close location of the foci to the cerebrospinal fluid pathways. Violation of the functional
the state of the adrenal cortex, manifested in a decrease in urinary excretion of C21-
corticosteroids, a decrease in the concentration of cortisol in the blood plasma. Protein and
especially the amino acid composition of the blood. Although the listed changes are not
specific for multiple sclerosis, the complex use of biochemical methods in
dynamics allows you to assess the nature of the course of the disease, the severity and stage of the process, as well as
the effectiveness of the treatment and prognosis.
Basic paraclinical studies allowing about 80%
cases to verify the diagnosis of multiple sclerosis, are specific findings on MRI,
EP and detection of oligoclonal immunoglobulins in cerebrospinal fluid.
Diagnostics and differential diagnostics. Polymorphism of clinical symptoms,
the variety of clinical forms significantly complicates the early diagnosis of multiple sclerosis.
Important criteria for correct diagnosis are: 1) the onset of the disease at a young age;
2) polymorphism of clinical symptoms at all stages of the disease; 3) impermanence,
“Flickering” of symptoms even throughout the day; 4) involvement of nervous structures in the process, which
can proceed in one of the following ways: a) two or more episodes of the disease, divided between
by themselves for a period of one - several months and each lasting at least a day; b) slow
gradual progression of symptoms within 6 months.
The study of VEP, SVP and SSEP makes it possible to determine the presence and localization
pathological process along the investigated conductor. Most common
pathological changes in multiple sclerosis are the absence of one or more components
lengthening of inter-peak latency periods, decrease in amplitudes, increase in absolute
peak periods. With the help of tomography, it is possible to identify subclinical lesions,
localization of the process. At various stages, the differential diagnosis of multiple sclerosis
should be carried out between neurotic disorders, vascular dysfunction,
labyrinthitis, retrobulbar neuritis, tumors of the brain and spinal cord, cerebellum,
disseminated encephalomyelitis, degenerative diseases of the nervous system. Certain
Difficulties may arise in the differential diagnosis between retrobulbar neuritis
infectious and allergic genesis, Leber's disease and the optic form of multiple sclerosis.
It should be borne in mind that visual impairments in the initial stage of multiple sclerosis
are transient, pathological changes occur more often in one eye, may
identify (not always) other neurological symptoms. With perennial (within 15-20
years) of observation of patients who have undergone retrobulbar neuritis, it was found that at least 80%
cases of this disease are associated with multiple sclerosis.
When performing differential diagnosis between multiple sclerosis and tumors
of the brain, primarily a cerebellar tumor and neuroma of the vestibular cochlear nerve (VIII)
it is important to consider the absence of signs of persistent intracranial hypertension, multifocal
lesions and remitting course, characteristic of multiple sclerosis. Decisive
have MRI results.
Spinal forms of multiple sclerosis must be differentiated from tumors
spinal cord. Unlike a tumor, spinal symptoms in multiple sclerosis in
in the initial stages of the disease, paresis is less pronounced (spasticity prevails),

sensitivity disorders and pelvic disorders. In diagnostically difficult cases, help
lumbar puncture (the presence of a block of the subarachnoid space in spinal cord tumors),
contrast studies and MRI.
The hyperkinetic form of multiple sclerosis may resemble the trembling form
hepatocerebral dystrophy. Symptoms may support multiple sclerosis
multifocal lesions of the nervous system. In doubtful cases, it is necessary to investigate
metabolism of copper and amino acids, determine the concentration of ceruloplasmin in the
blood. Changes to these
indicators are typical for hepatocerebral dystrophy. Identifying
the Kaiser-Fleischer corneal ring or its fragments - specific and obligate
a sign of Wilson-Konovalov's disease. Certain difficulties may arise when differentiating
renational diagnosis between the cerebellar form of multiple sclerosis and hereditary
cerebellar ataxia. The analysis of the pedigree of patients is important, the absence of remissions
and other signs of multiple sclerosis. Multiple sclerosis differs from Strumpell's disease
the presence of signs of damage to other parts of the nervous system.
Differential diagnosis between acute
multiple encephalomyelitis and multiple sclerosis. May have a certain value
the severity of cerebral and general infectious symptoms in the development of
encephalomyelitis,
the severity of the appearance of signs of diffuse brain damage, and subsequently regression of
clinical
symptoms. The final judgment in favor of a particular disease can be made at
based on the results of dynamic observation of the patient.
Treatment. Multiple sclerosis treatment consists of measures aimed at
the fight against demyelination of the central nervous system, and symptomatic
therapy. Methods against
demyelination, are divided into the treatment of exacerbations and the treatment of chronically
progressive
diseases. Obviously, this division is artificial, since in many cases a combined
the nature of the course of multiple sclerosis. This division is made even more difficult by the
fact that it is far from
all acute symptoms are caused by the appearance of a new focus of demyelination. Development
transient symptoms can be the result of an increase in body temperature or performance
physical exercise (Uthof symptom). These disorders usually disappear within 24 hours with
normalization of body temperature and a few hours after the termination of the load.
Neurological manifestations caused by the emergence of new areas of demyelination,
usually exist for more than 48 hours. As a rule, the symptoms of disorders reach their maximum
development within 1-2 weeks, and then more or less stabilized for several weeks, or
months. Some therapies are able to reduce the frequency or severity of relapses, but not
affect the progressive nature of the course of the disease. It must be remembered that the main
goal
the following treatments - stopping or slowing demyelination. Since neither
one of the known methods of therapy does not cause remyelination, then the main task is
slowing down or stabilizing the growth of a neurological defect. The doctor should explain
to patients and their relatives the modern level of therapeutic effects, so that their hopes for
treatment results were realistic.
ACTH and glucocorticoids. It is not yet possible to predict the effectiveness of steroids in
each individual patient. It is equally impossible to predict the effect of ACTH on
the production of endogenous corticosteroids. In response to the administration of ACTH, some
patients have
increased activity of corticosteroids; other patients do not have a similar reaction.
Apparently, this can explain the differences in the effect of ACTH treatment, observed clinically.

The mechanisms of action of glucocorticoids in demyelinating diseases remain
poorly studied. Although immunosuppression is the most important factor in steroid action,
a reduction in edema and a change in
electrophysiological properties of tissue. There are two groups of patients: reactive and
unresponsive to treatment. It is believed that the administration of ACTH or corticosteroids,
especially
indicated during acute episodes, with frequent relapses and in patients with optic neuritis
nerve (II). However, even with a chronic course with a constant increase in neurological deficit
a trial course of therapy should be carried out. ACTH can be administered according to the
following scheme: 40 UNITS
intramuscularly 2 times a day for 7 days, 20 units 2 times a day for 4 days, 20 units per day for
within 3 days and 10 units per day for 3 days. There was no evidence-based difference in results
treatment with intramuscular and intravenous administration of ACTH, however, some authors
insist
on intravenous administration during severe exacerbations.
Along with natural ACTH, its synthetic analogue synacthene is widely used.
It is prescribed intramuscularly at a dose of 1 mg / day every day for a week, followed by a
transition

for injection in 1-2 days.
The advantage of prednisolone is the ability to prescribe it by mouth. In most patients
prednisolone can be administered as follows: 80 mg daily for 6-10 days, 60 mg
daily for 5 days, 40 mg daily for 5 days, 30 mg daily for 5 days, 20
mg daily for 5 days, 10 mg daily for 5 days. Thus, the course of therapy
ends within 4-b weeks. The scheme varies widely and may be changed depending on
the course of the disease in each specific patient.
In recent years, with exacerbations of relapsing multiple sclerosis, it has been successfully
very high doses of methylprednisolone are used (10-15 mg / kg per day). It is injected
intravenously into
for 3-5 days, then they switch to taking a tablet preparation (1 mg / kg per day) in
gradually decreasing doses according to the scheme presented above. Many researchers note
greater efficiency of methylprednisolone compared with that of ACTH.
Since ACTH and corticosteroids can intensify tuberculosis, performing
a chest x-ray is strictly necessary before starting treatment. During therapy, it may
additional administration of potassium is required. These drugs should be used with extreme
caution.
used in patients with diabetes mellitus, peptic ulcer disease, arterial hypertension and
kidney disease. It is necessary to limit sodium intake and periodically check the condition
electrolyte balance. Control over body weight, blood pressure, hematocrit is required
and for the presence of latent blood in the stool.
It is necessary to prescribe antacids between meals and at night.
Ulceration of the gastric mucosa is one of the most dangerous complications; emergence
pain in the epigastric region is an indication for gastroscopy. During treatment with ACTH or
corticosteroids may develop mental disorders (confusion, euphoria,
depression, psychosis).
Immunosuppressants. For multiple sclerosis, azathioprine and cyclophosphamide are used.
Azathioprine is prescribed in long courses of 2-3 mg / kg per day. However, recently
the efficacy of azathioprine has been questioned and the more potent appears to be a priority
cyclophosphamide, which can be taken together with ACTH according to the scheme: ACTH (3-
week course, starting

from 25 IU intravenously daily with the transition on the 16th day to 40 IU intramuscularly), cyclophosphamide
(80-100 mg IV daily for 10-14 days). Similar intense immunosuppressive
therapy has some effect in patients with severe multiple sclerosis.
It has been shown that treatment with high doses of cyclophosphamide and ACTH in about 60% of patients
chronic progressive multiple sclerosis manages to stop the progression of the disease. but remission lasts only from 6 months to 2 years, on average 1 year. Given the possibility of significant
side effects from such treatment, it should be noted again that this regimen is only suitable for very seriously ill patients. Cyclophosphamide is prescribed only by doctors knowledgeable about
possible complications.
Total lymph node irradiation is an immunosuppression method that is more specific and having less toxic effect on tissues that are not involved in the immune response. Carry out fractional irradiation of the lymph nodes of the neck, axillary region, mediastinum, periaortic zone and pelvic area. The spleen may be included in the irradiation zone; rest body parts screen. As a result of treatment, severe leukopenia develops, which persists in for 4-5 years. Stabilization and slowing down of disease progression most clearly expressed in those patients who have a decrease in the number of lymphocytes to less than 900 in mm throughout the year after irradiation.
Copolymer-1. It is a polypeptide whose molecule consists of alanine, glutamic acid, lysine and tyrosine. In experiments on rabbits, guinea pigs, rats, chimpanzees and baboons it has been shown that this substance inhibits the development of experimental allergic encephalomyelitis.
It is administered subcutaneously. A. Millga et al. conducted a double-blind, randomized study the effect of copolymer-1 on patients with remitting multiple sclerosis. Noted
statistically significant decrease in the number of exacerbations in the experimental group compared with
a control group receiving a placebo. However, no effect of the drug on
disease progression. A similar trial was conducted in patients with chronic
the progressive course of multiple sclerosis. Significant difference between control and the experimental groups were not identified. The mechanism of action of the substance in multiple sclerosis
(if it exists at all) remains unexplained.
Interferons. There was a positive effect from subarachioid administration of beta interferon, but in clinical practice it is used to a limited extent. Headache and aseptic

meningitis - side effects of therapy. The effect of other immunomodulatory agents (gamma globulin, levamisole, tactivin) is problematic.
Plasmapheresis. The effect of plasmapheresis has been studied, usually in combination with prednisone or
azathioprine. The findings are contradictory. Hyperbaric oxygenation and cyclosporine for control studies did not give a positive result.
Since the hyperpolarization caused by the activity of Na, the K-dependent ATPase, disrupts conduction of impulses along demyelinated fibers, it is assumed that the use of cardiac glycosides will lead to improved high-frequency conduction. During the experiments, the it has been confirmed that they are removing the high-frequency conduction unit on demyelinated fibers. Clinical trials are needed.
Symptomatic therapy. Spasticity and painful flexor spasms are
significant disabling factors that significantly complicate the life of patients.

The most effective drug currently available is baclofen (lyoresal).
It is analogous to the inhibitory neurotransmitter GABA. Typically, 5 mg baclofen 3 is prescribed first
once a day, then the dose is increased by 5 mg every 3 days, bringing it to the maximum - 20 mg 4 times
in a day. Side effects include drowsiness, confusion, arterial
hypotension. Diazepam can also be used to treat spasticity and flexor spasms.
However, in most patients, an effective dose to relieve these spasms leads to
unacceptable sleepiness. Dentrolene sodium blocks skeletal muscle contraction. but
clinical use of this drug is limited by its hepatotoxicity. Positive
the effect is exerted by a new muscle relaxant agent sirdalud. It should be remembered that a number of
in patients, spasticity may have some adaptive function. Therefore, the application
some neuromuscular blockers and similar drugs for treatment
spasticity can cause deterioration of still preserved movements.
Spasticity and flexor spasms can be triggered reflexively. Necessary
prevent peripheral irritation of any kind, the occurrence of pressure sores, infection
urinary tract, since these irritating factors can increase spasticity and flexor
cramping.
Gait disorder (spastic or atactic) is the main cause
disability. Since severe physical inactivity can lead to the development of ankylosis and contractures, physical activity (at least small) should be encouraged. If active movements
impossible, you need to do it several times a day, exercises using the full spectrum
passive movements. Clonazepam, valproate, finlepsin, isoniazid are used to treat tremors
(up to 1200 mg / day).
The use of various orthopedic devices can often help the patient not
able to walk independently due to ataxia and spasticity. For this, use
crutch with support on the forearm and hand, cane with triple support, frame on wheels.
Bladder dysfunction is a severe manifestation of multiple sclerosis. Besides the fact that it is a source of constant discomfort and severe mental trauma, urinary disorders
contribute to the development of pressure ulcers and are one of the causes of death of patients, causing infection
urinary tract.
With a functional break of the spinal cord over the S1 segment, a reflex
neurogenic bladder.
Fluid intake should be regulated and, if possible, the bladder should be emptied
a certain time. Some patients require catheterization. Less often with scattered
sclerosis, an autonomous neurogenic bladder occurs as a result of the rupture of reflex arcs at the level of the cerebral cone. Bladder sensitivity may be impaired with large
the accumulation of residual urine and the impossibility of voluntary urination.
Care for bed-bound or chair-bound patients should be fixed on
prevention of contractures and skin care. Skin care includes the following activities. For
It is recommended to use water and air mattresses to reduce pressure. If applied
oilcloth, it must be dry - even a small amount of moisture can
promote the development of pressure ulcers. Constant pressure zones must be carefully insulated by pillows from contact with the bed surface. The skin is washed and dried
however, excessive dryness must be avoided. It is necessary to avoid minor skin lesions, since any of them can become a source of pressure ulcers. The position of the patient in bed must be changed every 2 hours, and the skin must be examined each time. Redness is an early sign of pressure ulcers development. A local increase in body temperature also

indicates the imminent appearance of an ulcer. When an area of redness appears, it is necessary completely eliminate any pressure on him.
Joint contractures develop due to immobility and spasticity. Most often
the deformity is flexion. Exercise within the limits of possible movements,
carried out at least 4 times a day can prevent the development of contractures. Feet for the night it is necessary to straighten and maintain them in this position using bandages or back splint.
Compression neuropathies are another common complication of prolonged immobilization. The ulnar nerve is most commonly affected by compression in the medial groove. epicondyle of the humerus. Prevention - putting a pillow under the elbow.
Treating "positive" symptoms. Treatment of trigeminal neuralgia with diffuse sclerosis is carried out according to the same rules as the treatment of idiopathic neuralgia. Recommended
exclusion of dental pathology, performing X-ray of the skull with visualization the base of the skull and especially the foramen ovale. In most patients, diphenin is effective. The therapeutic effect is usually achieved at a dose of 300-400 mg / day. If diphenin is ineffective,
Finlepsin is prescribed at a dose of 200-1200 mg / day. During treatment, careful monitoring of function of the liver, kidneys, cardiovascular system and blood picture. Attempts to withdraw the drug
should be undertaken every 3 months. Cancellation of the drug may also be required when the occurrence of side effects. Difenin and finlepsin are usually effective in treating muscle spasms.
Psychosocial correction. Although euphoria is considered the most common mental violation, the development of depression is also not uncommon. In some cases, effective tricyclic antidepressants.
Faintness is reported by 80% of patients with multiple sclerosis. Approximately 60% of patients are weak
interferes with daily activities. In some patients, a feeling of weakness serves manifestation of the disease. This weakness is different from that experienced by healthy people after
excessive physical exertion or lack of sleep. Pathological weakness is defined as a feeling of tiredness or lack of energy, far exceeding the amount of fatigue you can expect when performing daily functions. There is a report on the reduction of weakness with taking amantadine (midantan) at a dose of 100 mg 2 times a day. The drug is well tolerated. Possible
side effect - insomnia. Bilateral damage to the cortical-bulbar tract can result in to pseudobulbar paralysis with violent laughing or crying. It is important to remember that Some patients with this syndrome do not have facial expressions that reflect their true feelings. The patient may laugh involuntarily, despite the fact that he is in a state of depression.
The doctor should explain to family members the possible dissociation between facial expression and true
a ballroom experience. Amitriptyline at a dose of 25-75 mg per day may reduce the severity or completely prevent violent laughing and crying.
People with multiple sclerosis should avoid infection, toxicity, and overwork.
If signs of a general infection appear, bed rest is necessary,
antibacterial drugs and desensitizing agents. Women are advised to avoid pregnancy, although some publications have disputed this categorical prohibition.
Patients are not recommended to change climatic conditions and undergo hyperinsolation. Price forestry to restrict thermal physiotherapy procedures.
Forecast. In the early stages of the disease, the prognosis is very difficult. Especially indicative of the factors

indicating a "benign" course, include: pronounced remission, onset before 40 years, retrobulbar neuritis and sensory disturbances, prolonged remission after the first attack, favorable course in the first 5 years of the disease. Poor prognostic signs: progressive course without remission, onset after 40 years, pyramidal, cerebellar and pelvic disorders. Factors
having no prognostic value: number of exacerbations, MRI pattern, changes in EP, data immunophoresis of cerebrospinal fluid, HLA type, the nature of lymphocyte subpopulations. Some of these criteria are controversial. Thus, the presence of multiple
foci on MRI and (or) oligoclonal immunoglobulins in cerebrospinal fluid,
appears to indicate a poor prognosis.
In general, at least 25% of patients after 15 years retain the ability to independently movement. About 10% of patients cannot walk after 5 years. Average multiple sclerosis shortens life expectancy by 10 years.

ACUTE MULTIPLE ENCEPHALOMYELITIS

Acute disseminated encephalomyelitis (AEM) is an infectious-allergic disease nervous system, caused, apparently, by a neurotropic virus. Perhaps that may there are several types of viruses that can cause this disease. The main ways the introduction of the virus into the body are the upper respiratory tract and the gastrointestinal tract.
The penetration of the virus into the brain and spinal cord occurs hematogenously and perineurally. Big
a change in the body's immunocompetent systems, a decrease in permeability are important blood-brain barrier.
Pathomorphology. Diffuse inflammatory foci of various
size in the brain and spinal cord with pronounced vascular changes and participation microglia. The predominantly white matter is affected, the process has a demyelinating character.
PNS
violated
insignificantly
-
v
nervous
trunks
celebrated
periaxial demyelinating process. Axial cylinders in the brain and spinal cord, the spinal roots and peripheral nerves are much less affected.
Clinic and diagnostics. In most cases, the disease develops acutely, manifesting itself a combination of general infectious, cerebral, shell and focal symptoms. Temperature the body is moderately increased, catarrhal phenomena are often expressed, the headache has a diffuse
character. Focal symptoms are a reflection of diffuse damage to the nervous system. Disorders of cranial innervation are most often found: decreased vision, nystagmus, strabismus, asymmetry of facial innervation, bulbar symptoms. Paresis and paralysis of the limbs more often have a central, less often peripheral or mixed character. Violations are identified coordination tests, ataxia. In rare cases, hyperkinesis is noted: choreoathetosis, myoclonus, tremor. Often, patients complain of pain in the back, chest, limbs; come to light radicular symptoms. As a rule, especially in the acute stage, there are violations of the pelvic functions. All patients have disorders of vegetative-trophic innervation: hyperhidrosis,

pallor of the skin, tachycardia. Signs of damage to various parts of the central nervous system and PNS
are united by the term “encephalomyeloradiculoneuritis”. In some cases it is possible combined damage to the optic nerves (neuritis) and spinal cord (opticomyelitis) in the absence clinical signs of damage to the brain and other parts of the nervous system. Inflammation the optic nerve sometimes leads to blindness.
During puncture, cerebrospinal fluid flows out under moderately high pressure, the number of cells is increased mainly due to lymphocytes and reaches 20-30 cells per 1 mm; the protein concentration changes slightly. Serological tests are negative.
The course of the OREM in typical cases is characterized by the rapid development of general infectious and
neurological symptoms with their subsequent regression and moderately severe consequences, more often in the form of decreased vision, paresis of the extremities, ataxia. Rarely progressive for several days or weeks; sometimes the disease becomes chronic
a character with periodic improvements and exacerbations, usually occurring against the background
infectious diseases. In such cases, the differential
diagnostics with multiple sclerosis; only long-term observation of the course of the disease can solve this difficult question.
Treatment. It is carried out on the basis of the principles outlined in the description of multiple sclerosis.
Schilder's disease
Schilder's leukoencephalitis (diffuse sclerosis) was described in 1912.
severe bilateral demyelination of the cerebral hemispheres and brain stem (with fairly intact axial cylinders), pronounced glial and perivascular inflammatory reaction. The morphological picture and features of the course of the disease make it possible to refer it to
a group of demyelinating diseases.
Leukoencephalitis is equally common in both children and adults. Onset of the disease usually gradual, very rarely stroke. The first major manifestations leukoencephalitis can be behavioral changes, progressive disorders of higher mental functions (visual and auditory gnosis, praxis, speech, intelligence), epileptic seizures, psychotic conditions, pyramidal paresis. The "typical" picture of leukoencephalitis is not
exists. In some cases, the disease begins under the guise of a brain tumor, in others proceeds as a mental illness, in the third it resembles multiple sclerosis. Such the variety of clinical manifestations is due to the diffuse nature of the demyelinating process in the cerebral hemispheres, the size of the foci of demyelination, as well as the degree

severity of perivascular edema.
The most frequent and typical form of the course of leukoencephalitis is pseudotumorous the form. With her, signs of intracranial hypertension (headaches with vomiting, progressive stagnant changes in the optic nerve discs) accompany the growing predominantly one nofocal symptoms (Jacksonian seizures, pyramidal hemiparesis, central homonymous hemianopsia). Often, at the same time, focal pathological activity is detected on the EEG,
slight increased pressure of cerebrospinal fluid, protein-cell dissociation (protein 0.7-3.3 g / l with normal cytosis).
The features of the pseudotumorous form of leukoencephalitis in comparison with tumor the process is the presence of signs of multifocal and bilateral lesions; tendency towards fluctuations in the severity of symptoms and remissions, typical for the demyelinating process;

dissociation between stagnant optic disc changes and lack of enhancement
cerebrospinal fluid pressure and hypertensive changes on the craniogram; in liquid
significant hypergammaglobulinorchia, frequent pathological changes in the Lange reaction; on EEG -
early gross diffuse changes; in remission - a decrease in protein-cell dissociation in
cerebrospinal fluid, focal changes on the EEG. An extremely important diagnostic
the results of CT and especially MRI are important.
Complex therapy should be directed, as in the treatment of multiple sclerosis,
primarily to suppress autoimmune reactions and include adrenal cortex hormones.
Among the symptomatic agents, the most important are anticonvulsants and
myotonolytic.

Guillain-Barré acute inflammatory demyelinating polyradiculoneuropathy (OVDP)

In 1916, Guillain, Barre and Strohl described acute peripheral paralysis with protein
cellular dissociation in cerebrospinal fluid and a favorable prognosis. Described by them
the clinical picture practically did not differ from the "acute ascending paralysis" described
Landry, back in 1895, the OVDP meets with a frequency of 1.7 wa 100,000 population, evenly in different
regions, at any age, men are more likely than women. Currently, the OVDP is
the most common cause of the development of acute peripheral paralysis along with acute
polymyositis and myasthenia gravis. The etiology of the disease is unknown, sometimes it is associated with
commonplace infections. Detection of antibodies to PNS myelin in the serum of patients, as well as
development of segmental demyelination after administration of serum into the sciatic nerve of a rat
(experimental allergic neuritis) strongly suggests that the underlying
the pathogenesis of the disease is immunological disorders. The main site of the immune
the conflict is the subperineural space. Against the background of immune disorders, edema occurs,
inflammatory cell infiltration and diffuse primary segmental demyelination in
primarily in the anterior roots and proximal spinal nerves, plexuses,
nerves of the extremities and vegetative nodes.
In about half of patients, 1-3 weeks before the onset of the first neurological symptoms
there are diseases of the upper respiratory tract, transient acute intestinal disorders,
angina. At the beginning of the disease, 50% have paresthesia in the feet, myalgia in the legs, 20% -
sensorimotor disorders in the distal extremities, in 20% - only weakness, often -
cranial neuropathy (bilateral paresmic muscles, bulbar and oculomotor
violations).
Flaccid paralysis is the leading symptom. Muscles are usually diffusely affected and
symmetrical. Muscle weakness often spreads in an ascending direction, capturing
muscles of the legs and pelvic girdle, trunk, neck, respiratory muscles. Muscle weakness
usually progresses within 2-3 weeks (average 7-15 days), but sometimes tetraplegia can
develop within a few hours or days. In the early days of the disease, myalgias are often observed,
probably due to the myositis process, since they are accompanied by an increase
muscle aminotransferases. Myalgias usually subside without treatment after a week. At
the progression of the disease may develop respiratory failure and bulbar
violations, in connection with which it is necessary to transfer patients to mechanical ventilation and tube feeding. Translation
patients on mechanical ventilation is carried out with a vital capacity of the lungs less than 15 ml / kg. Defeat

phrenic nerve leads to limitation of the excursion of the diaphragm and paradoxical type abdominal breathing (retraction of the anterior abdominal wall during inhalation). Many patients in acute
phase of the disease and with severe movement disorders, there are autonomic disorders:

orthostatic hypotension, tachycardia, paroxysmal arrhythmia with ECG changes (depression *ST* segment , inversion of the H-wave, lengthening of the Q-T interval). Involvement of the autonomic apparatus of the heart
in rare cases, it can lead to its sudden stop. Dysfunction of the pelvic organs is possible in the acute phase of the disease. Vegetative disorders sometimes persist in the long-term period. Sharp
pandisautonomy, apparently, is a special variant of OVDP, in which
vegetative fibers.
All patients develop muscle hypotension. Muscle atrophy in the acute phase is not observed, however, in a number of patients with tetraparesis or tetraplegia in the recovery period, there is weight loss of the muscles of the proximal or distal extremities. Areflexia or
hyporeflexia is not associated with the severity of paralysis or muscle atrophy, but depends on demyelination and
block of conduction along the affected roots and nerves. Sensitive disorders are less pronounced heavier than motor, and are represented by paresthesia, pain, hypalgesia, hyperesthesia in distal extremities. In case of involvement of proprioceptive sensitivity
there are sensitive ataxia and stereoanesthesia. Symptoms of tension on the nerve trunks (Lasegue,
Neri) remain positive for a long time. Persistent conduction disorders of the senses
the diagnosis of AFDP is excluded. Sensorimotor disorders in AIDP are based on segmental demyelination. Involvement of cranial nerves is observed in half of patients (facial, bulbar and in 10% oculomotor). Sometimes with a large increase in content protein in the cerebrospinal fluid, there is a congestive papilla of the optic nerve. Protein-cell dissociation in the cerebrospinal fluid has an exceptional
diagnostic value in ARDP, however, in the 1st week of illness, the protein may be normal. There is no correlation between the protein content in the liquid and the clinical
picture. Detection
in cerebrospinal fluid more than 50 cells in 1 mm should always raise doubts about OVDP. The role of electrodiagnostics is very revolving. In the phase of progression of movement disorders
lengthening of the distal (motor) latency is detected, a decrease in the speed of conduction along motor and sensory fibers, lengthening of the F-wave, which is associated with segmental demyelination and block of conduction. In the first days of illness, electrophysiological indicators
can be normal (!). There are 3 main reasons for differential diagnosis.
diseases: Bannwart's syndrome, diphtheria and porphyria neuropathy.
Corticosteroid therapy has long been considered the mainstay of treatment. but
in the last decade, careful controlled studies have shown that
steroid therapy does not change the course of the disease, and may even contribute to the relapse of the disease. V
treatment with corticosteroids is not recommended at this time!
At the same time, a significant efficiency of plasmapheresis has been shown. In one procedure, which is usually carried out every other day, exchange 1.5-2 liters of plasma. The replacement fluid consists of
fresh frozen plasma, 4% albumin solution and plasma-substituting solutions. Also proved the effectiveness of intravenous administration of immunoglobulin.

For the prevention of thrombus formation in the immobilized limbs, it is recommended the appointment of heparin 5000 IU subcutaneously 2 times a day.
On average, OVDP treatment in a hospital setting is carried out for 2 months, the subsequent restoration of motor functions is observed within 1-2 years. Forecast in 1/4 of patients excellent, however, 2-5 patients die, 10-20% remain with motor impairments of varying degrees. Recovery can take 2 years.
Timely diagnosis of AIDP and rational therapy (plasmapheresis, mechanical ventilation, parenteral nutrition, psychological support, physiotherapy) significantly improve the prognosis.