MINISTRY OF HEALTH OF UKRAINE

ODESA NATIONAL MEDICAL UNIVERSITY

Faculty of Medicine №2

Department of Neurology and Neurosurgery

APPROVED BY
Vice-Rector for Scientific and Educational Work
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TEACHING MATERIAL
FOR LECTURES ON THE ACADEMIC SUBJECT

Faculty, Course: Medical, 4th year
Academic Discipline: **Neurology**

Approved by:
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LECTURES

Lecture No. 1

Topic: Introduction to neurology. Principles of the structure and functions of the nervous system. Symptoms of central and peripheral paresis. Syndromes of damage to the motor path at different levels. Automated involuntary movements. Coordination of movements. Extrapyramidal system and syndromes of its damage.

Actuality of theme. Justification of the topic: doctors of various specialties encounter various disorders of the nervous system, and therefore, knowledge of the principles of diagnosis and the ability to diagnose dysfunction of the nervous system helps the doctor to prescribe adequate treatment to the patient in a timely manner in order to improve his adaptation capabilities to the conditions of the external environment.

Entire lectures:

Educational:

1. To acquaint students with the regulatory function of the nervous system (1st degree of abstraction).
2. Based on the lecture material, the learner must learn anatomical and physiological features and symptoms of nervous system damage, anatomical and physiological features and syndromes of nervous system damage, basic methods of nervous system research (2nd degree of abstraction).
3. Based on the above, provide students with the opportunity to master the skills of diagnosing movement disorders and vegetative paroxysmal states. (3rd degree of abstraction).

Educational:

1. Aimed at the development of a professionally significant personality substructure;
2. Education of professional thinking in students
3. Ensuring that students learn the leading importance of domestic clinical, scientific and pedagogical schools, and especially Odesa, in the development of problems of domestic and world neurology
4. Acquisition of deontology and medical ethics skills by students.

Plan and organizational structure of the lecture

No	The main stages of the lecture and their content	Goals in levels of abstraction	Type of lecture, methods and means of activation of acquirers, equipment	Time allocation
I 1. 2.	Preparatory stage Setting an educational goal Providing positive motivation	 I I	According to the publication "Methodical recommendations for planning, preparation and analysis of a lecture"	5% (5 min)
II 3.	The main stage Presentation of lecture material according to the plan: 1. Actuality of theme 2. Definition 3. Classification	 II II II	Slide presentation of lecture material	85% (75 min)

	4. Etiology and main links of pathogenesis 5. Clinical picture 6. Diagnostics 7. Leading syndromes and differential diagnosis 8. Evaluation of the severity of the course 9. Treatment 10. Prevention	II II II II II II II	Extracts from the medical histories of patients. Excerpts from clinical protocols of the Ministry of Health of Ukraine on providing medical care to patients.	
III 4. 5. 6.	The final stage Summary of the lecture, general conclusions Answers to possible questions Tasks for independent preparation	 III III III	List of references, questions, assignments	10% (10 min)

Content of the lecture material.

The nervous system is of ectodermal origin, develops from the outer germ layer one cell layer thick as a result of the formation and division of the medullary tube. The following stages can be schematically distinguished in the evolution of the nervous system.

1. Reticular, diffuse, or asynaptic nervous system: in freshwater hydra. It has the form of a network and is fairly evenly distributed throughout the body, thickening around the oral appendages. Cells differ from the nerve cells of higher animals: they are small in size, do not have a nucleus and chromatophilic substance characteristic of a nerve cell. This nervous system conducts excitation diffusion in all directions, providing global reflex reactions. At further stages of development of multicellular animals, it loses its importance as a single form of the nervous system, but in the human body it is preserved in the form of Meissner's and Auerbach's plexuses of the alimentary canal.

2. The ganglion nervous system (in worms) is synaptic, conducts excitation in one direction and provides differentiated adaptive reactions. Special organs of movement and receptor organs develop, groups of nerve cells appear in the network, the bodies of which contain **a chromatophilic** substance. It has the property of being decomposed during the excitation of cells and restored in a state of rest. Cells with a chromatophilic substance are located in groups or nodes - ganglia, which is why the cells themselves are called **ganglion cells.** So, at the second stage of development, the nervous system **changed from reticular** to **ganglion-reticular.** In humans, this type of structure of the nervous system has been preserved in the form of paravertebral trunks and peripheral nodes (ganglia), which are characterized by autonomic functions.

3. Skeletal motor apparatuses with striated muscles arose from the **tubular nervous system (in vertebrates).** Initially, the segmental apparatus of the spinal cord is formed from the caudal, undifferentiated part of the medullary tube, and the main parts of the brain are formed from the front part of the brain tube as a result of cephalization (from the Greek kephale - head). In human ontogenesis, they develop sequentially according to the scheme: first, three primary brain bubbles are formed: front (prosencephalon), middle (mesencephalon) and diamond-shaped, or rear (rhomencephalon). In the future, the terminal (telencephalon) and intermediate (diencephalon) bladders are formed from the anterior cerebral bladder. The rhomboid brain bubble is also fragmented into two: posterior (metencephalon) and oblong (myelencephalon). Thus, herds; the stage of three bubbles is replaced by the stage of five bubbles, from which different departments of the central nervous system are formed: from telencephalon - - large hemispheres of the brain, diencephalon - intermediate brain, mesencephalon - - midbrain, metencephalon - bridge of the brain and cerebellum, myelencephalon - medulla oblongata.

The further development of the central nervous system led to the emergence of special functional relationships between the brain and spinal cord, built on the principle of subordination. The essence of the principle of subordination is that the evolutionarily younger brain structures not only regulate the functions of older, lower nervous structures, but also subordinate them to themselves by inhibition or excitation. Moreover, subordination exists not only between the brain and the spinal cord, it is observed between the cortex and the subcortex, between the subcortex and the brain stem, and to some extent even between the cervical and lumbar thickenings of the spinal cord.

Among the morphological stages of the development of nervous systems, centralization of the nervous system, cephalization, corticalization in chordates, and the appearance of symmetrical hemispheres in higher vertebrates should be mentioned. Functionally, these processes are related to the principle of subordination and growing specialization of centers and cortical structures.

The nervous system consists of nerve cells - neurons, which develop from neuroblasts. A neuron has a body and a large number of processes: axon and dendrites. An axon, or neurite, is a long process that conducts a nerve impulse in the direction from the cell body and ends with a terminal branch (it is one). Dendrites are a large number of short tree-like branched processes. They transmit the nerve impulse towards the cell body. The neuron body consists of cytoplasm and a nucleus with one or more nucleoli. Special components of nerve cells are chromatophilic substance and neurofibrils. Chromatophilic substance has the appearance of lumps and grains of different sizes, is contained in the body and dendrites of neurons and is never found in the axons and initial segments of the latter. It is an indicator of the functional state of the neuron: it disappears in case of exhaustion of the nerve cell and is restored during the period of rest. Neurofibrils have the form of thin threads located in the cell body and its processes. The cytoplasm of a nerve cell also contains a lamellar complex (reticular Golgi apparatus), mitochondria and other organelles. Clusters of nerve cell bodies form nerve centers, or the so-called gray matter.

Nerve fibers are the processes of neurons. Within the central nervous system, they form the white matter of the brain. Nerve fibers consist of an axial cylinder, which is an outgrowth of a neuron, and a shell formed by oligodendroglia cells (neurolemocytes, Schwann cells). Depending on the structure of the sheath, nerve fibers are divided into myelinated and unmyelinated. Myelinated nerve fibers are part of the brain and spinal cord, as well as peripheral nerves. They consist of an axial cylinder, a myelin sheath, a neurolemma (Schwann sheath) and a basement membrane. The membrane of the axon serves to conduct an electrical impulse and in the area of axonal endings releases a mediator, and the membrane of dendrites reacts to the mediator. In addition, it provides recognition of other cells in the process of embryonic development. Therefore, each cell searches for its designated place in the network of neurons. Myelin nerve fibers have areas of narrowing nodes (nodal intercepts of Ranvier). Unmyelinated nerve fibers are typical of the autonomic (autonomic) nervous system. They have a much simpler structure: they consist of an axial cylinder, a neurolemma and a basement membrane. The speed of nerve impulse transmission by myelinated nerve fibers is much higher (up to 40-60 m/s) than unmyelinated (1-2 m/s).

The main functions of a neuron are the perception and processing of information, its transmission to other cells. Neurons also perform a trophic function, influencing the metabolism in axons and dendrites. The following types of neurons are distinguished: *afferent, or sensitive,* which perceive irritation and transform it into a nerve impulse; *associative, intermediate,* or *interneurons* that transmit a nerve impulse between neurons; *efferent,* or *motor,* which ensure the transmission of a nerve impulse to the working structure. This classification of neurons is based on the position of the nerve cell in the reflex arc. Nervous excitation is transmitted through it in only one direction. This rule was called physiological, or dynamic, polarization of neurons. As for an isolated neuron, it is capable of conducting an impulse in any direction. Neurons of the cerebral cortex are divided into *pyramidal* and *non-pyramidal neurons based on morphological characteristics.*

Nerve cells contact each other through *synapses* , specialized structures where the nerve impulse passes from neuron to neuron. Synapses are mostly formed between axons of one cell and dendrites of another. There are also other types of synaptic contacts: axosomatic, axo-axonal, dendrodendritic. Therefore, any part of a neuron can form a synapse with different parts of another neuron. A typical neuron may have 1,000 to 10,000 synapses and receive information from 1,000 other neurons. The synapse consists of two parts: presynaptic and postsynaptic, between which there is a synaptic cleft. The presynaptic part is formed by the terminal branch of the axon of the nerve cell that transmits the impulse. It mostly looks like a small button and is covered with a presynaptic membrane. In the presynaptic endings there are vesicles, or vesicles, containing so-called mediators. Mediators, or neurotransmitters, are various biologically active substances. In particular, the mediator of cholinergic synapses is acetylcholine, and adrenergic synapses are norepinephrine and adrenaline. The postsynaptic membrane contains a special protein — a mediator receptor. Mechanisms of neuromodulation affect the release of a neurotransmitter. This function is performed by neuropeptides and neurohormones. The synapse ensures the one-sidedness of the conduction of the nerve impulse. According to their functional features, two types of synapses are distinguished: excitatory synapses, which contribute to the generation of impulses, and inhibitory synapses, which are able to cancel the action of signals. Nerve cells are characterized by a low level of excitability.

In addition to neurons that form the nerve cell parenchyma, an important class of cells of the central nervous system are glial cells (astrocytes, oligodendrocytes, and microgliocytes), the number of which is 10-15 times greater than the number of neurons and which form neuroglia. its functions: supporting, delimiting, trophic, secretory, protective. Glial cells are involved in higher nervous (mental) activity. With their participation, the synthesis of mediators of the central nervous system is carried out. Neuroglia also play an important role in synaptic transmission. It provides structural and metabolic support for the network of neurons. Therefore, there are various morpho-functional connections between neurons and glial cells.

Anatomical and topographic divisions of the nervous system

The nervous system unites a number of departments and structures that collectively ensure the connection of the organism with the environment, regulation of life processes, coordination and integration of the activities of all organs and systems. The nervous system is a hierarchy of levels, different in their structure, phylo- and ontogenetic origin. The idea of levels of the nervous system was scientifically proven based on Darwin's evolutionary theory. In neurology, this idea is rightly associated with the name of the Scottish neurologist Jackson. There are four anatomical and topographic divisions of the nervous system.

1. The receptor-effector department originates in the receptors of each of the analyzers, which actually determine the nature of the irritation, transform it into a nerve impulse, without distorting the information. The receptor department is the first level of analytical and synthetic activity of the nervous system, on the basis of which reactions and responses are formed. There are two types of effectors — motor and secretory.

2. The segmental division of the spinal cord and brain stem includes the anterior and posterior horns of the spinal cord with the corresponding anterior and posterior roots and their counterparts in the trunk region - the nuclei of the cranial nerves, as well as their roots. White matter is located along the spinal cord and trunk - ascending and descending conductive paths that connect the segments of the spinal cord with each other or with the corresponding nuclei of the brain. Processes of interstitial cells end with synapses within the gray matter of the spinal cord. At the level of the segmental division of the spinal cord, the brain stem, reflex arcs of unconditional reflexes are closed. Therefore, this level is also called the reflex department. The segmental-reflex department is a point of recoding of information perceived by receptors. Through the segmental-reflex level of the spinal cord and trunk formations, the cerebral cortex and subcortical structures are connected with the environment.

3. The subcortical integrative department includes subcortical (basal) nuclei: caudate nucleus, shell, globus pallidum, thalamus. It contains afferent and efferent channels of

communication connecting individual nuclei with each other and with the corresponding areas of the cerebral cortex. The subcortical department is the second level of information analysis and synthesis.

4. The cortical part of the brain is the third level of analysis and synthesis. Signals of varying degrees of complexity are sent to the cortex. This is where information is unchained, its higher analysis and synthesis of its impulses takes place. The highest form of apolitical and synthetic activity of the human brain ensures thinking and consciousness.

Voluntary movements and their pathology

Active movements of a person, which are formed in the process of life experience and are continuously controlled by all the activities of the brain, are called voluntary or conscious. A motor act is a complex reflex that is carried out with the participation of different levels of the nervous system. The controlling role in the formation of movements belongs mainly to afferent signals. Before the planned movement occurs, the sensory systems of the cerebral cortex receive information about the initial state of the articular-muscular apparatus, about its readiness to perform the movement, through sensitive conductive paths. Afferent impulses from proprioceptors constantly inform about the need to perform this or that voluntary movement, about the progress of its execution, carry out timely correction of the force, duration, sequence of muscle contractions, inform about the feasibility of suspending the movement or about its completion. The sensitive and motor zones of the cortex form a single sensorimotor system that regulates the work of the lower nerve centers and regulates human motor activity. The action of the proprioceptive analyzer is complemented by the functioning of the vestibular, visual, and auditory analyzers. An important role in the creation of precise, coordinated movements is played by numerous structures of the extrapyramidal system, the cerebellum, and the intermediate brain.

The motor centers of the cerebral cortex are located mainly in the precentral gyrus. In the anterior parts of the frontal lobes of the cerebral hemispheres (tertiary projection field), all information signals are integrated, and a model of a motor act is formed. Voluntary movements are also carried out with the participation of the premotor area (secondary projective movement fields). giant pyramidal motor cells described in 1874 are located in the fifth layer of the cerebral cortex. Kyiv neurohistologist V.O. Betsom Axons of Betz cells form a pyramidal path, which, together with the motor fibers of the peripheral nervous system and the muscular apparatus, ensure the execution of a given motor program. Irritation of motoneuron bodies in the precentral gyrus causes clonic or tonic seizures, called Jacksonian epilepsy after the English scientist Jackson John Hughlings (1835-1911), who described them. Typical for epilepsy of the Jacksonian type is the beginning of a seizure from a limited group of muscles while the patient is clearly conscious. Sometimes convulsions become generalized and turn into a general attack, when the patient loses consciousness.

Irritation of certain areas in front of the central gyrus causes focal spasms in the corresponding groups of muscles on the opposite side, since each of the departments of this area of the cortex is associated with a certain area of the body. In the central lobe and in the upper quarter of the precentral gyrus there are centers for the innervation of the muscles of the lower limb (from above - the foot, below - the leg and thigh), in the middle two quarters of the gyrus there are centers for the innervation of the muscles of the upper limb (from above the shoulder, below the forearm - hands), the muscles of the face, pharynx, larynx, and tongue are projected in the lower quarter of the gyrus. The projection of head muscles (including eye muscles) is localized in the back part of the middle frontal gyrus, and trunk muscles - in the back part of the upper frontal gyrus (somatotopic representation of motor functions). The area of each of the motor areas does not depend on the mass of the muscles, but on the complexity and accuracy of the functions performed by them. Muscles that perform the most complex differentiated movements (hands, fingers, lips, tongue) have the largest representation in the cortex. In order to carry out voluntary movement, the impulses generated in the cortex of the cerebrum must reach the corresponding muscles. This is achieved through a pathway that clinicians call **the corticomuscular pathway** .

This pathway consists of two neurons — *central and peripheral.* The central neuron is the body of giant pyramidal cells (Betz) with their axons, the peripheral neuron is the body and axon of cells of the anterior horns of the spinal cord, motor nuclei and roots of cranial nerves. Along the axons of the pyramidal cells, impulses go from the cerebral cortex to the motoneurons of the anterior horns of the spinal cord and the motor nuclei of the cranial nerves, forming a pyramidal pathway. Therefore, the pyramidal pathway consists of two bundles of nerve fibers — corticospinal (from the cortex to the spinal cord) and corticonuclear (from the cortex to the motor nuclei of the cranial nerves). The pyramidal tracts are located under the cortex in the form of a radiant crown, and then, gradually approaching each other, pass between the subcortical nuclei, forming the knee and the front two-thirds of the posterior leg of the internal capsule. Further, the fibers of the pyramidal pathway go through the base of the legs of the pons and medulla oblongata, on the front surface of which they form two noticeable ridges - pyramids. At the border of the medulla oblongata with the spinal cord, the corticospinal fibers partially cross. A larger, crossed, part of the fibers passes into the lateral cords of the spinal cord (lateral crossed corticospinal (pyramidal) pathway), a smaller, uncrossed, part passes into the anterior cords of the spinal cord (anterior uncrossed corticospinal (pyramidal) pathway). Bundles of fibers of the cortical-spinal tract, gradually thinning, end on the alpha motoneurons of the anterior horns of the spinal cord of all segments, to which they transmit motor impulses. The fibers of the anterior cortical-spinal tract pass to the opposite side at the level of the segments in the anterior white ganglion of the spinal cord. It is assumed that in the cervical and thoracic segments of the spinal cord, some of these fibers connect with the cells of the anterior horn of their side, thanks to which the muscles of the neck and trunk receive cortical innervation from both sides. The axons of peripheral neurons first exit as part of the anterior roots of the spinal cord, then as part of the nerve plexuses and peripheral nerves, they go to the skeletal muscles.

The fibers of the cortical-nuclear pathway, which end on the motor nuclei of the cranial nerves of the brainstem, transmit impulses to the motor cells of these nuclei, their axons form the motor fibers of the cranial nerves that innervate the corresponding muscles. The fibers of the cortical-nuclear pathway also make a partial crossing over the nuclei of the cranial nerves (see Fig. 2). Only the fibers that go to the lower part of the nucleus of the facial nerve and to the nucleus of the hyoid nerve carry out a complete supranuclear junction. Thanks to the incomplete crossing of the pyramidal pathway, bilateral cortical innervation of the muscles is ensured. But it is not present in all muscle groups in the same way. It is most pronounced in the muscles innervated by cranial nerves, in the muscles of the neck, trunk, and perineum, and least in the mimic muscles of the lower part of the face, the muscles of the tongue and limbs. It is these muscles that have predominantly unilateral cortical motor innervation from the opposite hemisphere of the brain. Therefore, in the case of a unilateral lesion of the pyramidal pathway, movements from the opposite side are lost precisely in the muscles of the limbs, tongue and lower half of the face. The function of most muscles innervated by cranial nerves, neck, trunk and perineum muscles is not disturbed. Thus, the first neurons of the motor path connect the motor part of the cortex with the segmental apparatus of the spinal cord and brain stem. The entire complex of cells, with the help of which this connection is carried out, is called *the pyramidal system.* Functions of the pyramidal system: participates in the execution of voluntary movements, sending impulses to the trunk and spinal segmental apparatuses; regulates the functions of the segmental apparatuses, activates the large alpha-motoneurons of the anterior horns of the spinal cord and inhibits the activity of the reflex arcs; limits the spread of excitation impulses along the neurons of the spinal cord; inhibits reflex automatisms of the subcortical, trunk and spinal levels.

If the corticomuscular motor pathway is affected at any level, the corresponding muscles do not receive innervation from the cortex, voluntary movements in the muscles become impossible, the muscle stops contracting, and its *paralysis (plegia) occurs.* In the case of partial damage to the motor path, paresis is observed - incomplete loss of voluntary movements, limitations of their volume, caused by a violation of muscle innervation. Since the giant pyramidal cells with their long axons forming the pyramidal pathway are central neurons, the paralysis or

paresis of muscles resulting from their damage is called *central.* Motoneurons of the anterior horns of the spinal cord and motor nuclei of the cranial nerves are called peripheral, therefore the paralysis that occurs in case of their damage is called *peripheral.* So, the type of paralysis is determined by the name of the affected neuron.

Features of central paralysis (paresis):

1. Damage to central neurons covers whole bundles of fibers of the pyramidal pathway. Therefore, central paralysis usually occurs not of individual muscles, but of entire groups of them

2. Central paralysis (paresis) is called spastic, as it is accompanied by an increase in muscle tone. As a result of damage to the central neurons, control of the pyramidal system over the tonic activity of the segmental apparatus and the function of the spinal reflex arcs is removed. Mainly, the muscle tone increases in the extensors of the lower limb and the flexors of the upper limb. This contributes to the appearance of the characteristic Wernicke-Mann posture (especially due to damage to the internal capsule) - the paralyzed upper limb is brought to the trunk, pronated and bent at the elbow joint, the hand and fingers are also bent, and the lower limb is extended at the hip and knee joints, as if stretched and " elongated". When walking, the patient describes a semicircle with this leg so as not to touch the floor with the toe of the outstretched leg.

3. Tendon and periosteal reflexes increase. This is explained by the fact that the reflex arcs are inhibited at the level of the segmental apparatus. Under such conditions due to significant tendon hyperreflexia clonus of the foot and hand occurs.

4. Abdominal skin and plantar reflexes are suppressed, the occurrence of which is connected with the formation of pyramidal pathways.

Pathological reflexes appear: the appearance of plantar flexor or extensor pathological signs indicates damage to corticospinal fibers, and reflexes of oral automatism indicate bilateral damage to cortico-nuclear pathways.

As a result of disinhibition of the segmental-reflex apparatus, spinal automatisms (protective movements) increase, pathological synkinesis appears.

Central muscle paralysis is often accompanied by urination and defecation disorders. The centers of these functions are located in the gray matter of the spinal cord at the level of the lateral horns of segments 52-54. Conscious control of urination is ensured thanks to the connections of these centers with the cortex of the cerebral hemispheres. Kirk's innervation of the pelvic organs is carried out by the fibers of the pyramidal pathways, their bilateral damage is accompanied by disorders of these functions. There is periodic urinary incontinence (periodic reflex emptying of the bladder without conscious control in case of stretching it with urine), sometimes urinary retention, imperative urges to urinate are observed.

Peculiarities of peripheral paralysis (paresis).

1. Damage to a peripheral neuron in any part of it leads not only to the interruption of the motor cortical-muscular pathway, but also to the rupture of the segmental reflex arc in its efferent link. Therefore, in the presence of peripheral paralysis, both voluntary and reflex movements become impossible. There is an areflexia or hyporeflexia of the reflexes that close at the level of the lesion.

2. Muscle atony or hypotonia is observed, because as a result of the rupture of the spinal reflex arc, the muscle fibers do not receive tonic impulses, therefore the peripheral paralysis got the name flaccid.

3. 2-3 weeks after the onset of damage to peripheral neurons, atrophy is observed in the corresponding muscles, because trophic influences do not reach them due to the rupture of the reflex arc.

4. The three characteristic signs of peripheral paralysis, areflexia, atony, and muscle atrophy may be accompanied by other symptoms. In the case of chronic progressive processes in the anterior horns of the spinal cord or in the motor nuclei of the cranial nerves, there is irritation of the bodies of peripheral motoneurons and fibrillar muscle twitches are observed.

5. The spread of peripheral paralysis is mostly limited, because mainly separate areas of the front horns, separate front roots or peripheral nerves are affected.

6. Peripheral paralysis is characterized by the occurrence of a reaction of regeneration or degeneration, which is manifested by changes in the reactions to electric current of the affected nerves and muscles innervated by them.

Peripheral paralysis occurs in the presence of damage to the anterior horns of the spinal cord, motor nuclei of cranial nerves and their roots, anterior roots of the spinal cord, cervical, brachial and lumbosacral plexuses, peripheral nerves.

Extrapyramidal system and syndromes of its damage

Conscious contraction of one or another muscle is provided by the pyramidal system. But, performing this or that arbitrary movement, a person does not think about which muscles need to be shortened at the right moment. Habitual movements that require the coordinated action of many muscles are performed automatically, imperceptibly, and the change of one muscle contraction by another is involuntary. Automated movements are the most advanced. They are energy-saving, optimal in terms of volume, time, energy consumption, etc. Consistency, duration of muscle contractions, and perfection of movements are ensured by the extrapyramidal system, which, compared to the pyramidal system, is a phylogenetically older motor-tonic apparatus. The extrapyramidal system creates prerequisites for performing motor reactions, the background against which fast, accurate, differentiated movements are performed, prepares muscles for action, ensures the appropriate distribution of tone between different muscle groups, necessary for performing movements of any complexity. The extrapyramidal system takes a direct part in creating a certain posture of a person, motor manifestations of emotions, creates an individual expression of human movements. It ensures the performance of automated, memorized motor stereotyped acts, as well as unconditional reflex protective movements.

The extrapyramidal system includes numerous cellular structures located in the brain and spinal cord, as well as their afferent and efferent pathways.

Four levels can be distinguished in the extrapyramidal system (Fig. 3):

- cortical formations - - premotor zones of cerebral hemispheres;
- subcortical (basal) nuclei: caudate and lenticular nucleus consisting of a shell, lateral and medial pallidum;
- main stem formations: substantia nigra, red nuclei, reticular formation, subthalamic nucleus, nuclei of the medial longitudinal bundle (Darkshevich), vestibular nuclei, cover of the midbrain;
- the spinal level is represented by closely connected descending conductive paths that end near the cells of the anterior horns of the spinal cord. Further, extrapyramidal influences go to the muscles through the system of alpha and gamma motoneurons.

From an evolutionary point of view, the extrapyramidal system is divided into two parts, neostriatal and paleostriaal (or pallidonigraal), based on morphological and functional features. *The neostriatal system (neostriatum)* includes cortical structures, the caudate nucleus and the shell. *The paleostriatal system* includes the lateral and medial globus pallidum, substantia nigra, subthalamic nucleus, nucleus of the medial longitudinal bundle, vestibular nuclei, midbrain cover, and some other structures. Neostriar and paleostriar systems, which function in harmony, balance each other, are conventionally united by the concept of striopallidar system . The neostriate system is younger than the paleostriate system, both in phylogenetic and ontogenetic terms, it is considered a higher subcortical regulatory and coordination center for the organization of movements, a powerful inhibitory regulator of the motor system. It inhibits the paleostriatal system, which activates motor function.

The subcortical nuclei are the leading structures of the extrapyramidal system. They have a large number of connections with other departments of the nervous system, which ensure the inclusion of extrapyramidal apparatus in the system of voluntary movements. Afferent fibers carry information from the thalamus, cerebellum, and retina. The neostriatal system receives afferent connections from many parts of the cerebral cortex, especially from the motor zones of the frontal

lobe. Descending impulses from the extrapyramidal system through the structures of the midbrain and medulla (red, vestibular nuclei, reticular formation, roof plate, motor nuclei of cranial nerves) reach the segmental apparatuses, coordinating muscle tone and motor activity. The functions of the extrapyramidal system are carried out thanks to the presence and its structures of neurotransmitters. The substantia nigra contains the producing neurons *dopamine,* which is formed here in granules. The latter enters the caudate nucleus via the dopaminergic nigrostriatal pathway, where it is released in the synoptic apparatus. Dopamine inhibits the function of the caudate nucleus, blocking the production of the excitatory mediator acetylcholine by striatal cholinergic neurons. Thus, dopamine reduces the inhibitory effect of the caudate nucleus on motility. Dopamine also enters limbic structures, the hypothalamus and the frontal lobe of the brain, providing control over mood, behavior, and the initiation of motor acts. A decrease in its content in these structures leads to an increase in the inhibitory effects of the caudate nucleus on motor activity with the appearance of hypo or akinesia, emotional disorders.

In addition, the inhibitory mediator *gamma-aminobutyric acid* (GABA) is produced in the caudate nucleus, which is transmitted to the substantia nigra through the gamkergic strionigral pathway and controls the synthesis of dopamine. There are other neurotransmitters in the structures of the extrapyramidal system - *norepinephrine, serotonin, glutamic acid, neuropeptides.* The function of all mediator systems is normally balanced, there is an equilibrium between them. In case of its violation, various pathological clinical syndromes arise. Damage to the substantia nigra and degeneration of the nigrostriatal pathway lead to a decrease in the synthesis and amount of dopamine, which is clinically manifested by the picture of hypertonic-hypokinetic syndrome, or parkinsonism.

This name of the syndrome comes from the name of the English doctor James Parkinson, who in 1817 described a hereditary disease with muscle rigidity, akinesia and tremor, which was later named Parkinson's disease. Similar symptoms also occur as a result of craniocerebral trauma, carbon monoxide (carbon monoxide) poisoning, manganese, after suffered lethargic encephalitis and for other reasons. In this case, it is called parkinsonism, adding the etiology (toxic, post-encephalitic, post-traumatic, etc.).

In the presence of parkinsonism, the effect of dopamine on the caudate nucleus decreases, which, due to the increase in cholinergic activity, is inhibited and increases its inhibitory effect on motor activity. Hypokinesia, muscle stiffness and static tremor occur. *Hypokinesia or akinesia* (poverty of movements) is manifested by a set of symptoms - hypomimia, rare blinking, monotony of speech (bradylalia), micrographia, disappearance of joint movements, especially in the upper limbs during walking (acheirokinesis), a decrease in general motor activity, movement initiative, a violation of the inclusion process in motion Under such conditions, patients seem to freeze during movement, cannot start walking immediately, and stomp in place. When walking, they cannot stop immediately. The gait is slowed, with small steps, shuffling, with a tendency to accelerate. While walking forward, the patient cannot stop suddenly. In this case, the trunk seems to overtake the lower limbs, the balance is disturbed and the patient may fall. This phenomenon is called propulsion. Similarly, the patient cannot stop suddenly while walking backwards (retropulsion) or sideways (lateropulsion).

Muscle stiffness, which occurs in the case of parkinsonism, is characterized by an increase in muscle tone evenly in all muscle groups, according to the type of waxy or plastic stiffness. During passive movements in the limbs, there is sometimes a kind of discontinuity, gradual stretching of the muscles, which has received the name of the "cogwheel" symptom. General stiffness, increased muscle tone determine the characteristic posture of the patient: the head is tilted forward, the body is hunched over, the arms are bent at the elbow joints (beggar's pose).

The tremor has a small rhythmic character with a frequency of 4-5 oscillations per 1 second, occurs at rest, increases in case of excitement, decreases or disappears during sleep and voluntary movements. Initially, tremors occur in the hand of one hand (such as "counting coins" or "taking pills", "flexion-extension" of the fingers). As the disease progresses, it spreads according to the hemitype, covers the head (like "yes-yes") or becomes generalized. Vegetative disorders in the

form of increased salivation, oily skin, excessive sweating, and delayed bowel movements often occur. The majority of patients have mental disorders such as lack of initiative, lethargy, a characteristic peculiar viscosity, insistence, tendency to repeat the same questions, depression, and dementia (lack of intelligence) may occur in the later stages of the disease.

Sometimes patients with parkinsonism have *paradoxical kinesias,* when they can temporarily perform voluntary movements (dancing, skating, etc.) due to a short-term decrease in muscle tone. Such a phenomenon, which has not yet found a final explanation, can be observed after waking up, during stressful situations. Patients with parkinsonism are characterized by the so-called fixation rigidity, which leads to an increase in tonic postural reflexes (position reflexes). Their essence is that the return to the original position of the body part after the movement is disturbed. Thus, as a result of increased plastic tone in the muscles of the neck and proximal parts of the upper limbs, the doctor raised the head of the patient, who is lying on his back, as if frozen in this position, then descends slowly *(a symptom of an air bag).* Passively bent in the knee joint, the patient's lower limb, lying on the stomach, remains in this position even after the cessation of irritation, and slowly descends. After a sharp passive dorsiflexion of the foot, it maintains this position for some time.

To detect hidden extrapyramidal muscle hypertension, the Noyka-Ganev test is used. When checking the muscle tone in the upper limb by means of passive movements in the elbow joint, the patient is asked to raise his leg. Simultaneous lifting of the lower limb causes increased tone in the muscles of the upper limb.

Correction of mediator shifts in the presence of parkinsonism is carried out with the use of anticholinergic drugs (cyclodol, parkopan, amisil) and drugs that stimulate dopaminergic transmission (levodopa, sinemet, nacom, madopar, parlodel, yumex, midantan, symmetrel, etc.).

Materials for the activation of education seekers during lectures

• What are the functions of the nervous system?
• What is the structural unit of the nervous system?
• Lists movement disorders
• State the difference between damage to the peripheral spinal nerves and the anterior horns of the spinal cord
• What are the neurochemical changes in parkinsonism?

General material and methodical provision of the lecture

1. Educational premises.
- a lecture room

2. Equipment.
- desks
- chairs
- blackboard, chalk

3. Equipment
- Stand "Basic neurological tools"
- Electrified model "Conducting pathways of the brain and spinal cord"
- Epidiascope, slides
- Neurological tools for patient examination

4. Illustrative materials
- Multimedia presentations
- Thematic patients

Literature

1. Neurology: study guide / [I.A. Hryhorova, L.I. Sokolova, R.D. Gerasymchuk, A.S. Son, etc.] edited by I.A. Grigorova, L. I. Sokolova - 3rd edition - Kyiv, Medical University "Medicine", 2020 - 640 p.

2. Topical diagnosis of pathology of the nervous system. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.

3. Methods of examination of a neurological patient: teaching. manual / edited by L. I. Sokolova, T. I. Ilyash. - 2nd edition. - Kyiv: Medicine, 2020. - 143 p.

4. Emergency medicine. Emergency medical care: textbook / I.S. Zozulya, V.I. Bobrova, H.G. Roschyn and others / edited by I.S. Cuckoos - 3rd edition, trans. and additional - Kyiv. - VSV "Medicine", 2017. - 960 p.

5. Negrych T.I., Bozhenko N.L., Matvienko Yu.Sh. Ischemic stroke: secondary inpatient care: education. manual Lviv: LNMU named after Danylo Halytskyi, 2019. – 160 p.

Additional literature

1. Bozhenko M.I., Negrych T.I., Bozhenko N.L., Negrych N.O. Headache. Study guide.- K.: Medknyga Publishing House, 2019. - 48 p.

2. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 1 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2020. - 258 p.

3. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 778 p.

4. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 642 p.

Electronic information resources

Clinical guidelines in neurology. (Order of the Ministry of Health of Ukraine No. 487 dated August 17, 2007) https://zakon.rada.gov.ua/rada/show/v0487282-07#Text

Lecture No. 2

Topic: Higher brain functions and their disorders. Localization of functions in the cerebral cortex and lesion syndromes.

Actuality of theme. For the study of higher nervous functions (language, gnosis, praxis, etc.), the issue of determining the localization of functions in the cerebral cortex is of significant importance. The correct methodological approach to the study of the main patterns of brain activity makes it possible to understand the localization of functions in the cerebral cortex , a process determined by the conditions of the internal and external environment. Disruption of language functions leads to the disintegration of the second signaling system of a person and disability.

Entire lectures:

Educational:

To acquaint students with various functions of the cerebral cortex. Create an idea about the importance of the second signaling system for normal human activity (first level of abstraction).

On the basis of the lecture material, provide knowledge about the cortical symptoms of impaired vision, hearing, taste, smell, praxis, speech, etc. (II level of abstraction).

Provide an opportunity to master the skills of diagnosing aphasia, agnosia, apraxia, anosmia, ageusia, alexia, agraphia, acalculia, etc. (III level of abstraction).

Educational:

Aimed at the development of a professionally significant substructure of the personality, education of modern professional thinking; ensuring the assimilation of the importance of domestic clinical scientific and pedagogical schools.

Plan and organizational structure of the lecture

No	The main stages of the lecture and their content	Goals in levels of abstraction	Type of lecture, methods and means of activation of acquirers, equipment	Time allocation
I 1. 2.	Preparatory stage Setting an educational goal Providing positive motivation	 I I	According to the publication "Methodical recommendations for planning, preparation and analysis of a lecture"	5% (5 min)
II 3.	The main stage Presentation of lecture material according to the plan: 1. Actuality of theme 2. Definition 3. Classification 4. Etiology and main links of pathogenesis 5. Clinical picture 6. Diagnostics 7. Leading syndromes and differential diagnosis 8. Evaluation of the severity of the course 9. Treatment 10. Prevention	 II II II II II II II II II II	Slide presentation of lecture material Extracts from the medical histories of patients. Excerpts from clinical protocols of the Ministry of Health of Ukraine on providing medical care to patients.	85% (75 min)
III 4. 5. 6.	The final stage Summary of the lecture, general conclusions Answers to possible questions Tasks for independent preparation	 III III III	List of references, questions, assignments	10% (10 min)

Content of the lecture material:

CORTEX OF THE HEMISPHERE OF THE BRAIN AND HIGHER BRAIN FUNCTIONS.

The brain (encephalon, cerebrum) includes the right and left hemispheres and the brain stem. Each hemisphere has three poles: frontal, occipital, and temporal. In each hemisphere, four lobes are distinguished: frontal, parietal, occipital, temporal, and insula.

The brain is built of multipolar nerve cells - neurons, the number of which reaches 10^{11} (one hundred billion). The average mass of the brain of an adult is 1450 g. The dependence of brain mass on the creative level of a person has not been found. The absolute brain mass of women is 100-150 g less than that of men.

The human brain differs from the brain of great apes in the significant development of the frontal lobe, which accounts for 29% of the entire mass of the brain, the frontal lobes continue to increase during the first 7-8 years of a child's life , they are associated with motor function. It is from the frontal lobes that the pyramidal pathway originates . IN the parietal lobe of the human

brain differentiates into the lower parietal lobe, its development is associated with the appearance of speech function.

The cerebral cortex covers its hemispheres, has furrows that divide it into lobes and convolutions, as a result of which its area increases significantly.

On the upper lateral (outer) surface of the hemisphere of the cerebrum, there are two larger primary furrows, the central furrow (sulcus centralis), which separates the frontal lobe from the parietal lobe, and the lateral furrow (sulcus lateralis), which is often called the Sylvian furrow; it separates the frontal and parietal lobes from the temporal lobe. The parietal-occipital groove (sulcus parietooccipitalis) can be distinguished on the medial (medial) surface of the cerebral hemispheres, which separates the parietal lobe from the occipital lobe. Each hemisphere of the cerebrum also has a lower (basal) surface.

The cortex of the cerebrum is evolutionarily the youngest formation, the most complex in terms of structure and function. It is extremely important in the organization of the vital activity of the body.

The cerebral cortex developed as an apparatus for adaptation to changing environmental conditions. Adaptive reactions are impossible without the interaction of somatic and vegetative functions. It is the cerebral cortex that provides the integration of these functions through the limbic-reticular complex. It does not have a direct connection with receptors, but receives the most important afferent information, partially already processed at the level of the spinal cord, in the stem and subcortical part of the brain. In the cortex, sensitive information is subjected to analysis and synthesis. Even according to the most careful estimates, about 10^{11} elementary operations are carried out in the human brain during one second (O. Forster, 1982). It is in the cortex that nerve cells, interconnected by many processes, analyze signals entering the body and make decisions about their implementation.

Emphasizing the leading role of the cerebral cortex in neurophysiological processes, it should be noted that this higher part of the central nervous system can function normally only under the condition of close interaction with the subcortical formations, the reticular formation of the brainstem. Here it is appropriate to recall the opinion of P.K. Anokhin (1955) that, on the one hand, the cerebral cortex is developing, and on the other - its energy supply, that is, the reticular formation. The latter controls all signals directed to the cerebral cortex, passes a certain number of them; redundant signals are accumulated, and in case of information starvation, they are added to the general flow.

Cytoarchitectonics of cerebral cortex

The cerebral cortex is the gray matter of the surface of the large hemispheres, 3 mm thick. It reaches its maximum development in the precentral gyrus, where its thickness approaches 5 mm. About 70% of all neurons of the central nervous system are located in the human cerebral cortex.

The mass of the cerebral cortex in an adult is 580 g, which is 40% of the entire mass of the brain. The total area of the cortex is about 2200 cm2, which is three times greater than the area of the inner surface of the cerebral skull, to which it adjoins. Two-thirds of the cerebral cortex is hidden in a large number of furrows.

The first rudiments of the cortex of the cerebrum are formed in the human embryo on the 3rd month of embryonic development, on the 7th month most of the cortex consists of six plates, or layers. The German neurologist K. Brodmann (1903) gave the layers the following name: molecular plate (lamina molecularis), external granular plate (lamina granularis externa), external pyramidal plate (lamina pyramidalis externa), internal granular plate (lamina granularis interna), internal pyramidal plate (lamina pyramidalis interna seu ganglionaris) and multiform plate (lamina miltiformans).

The morphological structure of the cortex of the cerebrum in its various sections was described in detail by Professor V. O. Bets of Kyiv University in 1874. He first described giant pyramidal cells in the fifth layer of the cortex in front of the central gyrus. These cells are known as Betz cells. Their axons go to the motor nuclei of the brain and spinal cord, forming a pyramidal

pathway. V.O. Betz first introduced the term "cortical cptoarchitectonics". This is the science of the cellular structure of the cortex, the number, shape and location of cells in its various layers. Cytoarchitectonic features of the structure of various areas of the cortex of the cerebrum are the basis for dividing it into areas, subdivisions, fields and subfields. The topography of the cortical fields of the human cerebrum was studied in detail by K. Brodman, who compiled the corresponding maps of the cortex. According to K. Brodman, the entire surface of the cortex is divided into 11 areas and 52 fields, which differ in the features of cellular composition, structure and executive function.

In humans, three formations of the cerebral cortex are distinguished: new, ancient and ancient.

The new cortex (neocortex) is 96% of the entire surface of the cerebrum and includes the occipital lobe, the upper and lower parietal, precentral and central gyri, as well as the frontal and temporal lobes of the brain, the insula. This is a homotypic bark, it has a lamellar type of structure and consists mainly of six layers. Plates vary in their power of development in different fields. In particular, in the precentral gyrus, which is the motor center of the cerebral cortex, the external pyramidal, internal pyramidal and multiform plates are well developed, and the external and internal granular plates are worse.

The ancient cortex (paleocortex) includes the olfactory tubercle, transparent septum periamygdalar and prepiriform areas. It is connected with ancient functions of the brain related to smell and taste. The ancient bark differs from the bark of the new formation in that it is covered with a white layer of fibers, part of which consists of fibers of the olfactory tract (tractus olfactorius). The cortex of the limbic system is also an ancient part of the cortex, it has a three-layer structure.

The ancient bark (archicortex) includes Ammon's horn, dentate gyrus. It is closely related to the area of the hypothalamus (corpus mammillare) and the limbic cortex. The ancient crust differs from the ancient one in that it is clearly separated from the subcortical formations. It is functionally related to emotional reactions.

The ancient and ancient cortex makes up about 4% of the cortex of the cerebrum. It does not go through the period of the six-layered structure during embryonic development. Such a bark has a three- or one-layer structure and is called heterotopic.

The myeloarchitectonics of the cortex is characterized by the presence of six layers of fibers within the cerebral cortex with different terms of their myelination.

Among the nerve fibers of the cerebrum, there are associative fibers that connect separate areas of the cortex within one hemisphere, commissural fibers that connect the cortex of different hemispheres, and projection fibers that connect the cortex with lower parts of the central nervous system.

Regarding functions, three main types of cortical activity are distinguished. The first type is related to the activity of individual analyzers and provides the simplest forms of cognition. This is the first signal system. The second type includes the second signal system, the work of which is inextricably linked with the function of all analyzers. This is a more complex level of cortical activity that is directly related to language function. For a person, words are the same conditioned stimulus as reality signals. The third type of cortical activity ensures purposefulness of actions, the possibility of prospective planning, which is functionally related to the frontal lobes of the cerebral hemispheres. So, abstract thinking is connected with the second signaling system, which is the highest form of human nervous activity.

Localization of functions in the cerebral cortex

The primary projection fields correspond to those architectural areas where the cortical parts of the analyzers are localized: the analyzer of general sensitivity - in the central gyrus, olfactory and auditory - in the temporal lobe, visual - - in the occipital lobe. These fields are associated with simple, elementary functions: general sensitivity of the skin, hearing, smell, vision. These are fields that cannot provide an integral function of perception, they only respond to certain

stimuli of one modality and do not respond to stimuli of another modality. In the primary fields, neurons of the IV afferent layer are the most developed. Primary projection fields are characterized by the somatotopic principle of structure, that is, the representation of sensitive functions in certain areas of the cortex.

Secondary projection fields are located around the primary ones. They are not directly related to specific pathways. In the secondary cortical fields, the neurons of the second and third layers of the cortex gain predominant development; there is a large number of multisensory neurons, which provides, compared to the primary fields, a different nature of response. Electric stimulation of secondary fields causes complex visual images and melodies in people, in contrast to elementary sensations (flash, sound) that occur in case of stimulation of primary fields. In secondary fields, higher analysis and synthesis takes place, more detailed processing of information, awareness of it.

Secondary fields, together with primary fields , make up the central part of the analyzer, or its core. The interaction between the neurons of these wear zones is complex, ambiguous in nature, and under conditions of normal brain activity, it is based on the sequential change of excitatory and inhibitory processes in accordance with the nature of the final result. This provides dynamic properties of localization. The described functional organization of the cortex in the form of clearly divided fields according to the principle of modal specificity is most pronounced in humans and higher representatives of the animal world.

Tertiary projection fields are associative zones located in overlapping areas of individual analyzers. Two main associative zones are distinguished: in the frontal lobe in front of the precentral gyrus and at the border between the secondary zones of the parietal, occipital and temporal lobes. Tertiary projection fields, or overlap zones, are not directly connected to peripheral receptor apparatuses, but they have a close connection with other areas of the cortex, including projection zones. Signals from the associative nuclei of the thalamus also arrive here.

In the cerebral cortex, especially in the area of association zones, neurons are arranged according to the type of functional columns. The columnar organization of cortical zones is characterized by the vertical arrangement of neuronal elements (columns) with similar functional properties. This means that all six layers of cortical cells of the associative zones lying perpendicular to its surface are involved in the processing of sensory information coming from peripheral receptors. Most of the neurons of the tertiary zones have multimodal properties. They provide integration of signals coming from different analyzers. This is where the formation of relevant feelings is completed, and complex analytical and synthetic functions are performed. Tertiary projection fields are directly related to higher mental functions. Learning and memory processes are connected with the function of these zones. They are characteristic only of the human brain. The sensory areas of the cerebral cortex are closely connected with the motor areas located in front of the central sulcus. Together they form a single sensorimotor field. Primary, secondary and tertiary zones are also distinguished in the motor cortex.

The primary motor zone of the cortex (field 4) is located directly in front of Roland's sulcus. This is the precentral gyrus, from the 5th layer of which originates the pyramidal path connecting the cortex of the cerebrum with the cells of the anterior horns of the spinal cord. Like the somatosensory zone, it has a clear somatotopic organization. In more than 50% of the surface of this area in humans, the upper limbs and muscles of the face, lips, and tongue are represented, taking into account the importance of the function they perform (subtle movements, speech).

The secondary motor zone of the cortex is premotor (field 6), located in front of the primary zone of the cortex and in the depth of the sylvian sulcus. This area of the cortex, together with the primary motor area, the subcortical nuclei and the thalamus, controls many more complex movements.

The tertiary motor zone of the cortex covers the frontal parts of the frontal lobes (prefrontal area). Neurons of this cortical zone receive numerous impulses coming from the sensorimotor cortex, visual and auditory areas of the cortex, thalamus, as well as from subcortical nuclei and

other structures. This zone ensures the integration of all information processes, the formation of plans and action programs, and controls the most complex forms of human behavior.

The primary sensory and motor areas of the cortex are mainly associated with the opposite half of the body. As a result of this organization of contralateral connections, the sensory and motor functions of both hemispheres of the cerebrum are symmetrical in both humans and animals.

The processes of excitation and inhibition in the central nervous system play a significant role in performing the functions of the cerebral cortex. Excitation is associated with the occurrence of temporary depolarization in the neuron. Exciting mediators can be various substances: norepinephrine, dopamine, serotonin. Inhibition in the cerebral cortex is carried out by inhibitory interneurons. The main mediator of cortical inhibition is GABA. Due to the convergent-divergent organization of the nervous system, similar specific oscillations and the corresponding distribution of excitation and inhibition occur simultaneously in the cortical and subcortical neurons of the brain. This creates the basis for integrative activity of the brain, which is associated with higher mental functions: perception, cognition, memory, state of consciousness.

The left hemisphere of the brain plays an exclusive role in linguistic and speech activity, specializes in sequential analytical processes (categorical hemisphere). It is the basis of logical, abstract thinking and functions under the direct influence of the second signaling system. The right hemisphere of the brain is functionally connected with the perception and processing of exteroceptive, proprioceptive, and interoceptive impulses, which ensures the perception of specific images, objects, people, animals, i.e. performs a gnostic function, including gnosis of one's own body (representative hemisphere). Its importance in perceiving space, time, and music has been proven. The right hemisphere is the basis of imaginative, concrete thinking. Therefore, the right hemisphere of the brain should not be considered subordinate to the left. The result of research in recent years was the replacement of the theory of dominant hemispheres with the concept of complementary (corresponding) specialization of the hemispheres.

Disorders of higher brain functions

Higher brain functions are functional systems with a complex hierarchical structure. They are conditionally reflexive in their mechanism, have a social-historical origin and are formed in every person after his birth and only in the conditions of the social environment, under the influence of the culture of the given society, including language. Higher brain functions include language, gnosis, praxis, memory, thinking, consciousness, and other mental functions.

Language and its disorders. Language is an exclusively human form of activity, which is a means of thinking and communication between people. Language is one of the functional foundations of human intelligence and a leading element of culture. Cytoarchitectonic fields of the cerebral cortex associated with language are characteristic only of humans: premotor zone – the back part of the lower frontal gyrus (fields 44, 45); central gyrus lower part of cortical fields 1, 2, 5, 7; posterior part of the upper temporal gyrus cortical field 22 (Wernicke); lower parietal lobe cortical fields 39, 40; posterior parts of the temporal lobe cortical field 37; frontal areas of the frontal lobe — cortical fields 9, 10, 11 and 46 of the categorical (left) hemisphere.

Therefore, there is no single language center. The language function is connected mainly with the secondary projection fields of the analyzers, as well as the tertiary zones of the cortex (fields 39, 40). Their representation in the cortex is asymmetrical: in most right-handed people - in the left hemisphere, in left-handed people - in the right hemisphere.

Wernicke's center (zone) is responsible for the perception of auditory and visual information. Through an arcuate bundle, it connects with Broca's center (zone), where information received from Wernicke's center is detailed and organized. The information then passes through the speech articulation area in the insula to the motor cortex, initiating the corresponding movements of the lips, tongue, and larynx, reproducing speech.

In language, two communication channels are distinguished: verbal, purely human, left-hemisphere and prasodic (intonational) right-hemisphere. Therefore, both hemispheres of the brain participate in the implementation of language activity, but different areas of the cortex play different roles in this process.

There are two main types of speech: impressive and expressive. Impressive language provides understanding of spoken and written language.

Expressive speech is the process of expressing thoughts in the form of active speech or independent writing.

So, the structure of language consists of two processes: speech and perception. Disturbance of the speech process is called motor, expressive, aphasia, speech perception disorder - - impressive aphasia. Aphasia (from the Greek phasis - speech) is a violation of the ability to speak or understand language, which occurs as a result of damage to the cortical language centers of the categorical hemisphere. In this case, the corresponding muscles (larynx, tongue, lips), as well as the innervation of the speech apparatus are intact.

Depending on the localization and degree of brain tissue damage, two main types of aphasia are distinguished: motor (expressive) and sensory (impressive, receptive). Semantic, amnestic and total aphasia are also distinguished.

According to the classification of A.R. Luria (1969), three forms of expressive language disorders can be observed: afferent, efferent and dynamic motor aphasia.

Afferent motor aphasia occurs in case of damage to the lower parts of the central gyrus, which provide the kinesthetic basis for the movements of the articulatory apparatus. As a result, the articulation of sounds that are similar in place (pre-lingual: "l", "n", "t", "d") or in the manner (syllabic: "sh", "z", "sh", "x) is especially grossly disturbed ») education. This leads to the replacement of some articulations by others, to the replacement of sounds - phonemes (instead of "l" is pronounced "n", or instead of "sh" - "z", etc.). The patient loses all types of oral speech - spontaneous, automated, repeating suggested words, naming objects. Reading and writing also suffer. Often this type of aphasia is combined with oral apraxia.

Efferent motor aphasia (Broca's aphasia) occurs if Broca's center is affected in the posterior lobe of the inferior frontal gyrus of the left hemisphere (in right-handed people). This type of aphasia is characterized by a violation of switching processes from one language unit (sound, word) to another. In contrast to afferent motor aphasia, the articulation of individual sounds is preserved, but the pronunciation of a series of sounds or a phrase suffers. Productive speech is replaced by constant repetition of individual sounds (literal perseveration) or words (verbal perseveration), and in severe cases manifests as a speech embolus. A characteristic feature of speech in the case of efferent aphasia is the so-called telegraphic style: sentences are built mainly from nouns, verbs are almost absent in them. This variant of aphasia is also accompanied by impaired writing, reading, and naming of objects.

Dynamic motor aphasia occurs if the cortical area anterior to Broca's center is affected. The main defect of this form of aphasia is the lack of language initiative, language spontaneity. The patient cannot actively express his opinion, ask questions, but he repeats certain words and sentences well, answers questions correctly. The basis of this form of aphasia is a violation of internal language, the main function of which is programming and sentence construction.

Sensory aphasia (Wernicke's aphasia) is characterized by the loss of the ability to understand language in general, both foreign and own. This type of aphasia occurs if the posterior part of the superior temporal gyrus (Wernicke's center) is affected. The basis of language comprehension disorder is phonemic hearing disorder. A phoneme is understood as a semantic and recognition feature of a language. The patient loses the ability to recognize sounds and understand words. Language is perceived by him as noise or conversation in an unfamiliar language. Not understanding the language of others, the patient tries to speak quickly and a lot (logorrhea - speech incontinence). In severe cases, the speech of such patients is a set of words unrelated in meaning (verbal jumble), with numerous literal (replacement of one letter by another) and verbal (replacement of one word with another, close in meaning) paraphasias. Patients usually do not realize their speech defect.

So, sensory aphasia is characterized by difficulty understanding foreign language and poor auditory control over one's own language. Sensory aphasia is usually combined with impaired reading (alexia) and writing (agraphia). *Semantic* aphasia occurs if the temporal-parietal-occipital

region of the left hemisphere is affected in right-handed people. This is one form of expressive language disorder. The basis of aphasia is a violation of spatial synthesis, as a result of which the patient does not understand the content of sentences reflecting spatial relations. Such patients are not understand relationships expressed using prepositions (a circle under a square or a triangle under a circle). In the presence of semantic aphasia, patients cannot understand the semantic differences between comparative ("Ira is darker than Katya, but lighter than Olya. Who is the fairest?"), attributive ("father's brother" and "brother's father") and inverse ("a cat with ate a mouse", "a mouse ate a cat") constructions. This form of aphasia is combined with a counting disorder (acalculia).

Amnestic aphasia occurs if the lower part of the parietal and posterior parts of the temporal lobes are affected. It consists in the fact that the patient forgets the names of objects, but knows their purpose. For example, if you show a pen, the patient will say "this is what they write with." Sometimes it is enough to suggest the first syllable of a word forgotten by the patient so that he can name it correctly. Language comprehension is usually not impaired. Verbs predominate in the language of a patient with amnestic aphasia, and there are few nouns in it.

Total aphasia is characterized by the loss of expressive and expressive language in all its manifestations. Most often, this is found in the case of large foci of damage to both motor and sensory speech centers, which occurs in the case of brain infarction due to blockage of the middle cerebral artery.

It is necessary to remember that all parts of the language zone function in the closest interaction. Therefore, "pure cases" of language disorders are almost never observed in the clinic : elements of sensory aphasia can often be detected in the presence of motor aphasia , and vice versa. Amnestic disorders are often combined with sensory and semantic aphasic disorders.

Damage to the representative hemisphere of the brain negatively affects the ability to recognize differences in intonation.

The study of speech disorders is carried out according to a certain system. In clinical practice, the examination begins during the collection of the patient's history. Evaluation of expressive language begins with familiarization with spontaneous language. At the same time, they pay attention to the lexical support of the patient's language, the correct construction of phrases, possible difficulties when choosing words and composing phrases. Then they move on to the study of repeated language.

It is proposed to repeat individual sounds that are similar in place or method of formation. The ability to reproduce individual syllables: "ba-pa", "da-ta", "to-do", simple words, individual phrases is studied. The possibility of an automated language is tested by the ability to perform calculations, a list of days of the week. It is also suggested to name and show objects to detect amnestic disorders.

The study of impressive language begins with checking the understanding of spoken language, individual words, phrases. The patient is offered to perform various simple actions. Tasks on the content of complex logical-grammatical constructions are also offered : comparative, inverse, attributive. Along with oral language, comprehension of written language and reading aloud is tested. *To* study writing, they suggest writing something independently, writing under dictation.

In everyday clinical practice, other types of speech disorders are also observed, in particular, *dysarthria.* The latter, in contrast to motor aphasia, is characterized by articulation disorders, vagueness, and unintelligibility of speech. However, no matter how difficult and unclear the patient speaks, he forms phrases and sentences correctly; vocabulary in case of dysarthria also does not suffer. In severe cases, speech becomes impossible due to lack of articulation (anarthria). In the case of dysarthria, unlike aphasia, the ability to write and understand oral and written language is preserved. Dysarthria can occur in the presence of peripheral paresis or paralysis of the muscles of the articulatory apparatus (an element of the bulbar syndrome). , as well as under the condition of central paresis of these muscles, which is observed in the case of bilateral damage to the cortical-nuclear pathways (an element of the pseudobulbar syndrome). Dysarthria also

occurs if other parts of the brain are affected - striopallidar system, cerebellum. In the case of parkinsonism, the speech becomes monotonous, indistinct, fading, in the case of cerebellar diseases, chanted. Deaf-mute and mutism are well-known non-aphatic speech disorders . The latter can be a manifestation of reactive neurosis, hysteria or mental illness. It is also interesting that deaf patients in case of damage to the categorical hemisphere of the cerebrum lose the ability to communicate in sign language (V. Ganoni, 2002).

Alexia is a reading disorder caused by impaired understanding of the text. It occurs in the presence of almost all types of aphasia, but it can also be observed in the case of damage to the left angular gyrus . During the study , it is suggested to read both aloud and about oneself.

Agraphia is characterized by the loss of the ability to write correctly , provided the motor function of the upper limb is preserved. It is also often combined with motor and sensory aphasia. Sometimes agraphia occurs in isolation, if the affected center of writing is the back part of the middle frontal gyrus. Another center of writing is located on the border between the occipital , temporal and temporal lobes. It should be noted that the center of the graph is two-sided. The written language is examined by asking the patient to perform the following actions: write down individual letters, words, phrases; write words, letters, phrases under dictation; write the name of the displayed items; write your last name, first name, address, days of the week, months (automated writing); write about your illness (spontaneous writing).

Acalculia is a violation of the ability to perform arithmetic operations caused by damage to the left angular gyrus (field 39). Very often it is combined with semantic aphasia. The research methodology involves automated counting (multiplication table), performing various mathematical operations: addition, subtraction, multiplication, division.

Apraxia is the loss of the ability to perform purposeful motor skills developed in the process of individual experience, in the absence of paresis or movement coordination disorders.

There are several main types of apraxia.

Kinesthetic, or afferent, apraxia occurs if the lower parts of the parietal lobe of the left hemisphere of the cerebrum are affected; it is caused by a violation of the kinesthetic synthesis of movements. Kinesthetic apraxia may not manifest itself in the whole limb, but only in the muscles of the fingers, and especially during fine movements. For example, the patient cannot fasten buttons, *light* a match, pour water into a glass. Voluntary movements are performed only under the condition of constant visual control.

At the same time, difficulty in the movements of speech muscles, in particular lips, tongue, cheeks *(oral kinesthetic apraxia) can be observed.* The patient is unable to perform articulatory movements according to the task, cannot pronounce sounds close in articulation. Oral apraxia is always combined with motor afferent aphasia.

Kinetic, or *efferent, apraxia* occurs if the premotor zone of the frontal cortex is affected. It is characterized by a violation of not only complex movements, but also actions according to the task, according to imitation. Often in the case of this form of apraxia, perseveration is observed, that is, repetition of the same movement. For example, the patient continues to open his mouth when asked to open his mouth and at any new task (close his eyes, show his tongue). Oral and articulatory kinetic apraxia often occur at the same time.

Ideational apraxia, or *apraxia of thought,* occurs as a result of damage to the supramarginal gyrus of the parietal lobe of the left hemisphere of the brain (in right-handed people) and is always bilateral. This center of praxis is unilateral, its connection with the opposite hemisphere is carried out through the corpus callosum . Damage to the fibers of the latter is accompanied by apraxia only in the left hand. In the presence of this form of apraxia, the plan or idea of a complex action is lost, its phases are rearranged. For example, when offered to light a cigarette, the patient may rub the box not with a match, but with a cigarette. The patient cannot also perform certain verbal tasks (threaten with a finger, give a military salute), but can repeat and imitate the doctor's actions.

Constructive apraxia is most often caused by damage to the angular gyrus of the parietal lobe of the left hemisphere of the brain. With this type of apraxia, the patient cannot make a whole

out of parts, for example, make a certain shape from matches (rhombus, square, triangle). Apraxic construction *disorders* are also bilateral.

Spatial apraxia occurs as a result of damage to the inferior parietal and parietal-occipital areas of the left hemisphere. It is accompanied by a violation of spatial relations during the performance of complex motor acts. For example, a patient cannot draw a plan of a room according to the task.

To detect apraxia, the patient is offered to perform certain actions first with real objects (combing a hair, cutting bread, lighting a match), and then with imaginary ones (showing how to drive a nail, pour water into a glass). It is also necessary to offer the patient to perform some actions (fasten buttons, give a military salute, make a figure out of matches).

Agnosia is a violation of the recognition of familiar objects based on their characteristics. Agnosia occurs under the condition of violation of secondary simple receptor functions, which are the basis of cognition. This means that in the case of agnosia, elementary forms of sensitivity are preserved, and complex forms of analytic-synthetic activity are disturbed within the limits of a certain analyzer. The following types of agnosia are distinguished: visual, auditory, and tactile and non-aesthetic. Taste and smell agnosia is relatively rare .

Visual (optical) agnosia occurs if the outer surface of the left occipital lobe is affected. Patients cannot recognize objects by their external appearance, but they immediately recognize them when they take them in their hands and feel them. Partial optical agnosia includes color agnosia - the inability to distinguish colors. One of the types of visual agnosia is alexia.

Auditory agnosia is the loss of the ability to recognize objects by their characteristic sounds: a clock ticks , a dog barks . The basis of this type of agnosia is damage to the temporal lobe, often bilateral. One of the forms of auditory agnosia is sensory aphasia.

Tactile-kinesthetic agnosia, or astereognosis, consists in the impaired ability to recognize objects by feeling them. Astereognosis is detected if the upper parietal lobe is affected mainly in the left hemisphere of the brain. In this case, all elementary types of sensation and kinesthetic sensations are preserved. Non-recognition of objects by palpation by patients with loss of superficial and deep sensitivity in the studied upper extremity is referred to as *pseudostereognosis.* Such disorders occur if the middle section of the central gyrus is affected, in the case of polyneuropathy

Nyukhov and gustatory agnosia is the loss of the ability to identify olfactory and gustatory sensations. It is observed as a result of damage to the mediobasal areas of the cortex of the temporal lobe.

Disorders of the body scheme are most often caused by damage to the cortex of the parietal lobe of the right hemisphere of the cerebrum around the fissura interparietalis. their variety is *autotopoagnosia* - - impaired recognition of one's own body and its parts. It may seem to a patient with a violation of the body scheme that his limbs are enlarged (macropsia), then reduced (micropsia) or changed not only in size, but also in shape (metamorphopsia) . A variant of autotopoagnosia is agnosia of the fingers of the hand and other parts of the body. Patients confuse right and left sides, claim that they have many arms or legs (polymelia). Most often, patients feel a third arm and leg. *Anosognosia (Anton's syndrome)* belongs to the same group of disorders , when the patient is not aware of his defect (motor, auditory, etc.). Anosognosia is often combined with autotopoagnosia and occurs against the background of gross disorders of proprioceptive sensitivity.

Symptoms of damage to different parts of the brain

The frontal lobe of the cerebral hemispheres is located in front of Roland's sulcus and includes the precentral gyrus, premotor and pole-prefrontal zones. On the outer surface of the frontal lobe, in addition to the vertical precentral gyrus, three more horizontal ones are distinguished: the upper, middle, and lower frontal gyri. On the inner surface, the frontal lobe is separated from the lumbar gyrus by the callosal-marginal furrow. Orbital and rectus gyri are located on the basal (lower) surface. The latter is localized between the inner edge of the hemisphere and the olfactory groove. The olfactory bulb and the olfactory pathway are located in

the depth of this furrow. The cortex of the basal part of the frontal lobe of the cerebrum is phylogenetically older than the cortex and architecturally closer to the limbic system.

The function of the frontal lobes is related to the organization of voluntary movements, motor mechanisms of speech and writing, regulation of complex forms of behavior, and thought processes.

Clinical symptoms of damage to the frontal lobe of the cerebrum depend on the localization, prevalence of the pathological process, as well as its stage: loss of function due to damage or functional blockade or irritation of certain structures.

efferent motor systems originate in the cortex of the frontal lobes . In particular, in the fifth layer of the precentral gyrus there are giant pyramidal neurons, the axons of which form corticospinal and corticonuclear pathways (pyramidal system). Therefore, in case *of destruction of the cortex of the precentral gyrus ,* central paresis or paralysis is observed on the opposite side of the body according to the monotype, i.e., the upper or lower limb is damaged depending on the location of the cortex lesion.

Irritation of the precentral gyrus is accompanied by attacks of cortical (Jackson's) epilepsy, which is characterized by clonic convulsions of individual muscle groups corresponding to the areas of the cortex that are irritated. These attacks are not accompanied by loss of consciousness. They can turn into a general convulsive attack.

As a result *of damage to the posterior part of the middle frontal gyrus ,* there is a paresis of looking in the opposite direction (the eyes passively turn towards the lesion). If this zone is irritated, convulsive twitching of the eyes, head, and the whole body in the opposite direction from the pathological focus (adversative attacks) occur . Irritation of the inferior frontal gyrus causes attacks of chewing movements, sucking, licking, etc. (opercular attacks).

From the premotor zone of the cortex of the frontal lobe, there are numerous efferent pathways to the subcortical and trunk formations (frontothalamic, frontopalidar, frontorubral, frontonigral pathways), which are necessary for the implementation of automated habits, activity and purposefulness of actions, motivation of behavior and provision of an appropriate emotional state. Therefore, in the presence *of damage to the premotor cortex,* patients experience various extrapyramidal disorders. Most often , hypokinesia is observed , which is characterized by a decrease in motor initiative and activity. The peculiarity of this syndrome, unlike Parkinson's, is that it is almost never accompanied by tremors. Changes in tone are unclear, but in the case of deep lesions, muscle stiffness is possible. Moreover, hypokinesia or akinesia affects not only the motor, but also the mental sphere. Therefore, along with brady- and oligokinesia, bradypsychia, slowing down of thinking processes, initiatives are observed (O. R. Vinnytskyi, 1972).

In the frontal lobe injury clinic , other extrapyramidal disorders may be observed: grasping phenomena - involuntary automatic grasping of objects placed in the palm (Yanishevsky-Bekhterev reflex). Much less often, this phenomenon is manifested by obsessive grasping of objects that appear before the eyes.

Other extrapyramidal phenomena include Kokhanovsky's symptom - involuntary tightening of the orbicularis oculi muscle in response to attempts to passively open the eyelid fissure.

Damage to the frontal lobes can be accompanied by the appearance of reflexes of oral automatism (oral reflex of Bekhterev, nasolabial reflex of Astvatsaturov and distant-oral reflex of Karchikyan), as well as subcortical reflexes (palm-chin reflex of Marinescu-Radovich). Sometimes there is a bulldog reflex (Yanishevsky's symptom), when the patient convulsively clenches his jaws or grasps an object with his teeth in response to touching the lips or the mucous membrane of the oral cavity .

As a result of *damage to the front parts of the frontal lobes,* isolated (without pyramidal disorders) asymmetry of the innervation of facial muscles can occur , which is revealed during the patient's emotional reactions. This is the so-called mimic paresis of the facial muscles. It is caused by a violation of the connections of the frontal lobe with the thalamus.

It is known that the frontal-pontine-cerebellar pathways, which belong to the system of coordination of voluntary movements, begin from the pole part of the frontal lobe, or the prefrontal zone of the cortex . As a result of their damage, cortical (frontal) ataxia occurs, which is manifested mainly by trunk ataxia, gait and standing disorders (astasia-abasia). In the case of a mild injury, there is a wobble during walking with a deviation in the direction of the lesion. Patients with damage to the cortex of the frontal lobes, especially the premotor zone, may develop frontal apraxia, which is characterized by the incompleteness of actions.

Mental disorders can occur as a result of damage to the cerebral cortex of various locations. But they occur especially often in the case of pathology of the frontal lobe. Changes in behavior and mental and intellectual disorders are observed. They come down to loss of initiative, personality, interest in the environment.

Patients lack criticism of their own actions: they are prone to banal jokes (moriya), euphoria. The sloppiness of the patient is typical. Such a peculiar change in behavior and psyche is interpreted as frontal psyche.

Of the symptoms that arise in the presence *of damage to the frontal lobe of only the left hemisphere (or the right - in left-handed people), various variants of aphasia* have topical diagnostic value . Efferent motor aphasia is observed as a result of damage to Broca's center, that is, the posterior part of the inferior frontal gyrus. Dynamic motor aphasia occurs when the area anterior to Broca's center is affected. In the case of pathological processes in the posterior part of the middle frontal gyrus of the left hemisphere (in right-handed people), isolated agraphia develops .

Under the condition of *frontal-basal processes,* in particular in the case of a tumor in the area of the olfactory fossa, Kennedy syndrome develops: loss of smell or hyposmia and blindness due to atrophy of the optic nerve on the side of the lesion , and on the opposite side - stagnation in the fundus due to intracranial hypertension.

The parietal lobe is located behind the central sulcus. On its outer surface, a vertically located central gyrus and two horizontal lobes are distinguished: the upper parietal and the lower parietal. Two convolutions are distinguished in the latter: the supramarginal one, covering the end of the lateral (Sylvian) furrow, and the angular one, which is directly adjacent to the upper temporal lobe.

In the central gyrus and parietal lobes, the afferent pathways of surface and muscle-articular sensitivity end. But most of the parietal lobe is the secondary projection cortical fields, or associative zones. In particular, the somatosensory associative zone is located behind the central gyrus. The lower parietal lobe (fields 39 and 40) occupies a transitional position, which provides it with close connections not only with the tactile or kinesthetic associative zone, but also with the auditory and visual. This zone belongs to the tertiary associative zone of higher organization. It is the material substrate of the most complex forms of human perception and cognition. Therefore, E. K. Sepp (1950) considered this part of the cortex to be the highest generalizing apparatus of cognitive processes, and U. Penfield (1964) called it the interpretive cortex.

In the case *of damage to the central gyrus* in the stage of loss, anesthesia or hypoesthesia of all types of sensitivity occurs in the corresponding parts and on the opposite side, that is, according to the monotype, depending on the place of damage to the cortex. These disorders are more clearly manifested on the inner or outer surface of the limbs, in the area of the hands or feet. In the stage of irritation (irritation), there are sensations of paresthesias in the parts of the body corresponding to the irritating zones of the cortex (sensory Jackson attacks). Such local paresthesias can be the aura of a general epileptic attack. Irritation of the parietal lobe behind the central gyrus causes paresthesias on the entire opposite half of the body (hemiparesthesias).

Damage to the upper parietal lobe (fields 5, 7) is accompanied by the development of astereognosis - a violation of the ability to recognize objects by feeling them with closed eyes. Patients describe individual qualities of an object, but cannot synthesize its image. If *the middle part of the central gyrus,* where the sensitive function of the upper limb is located, is affected, the

patient also cannot recognize an object by feeling, but in such a situation he cannot describe its quality (pseudostereognosis), since all types of sensitivity in the upper limb are lost.

A pathognomonic syndrome in the case of *damage to the lower parietal lobe is* the appearance of body diagram disorders. *Damage to the supramarginal gyrus,* as well as *the area around the intraparietal sulcus,* is accompanied by agnosia of the body scheme, or autotopoagnosia, when the patient loses the sensation of his own body. He is unable to realize where his right and left sides are (right-left agnosia), he does not recognize his own fingers (digital agnosia). Mostly, this pathology occurs in the case of right-sided processes in left-handed people. Another type of disorder of the body scheme is anosognosia — not being aware of one's defect (the patient claims that he is moving his paralyzed limbs). Such patients may experience pseudopolymelia - a feeling of an extra limb or part of the body.

As a result of damage to the cortex of the angular gyrus, the patient loses the sense of spatial perception of the surrounding world, the position of his own body and the interrelationships of its parts with each other. This is accompanied by various psychopathological symptoms: depersonalization, derealization. They can be observed under the condition of full preservation of consciousness and critical thinking.

Damage *to the parietal lobe of the left hemisphere of the cerebrum* (in right-handed people) causes apraxia - a disorder of complex purposeful actions, provided elementary movements are preserved.

A lesion in the area of the supramarginal gyrus causes kinesthetic or ideational apraxia, and *a lesion of the angular gyrus* is associated with the occurrence of spatial or constructive apraxia.

In the case of pathological processes in the lower parts of the parietal lobe, agraphia often occurs. In this case, spontaneous and active writing suffers the most. There are no language disorders . It should be noted that agraphia also occurs if the posterior parts of the middle frontal gyrus are affected, but then it is accompanied by elements of motor aphasia.

If the left angular gyrus is affected, there may be a disorder of reading both aloud and about oneself (alexia).

Pathological processes in the area of the lower part of the parietal lobe are accompanied by a violation of the ability to name objects - amnestic aphasia. If the pathological process is localized on the border of the parietal, temporal and occipital lobes of the left hemisphere of the brain, right-handed people may develop semantic aphasia - a disorder in the understanding of logical and grammatical structures of speech.

The temporal lobe is separated from the frontal and parietal lobes by a lateral furrow, in the depth of which is an island (Rayleigh). On the outer surface of this lobe, the upper, middle and lower temporal gyri are distinguished, which are separated from each other by the corresponding furrows. On the basal surface of the temporal lobe, the occipital-temporal gyrus is located laterally, and the parietal (parahippocampal) gyrus is located more medially.

The primary projection fields of the auditory (superior temporal gyrus), statokinetic (at the border of the parietal and occipital lobes), gustatory (cortex around the insula) and olfactory (seaconical gyrus) analyzers lie within the temporal lobe. Each of the primary sensory zones has a secondary associative zone adjacent to it. In the cortex of the upper temporal gyrus, closer to the occipital region on the left (in right-handed people), the center of speech understanding (Wernicke's center) is located. Efferent pathways diverge from the temporal lobe to all areas of the cortex (frontal, parietal, occipital), as well as to the subcortical nuclei and brain stem. Therefore, if the temporal lobe is affected, there are violations of the functions of the corresponding analyzers, disorders of higher nervous activity.

As a result of irritation of the cortex of the middle part of the upper temporal gyrus, auditory hallucinations occur. Irritation of the cortical projection zones of other analyzers causes corresponding hallucinatory disturbances, which can be the initial symptom (aura) of an epileptic attack. Damage to the cortex in these areas does not cause noticeable disorders of hearing, smell

and taste, since the connection of each hemisphere of the brain with its perceptual devices on the periphery is bilateral. Auditory agnosia occurs with bilateral damage to the temporal lobes.

Attacks of vestibular-cortical vertigo, which are systemic in nature, are quite typical for damage to the temporal lobe. Ataxia occurs as a result of a pathological process in those areas where the temporal-pontine cerebellar pathway begins, which connects the temporal lobe with the opposite hemisphere of the cerebellum. Manifestations of astasia-abasia with falling back and to the side opposite from the lesion are possible. Pathological processes in the depth of the temporal lobe lead to the appearance of upper quadrate hemianopsia, sometimes visual hallucinations.

A peculiar manifestation of memory hallucinations are the phenomena of "deja vu " (already seen) and "jame-vu" (never seen), which occur in case of irritation of the right temporal lobe and are manifested by complex mental disorders, a dream-like state, and an illusory perception of reality.

Mediobasal lesion of the temporal lobe causes the occurrence of temporal

automatism, characterized by a violation of orientation in the surrounding world. Patients do not know the street, their house, the location of the rooms in the apartment. At the same time, irritation of the cortex very often causes various variants of temporal epilepsy, which are accompanied by autonomic and visceral disorders.

If *the posterior part of the upper temporal gyrus on the left* (in right-handed people) is affected, Wernicke's sensory aphasia occurs, when the patient ceases to understand the meaning of words, although he can hear sounds well. Amnestic aphasia is typical for processes in the posterior parts of the temporal lobe.

The temporal lobe is related to memory. Violations of working memory in the presence of its damage are associated with damage to the connections of the temporal lobe with the analyzers of other lobes of the brain. Disorders in the emotional sphere (emotional lability, depression, etc.) are frequent .

The occipital lobe on the inner surface is separated from the parietal lobe by the parietal-occipital furrow; on the outer surface, it does not have a clear boundary that would separate it from the parietal and temporal lobes. The inner surface of the occipital lobe is divided into a wedge and a lingual gyrus by a spur groove. The occipital lobe is directly related to the function of vision. The visual pathways end on its inner surface in the area of the spur furrow, that is, the primary projection cortical fields of the visual analyzer are located (field 17). Around these zones, as well as on the outer surface of the occipital lobe, there are secondary associative zones (fields 18 and 19), where a more complex and subtle analysis and synthesis of visual perceptions is carried out.

Damage to the area above the spur groove (wedge) leads to lower quadrant hemianopsia, and below it (lingual gyrus) to upper quadrant hemianopsia. If the focus of the lesion is insignificant, a defect in the form of an island appears in the opposite fields of vision, the so-called scotoma. Destruction of the cortex in the areas of the spur groove, wedge, and lingual gyrus is accompanied by hemianopsia on the opposite side. In the case of such localization of the process, the central, or macular, vision is preserved, since it has a bilateral cortical representation.

If the higher optical centers (fields 18 and 19) are affected, there are various variants of visual agnosia - the loss of the ability to recognize objects and their images. In the case of localization of the lesion on the border of the occipital lobe with the parietal lobe, along with agnosia, alexia occurs - the inability to read due to impaired understanding of written language (the patient does not recognize letters, cannot combine them into a word - verbal blindness).

The most characteristic disorders in case of irritation of the cortex of the inner surface of the occipital lobe are photopsias - flashes of light, lightning, colored sparks. These are simple visual hallucinations. More complex hallucinatory experiences in the form of figures, moving objects, with impaired perception of their form (metamorphopsia) occur in case of *irritation of the outer surface of the occipital lobe of the cortex,* especially at the border with the temporal lobe.

The limbic section of the cerebral hemispheres includes the cortical areas of the olfactory (seahorse, or hippocampus; transparent septum, cingulate gyrus) and gustatory (cortex around the islet) analyzers. These sections of the cortex have close connections with other mediobasal

formations of the temporal and frontal lobes, the hypothalamus, and the reticular formation of the brainstem. All of them make up a single system - the limbic-hypothalamic-reticular complex, which plays an important role in the regulation of all vegetative-visceral functions of the body.

Damage *to the central apparatus of the limbic department* is manifested by symptoms of irritation in the form of vegetative-visceral paroxysms or clinical signs of loss of functions. Irritative processes in the cortex lead to the development of epileptic paroxysmal disorders. They can also be limited to short-lived visceral auras (epigastric, cardiac).

Irritation of cortical olfactory and gustatory areas gives corresponding hallucinations.

Frequent symptoms *of damage to the limbic cortex of the hemispheres are* memory disorders of the Korsak syndrome type with amnesia, pseudoreminiscences (false memories), emotional disorders, phobias.

The corpus callosum connects the cerebral hemispheres. In the anterior part of this large confluent of the brain, that is, in the knee, commissural fibers pass that connect the frontal lobes; in the middle section - fibers connecting both parietal and temporal lobes; in the back part - fibers connecting the occipital lobes.

Symptoms of damage to the corpus callosum depend on the localization of the pathological process. In particular, in the presence of a *lesion in the anterior parts of the corpus callosum,* mental disorders (frontal psychosis) and fronto-callosis syndrome come to the fore . The latter is accompanied by akinesia, amimia, spontaneity, astasia-abasia, memory impairment, and decreased self-criticism. Patients have apraxia, reflexes of oral automatism, and grasping reflexes. *Damage to the connections between the parietal lobes* leads to the occurrence of violations of the body scheme, apraxia in the left hand; for *damage to the fibers connecting the temporal lobes of the brain,* amnesia, pseudoreminiscences, as well as psychoillusory disorders (already seen syndrome) are characteristic. *A pathological focus in the posterior parts of the corpus callosum* causes the development of optical agnosia. As a result of damage to the corpus callosum, pseudobulbar disorders are often observed.

Symptoms of damage to the cerebral hemispheres

Three groups of symptoms are characteristic *of a right hemisphere lesion* : a violation of the body scheme, changes in mental activity, parakinesia , or automated gesturing. Violations of the body scheme are manifested in the form of autotopoagnosia, pseudopolymelia, as well as anosognosia. A variety of mental disorders are observed, which are classified as a psychosyndrome of the right hemisphere: euphoria, reduced criticism of one's own condition, memory disorders, pseudoreminiscences, and confabulation. Acute damage to the right hemisphere (in case of a stroke) is accompanied by parakinesis, or automated gesticulation (unconscious movements of "healthy" limbs). Aphasia, agraphia, alexia, acalculia, and sometimes apraxia develop as a result of *damage to the left hemisphere of the cerebrum* .

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Electronic information resources

Clinical guidelines in neurology. (Order of the Ministry of Health of Ukraine No. 487 dated August 17, 2007)

https://zakon.rada.gov.ua/rada/show/v0487282-07#Text

Lecture No. 3

Topic: Vascular diseases of the brain and spinal cord

Actuality of theme. Justification of the topic. Vascular diseases of the brain make up from 30 to 50% of diseases of the cardiovascular system. According to the WHO, mortality from strokes is 12-15% of total mortality, that is, it ranks 2nd-3rd after heart diseases and malignant tumors, only 18-20% return to work. The issues of prevention and treatment of vascular diseases of the nervous system are of great medical and social importance.

Entire lectures

Educational:

To acquaint students with the etiology and pathogenesis of HPMK (1st degree of abstraction)

During the lecture, provide knowledge of the classification of HPMK, the main clinical symptoms of strokes (2nd level of abstraction)

On the basis of the lecture material, provide an opportunity to master the skills of diagnosis and treatment of hemorrhagic and ischemic strokes and transient disorders of cerebral circulation (3rd degree of abstraction)

Educational:

Aimed at the development of a professionally significant personality substructure;

Education of modern professional thinking;

Ensuring the assimilation of the leading value of domestic clinical, scientific and pedagogical schools.

Plan and organizational structure of the lecture

No	The main stages of the lecture and their content	Goals in levels of abstraction	Type of lecture, methods and means of activation of acquirers, equipment	Time allocation

I 1. 2.	Preparatory stage Setting an educational goal Providing positive motivation	 I I	According to the publication "Methodical recommendations for planning, preparation and analysis of a lecture"	5% (5 min)
II 3.	The main stage Presentation of lecture material according to the plan: 1. Actuality of theme 2. Definition 3. Classification 4. Etiology and main links of pathogenesis 5. Clinical picture 6. Diagnostics 7. Leading syndromes and differential diagnosis 8. Evaluation of the severity of the course 9. Treatment 10. Prevention	 II II II II II II II II II II	 Slide presentation of lecture material Extracts from the medical histories of patients. Excerpts from clinical protocols of the Ministry of Health of Ukraine on providing medical care to patients.	85% (75 min)
III 4. 5. 6.	The final stage Summary of the lecture, general conclusions Answers to possible questions Tasks for independent preparation	 III III III	List of references, questions, assignments	10% (10 min)

Content of the lecture material:

According to the current International Classification of Diseases of the Tenth Revision, vascular diseases of the brain are classified not in Class VI "Diseases of the Nervous System", but in Class IX "Diseases of the Circulatory System", and are considered in headings 1.60-1.69, which are included in the block "Injuries of Vascular Diseases" the brain does not exist in MKH-10, they are a syndrome of cardiovascular diseases. This means that acute disorders of cerebral blood circulation (CBI) can occur in the event of dysfunction of various links of the complex circulatory system: the heart, which performs the role of a pump that ensures the rhythmic flow of blood into the vessels; endothelium of blood vessels and vascular content, which is understood as the amount, composition and properties of blood. Therefore, a cerebral stroke is not a local process with damage to only the vessels of the brain, but a systemic vascular pathology.

Classification of vascular diseases of the brain
Diseases and pathological conditions leading to stroke

II. The main clinical forms of stroke.
A. Initial phenomena of insufficiency of blood supply to the brain
B. Stroke
1. Transient violations of the MK
a) transient ischemic attacks
b) cerebral hypertensive crises

2. Acute hypertensive encephalopathy

3. Membranous hemorrhages
a) subarachnoid
b) Epi - and subdural

4. Hemorrhage in the brain
a) parenchymatous
b) parenchymatous-subarachnoid
c) ventricular

5. Brain infarction (not embolic)
a) in the case of pathology of the main arteries of the head
b) in case of pathology of intracerebral vessels
c) other etiology

6. Brain infarction (embolic)
a) cardiogenic
b) other etiology
B. Impairment of cerebral blood circulation is slowly progressive

1. Dyscirculatory encephalopathy
D. The nature of cerebral blood circulation disorder is not defined
D. Consequences of a previously suffered cerebral stroke

Sh. Localization of the lesion
1. Hemispheres of the brain
2. Brain stem
3. Ventricles of the brain
4. Multiple foci
5. Localization is not defined

IV. Character and localization of vascular changes
A. Nature of vascular pathology
B. Localization of pathology

V. Characteristics of clinical syndromes

VI. State of working capacity

Brain strokes

Hemorrhagic strokes can be arterial or venous, occur by rupture of blood vessels and by diapedesis. By localization, parenchymal, ventricular, subarachnoid, epidural and subdural hemorrhages are distinguished. Combined and combined hemorrhagic strokes (parenchymal-subarachnoid, parenchymal-ventricular, multiple, etc.) are common.

Parenchymal hemorrhage

Cerebral hemorrhage is characterized by a history of hypertension, hemorrhagic diatheses, post-traumatic and congenital aneurysms, septic endocarditis, alcoholism; rapid development of clinical symptoms against the background of acute psychoemotional stress or physical overload, nowadays; significant increase in blood pressure; rare tense pulse; significant hyperthermia (especially when blood breaks into the ventricles of the brain), pronounced violation of vegetative and vital functions (cardiac activity and breathing); very strong headache; vomiting (sometimes "coffee grounds"); meningeal symptoms, floating movements of the eyeballs; hormetonia;

convulsive attacks; hyperglycemia; facial hyperemia; congestive discs of the optic nerves, dislocation of the middle structures according to ECHO-EG by 4-8 mm or more, blood in the cerebrospinal fluid; unconscious comatose state; cerebral edema clinic. Complications such as bedsores, pulmonary edema, thromboembolism of the pulmonary artery, etc. occur early. A fairly frequent complication of hemorrhage into the brain substance is the breakthrough of blood into the ventricles, which are accompanied by a sharp deterioration of the patient's condition, hyperthermia, (40-41 ^{0} C) breathing, deepening of other vegetative disorders, the development of hormonal syndrome, which is manifested by a constant change in the tone of the limbs, when they are hypertensive states with a sharp dissolution pass into a hypertensive state. Paroxysmal increase in muscle tone, especially if it prevails in the extensors, is similar to defibrillation rigidity, which is observed not only in the case of blood breakthrough in the ventricle meningeal symptoms of leukocytosis in peripheral blood, smooth or pendulum-like movements of the eyeballs also appear. With a favorable course of the disease, patients gradually come out of a comatose state, which turns into a soporose state. Consciousness is gradually restored, corneal and tendon reflexes appear. The patient begins to swallow. General brain symptoms gradually regress, and focal symptoms come to the fore. The restoration of movements begins gradually, first in the proximal parts of the limbs, in the leg, and then in the hand. Hemiplegia turns into deep hemiparesis. Muscle tone of paralyzed limbs is restored. In parallel with the restoration of movements, there is a restoration of sensitivity.

Diagnostics

An incompletely closed eye, slow lowering of the raised eyelid, smoothed nasolabial fold, displacement of the corner of the mouth to the healthy side, symptom of "sail" on the side of paresis of facial muscles, external rotation of the paralyzed foot, symptom of dropping hip, atony of paralyzed muscles helps to determine the side of the paralysis. "muscles - because of which the raised hand on the side of the paralysis falls faster ("falls like a whip"), the absence or significantly low expression of Kernig's symptom on the paralyzed side, weak expression of the mimic pain reaction when the hypothesized and immobile half of the body is irritated, unilateral pathological reflexes.

The most informative research methods are computer and magnetic resonance imaging, general analyzes of urine and blood, biochemical blood tests (glucose, residual nitrogen, urea nitrogen, creatinine, coagulogram, hematocrit, blood serum osmolality, indicators of acid-base status, minerals (potassium , calcium, sodium), lipids, protein fractions, samples reflecting the functional state of the liver). Often, the question of differential diagnosis is clarified or determined after a lumbar puncture. To raise the question about the need for neurosurgical intervention, contrast methods of research (angiography, etc.) are also used.

Treatment of patients with acute cerebrovascular accident (ACC): pre-hospital stage, intensive care unit, neurological department, rehabilitation department, rehabilitation in sanatorium conditions and further dispensary observation with a continuous program of pharmacotherapy in conditions of secondary prevention and continuation of rehabilitation.

The pre-hospital stage includes the provision of medical care until the moment of urgent hospitalization of patients with HPMK in stroke, neurological or neurosurgical departments, assistance should be provided to patients in the first minutes, hours after the onset of stroke and is provided by specialized neurological teams, linear ambulance teams, neurologists, therapists, general practitioners (family doctors).

The following standard is generally accepted for the organization of providing medical care to patients with HPMK at the pre-hospital stage.

1. Assessment of the condition of the respiratory tract, breathing, blood circulation
2. Restoring the patency of the respiratory tract, elimination of tongue depression. If necessary, tracheal intubation.
3. Inhalation of humidification of oxygen
4. Artificial lung ventilation (as indicated)
5. Puncture of a vein, installation of a catheter for intravenous infusions.
6. Determination of the level of glucose in the blood by an express method.

After providing primary medical care, the patient is urgently hospitalized in a neurological or stroke (if available) department. This standard should take up to 30 minutes to complete. Arterial pressure at this stage should not be reduced below 200/120 mm Hg, and if it is higher, it should be reduced by no more than 15%-20% labetolol 5-20 mg bolus or IV drip constantly 2-5 mg per minute; propronolol 1-5 mg, bolus IV, metoprolol 5-10 mg bolus IV, esmolol 200-300 mg. In the absence of adrenoblockers, magnesium sulfate 25% - 10.0 - 20.0 ml IV, captopril 6-12.5 mg per os, clofelin 0.15-0.075 mg per os should be used. In case of arterial hypertension, vasopressor drugs should be administered - dopamine 5-20 mcg/kg/min IV drip, or (i) adrenaline 1-5 mcg/kg/min IV drip on the background of infusion therapy. In the presence of a convulsive syndrome, intravenous administration of benzodiazepines (sibazone, midazolam) is indicated. To prevent cerebral edema, the compression of the neck veins should be reduced, the head position should be raised by 20^{0}-30^{0}.

At the hospital stage , the patient should undergo computer tomography of the brain, ECG, if necessary - echocardiogram, blood analysis - hemoglobin, hematocrit, erythrocytes, leukocytes, platelets, blood glucose, plasma electrolytes, blood coagulation parameters, biochemical tests - urea, transaminases, creatinine Duplex scanning of the main vessels of the head (for 1-3 days) in ischemic stroke. If necessary, make an X-ray of the chest organs.

Correction of respiratory disorders is carried out (tracheal intubation, inhalation of an oxygen-air mixture, early activation of the patient, respiratory gymnastics, APC). Measures to stabilize the function of the cardiovascular system and blood pressure continue, for which the same drugs as at the pre-hospital stage continue to be administered. Normalize the water-electrolyte balance. In the presence of cerebral edema, 300-350 ml of fluid is injected; in case of diarrhea, vomiting, hyperhidrosis and high temperature - the amount of liquid is increased. Monitor the blood glucose level: if it rises above 10 mmol/l, insulin should be administered, if it falls below 2.8 mmol/l, a 10% glucose solution should be administered. Body temperature is constantly monitored. When it increases, paracetamol 500-1000 mg, analgin 50% - 2.0-4.0 ml IV or IV, diphenhydramine 1% - 1.0-2.0 ml are administered. If these measures do not help and the temperature is maintained, then antibiotics are prescribed. Control of dysphagia is carried out (probing in the presence of bulbar phenomena, fight against meteorism, laxatives, hypertonic enemas are given) Prevention of complications is carried out - urinary tract infections (catheterization according to the indication, general urinalysis and bacteriological examination of urine, antibiotics are prescribed if necessary), bedsores, thrombosis deep veins (compression stockings, fraxiparin, fragmin 7-10 days, warfarin per os). They monitor the improvement of the function of the gastrointestinal tract (metoclopramide).

Antiplatelet agents: Acetylsalicylic acid 100-300 mg (per day), Clopidogrel (Plavix) 75-300 mg (per day).

Anticoagulants: Fraxipalin (nodraparin) 0.3-0.6 ml once a day subcutaneously in the fold of the anterior-lateral abdominal wall. Warfarin, phenylin (anticoagulants of indirect action) are prescribed a day before the withdrawal of fraxiparin. Drugs that intensify venous outflow from the cranial cavity. Troxevasin 5.0 ml - 10% intravenous solution, or 1-2 g per day for the first 5-7 days of the disease, and then 2 capsules. 2-3 g per day for 2-3 months.

Hemodilution involves the goal of correcting blood viscosity under the control of hematocrit, which should normally be 30-35%. It is best to combine it with means that normalize or correct the functional activity of the myocardium and blood pressure. For hemodilution,

reopoliglukin or rheomecrodex is used in a dose of 150.0 ml to 400.0 ml IV drip 2 times a day, depending on blood pressure. Contraindications to hemodilution are severe heart and kidney failure, sudden heart enlargement, unstable angina, hemorrhagic transformation brain infarction. **Counter-part** . On the first day, 20,000-30,000 IU per 300-500 ml of an isotonic sodium chloride solution are prescribed intravenously as a drip over a period of 90-120 minutes. In the next 5-10 days, intravenous drip of 10,000 units of 2 g per day in 200-300 ml of isotonic sodium chloride solution. During treatment with kontrikal, amylase content in blood and urine, as well as trypsin in blood, which should not exceed normal values, are checked.

Hordox. It is prescribed on the first day intravenously in a dose of 500,000 units, and then 100,000-300,000 units every 2-3 hours.

Constant laboratory control of blood coagulation indicators is carried out. To prevent the development of angiospasm in case of subarachnoid hemorrhage, Nimotop (an active blocker of L-type calcium channels and transmembrane calcium influx) 2 pills (one pill contains 30 mg of Nimotop) every 6 hours for 21 days; or nemotan (nimodipine) 2 pills every 4 hours for 14-21 days. From the first day, it is necessary to carry out passive rehabilitation to reduce the risk of developing countertactures, joint pain, bedsores, pneumonia, deep vein thrombosis and pulmonary artery thromboembolism. Passive rehabilitation includes massage of large muscle groups and passive movements in all joints of the limbs. Procedures are carried out daily after washing and treating the patient's skin with moisturizing and, if necessary, antiseptic creams.

Violation of spinal blood circulation

Etiological factors:

A) ***Pathology of the aorta:*** Atherosclerosis of the aorta, which is characterized by the development of symptoms of insufficiency of blood supply to the lower extremities (Lehrish syndrome, intermittent claudication, ischemic neuritis of the sciatic nerve).

Coarctation of the aorta: a symptom of arterial cerebral hypertension, segmental diapedesis hemorrhages (C_{I} - C_{iv}), sometimes Brown-Sécart syndrome, which is associated with ischemic phenomena in the spinal cord below the level of stenosis.

B) ***Anomaly of spinal vessels (aneurysms, varicose veins)***

Symptoms: sharp radicular pain, focal spinal symptoms that vary depending on the level of damage. The course with remissions and exacerbations is progressive.

B) ***Osteochondrosis of the intervertebral discs.***

Acute ischemic disorders of blood circulation most often occur in the lower parts of the spinal cord, less often in the cervical region. Provocative factors are light trauma, physical overexertion, sharp movements, alcohol consumption, cooling. Occurs stroke-like over the course of one hour to one day, sometimes at night during sleep. The clinical course depends on the level of damage. Movement disorders are combined with sensitive ones (segmental in the area of damage, lower - conductor type). Pathogenetic links with compression and irritation of the anterior spinal or radiculomedullary artery (most often Adamkevich's artery), degenerative - with a changed disc or osteophyte.

Ischemia can proceed according to the type of transient blood circulation disorders or according to the type of heart attacks. In the acute period (day 3-5), the temperature and SRH may increase with normal leukocytosis. With mild degrees of damage, the regression of symptoms begins after a week or a little later (flabby paralysis becomes spastic, the level and degree of sensitive disorders decreases, the function of sphincters is restored). In the case of widespread heart attacks, the end is usually fatal: in the immediate period - as a result of joining heart disorders and respiratory disorders, in the distant period - from pneumonia, urogenital infection, intoxication due to bedsores and sepsis.

Slowly progressive spinal ischemia (discicular ischemic myelopathy, or cervical discogenic myelopathy)

Etiology and pathogenesis: compression factor - blood vessels are more likely to be compressed than brain tissue; secondary adhesion membrane process at the level of the disc and beyond; changes in the vessels of the spinal cord as a result of proliferation of the intima and

adventitia with subsequent secondary disruption of perimedullary and collateral blood circulation. It most often occurs in men at the age of 40-60. Provocative factors are injuries, hard physical work, sudden movements, intercurrent infections and surgical interventions. It is often localized in the cervical, less often in the lumbar region of the spinal cord. The course is often long, stable on the prosthesis for 5-10 years, sometimes progressive.

Main syndromes **:** amyotrophic (pronounced muscle atrophy of the proximal parts of the limbs, sensitive disorders); spastic-atrophic (sweetness and atrophy of one of the limbs with a transition to the opposite side with a gradual increase in atrophy in the hands and spasticity in the legs, sometimes with bulbar syndrome, sensory disorders are weak, segmental type, often - radicular pain, in the late stage - sphincter disorders) ; spastic (begins with numbness in the hands and feet, pain in the joints followed by the development of spastic tetraparesis, sensitive disorders increase in the caudal direction, in the later stages – pelvic disorders)

All forms of myelopathies are characterized by the predominance of motor disorders over sensitive ones.

There are no parallels between the intensity of the ischemic process and the degree of dystrophic changes in the spine. These conditions should be differentiated from other sciatic diseases of the spinal cord, tumors, amyotrophic lateral sclerosis, syringomyelitis, myelitis, multiple sclerosis, hereditary diseases.

Hemorrhagic disorders of spinal circulation :

Hematomyelia (Brown-Sécar syndrome, Minor's syringomyelia syndrome, anterior horn syndrome)

Hematorachis, most often occurs at the dissection of an arterio-venous aneurysm, injuries of the spine. A severe painful radicular syndrome with irradiation in all directions is observed. Occurs suddenly. Often there is a sharp scapular pain along the spine, headache, nausea, vomiting, light stupor, lethargy, lethargy. Appears: Kernig's symptom, often in combination with Laseg's pain symptom; the rigidity of the occipital muscles recedes into the background. Focal spinal symptoms can appear at any time of compression of the spinal cord of varying severity.

Epidural hematoma most often occurs during the dissection of a vascular-spinal malformation. At the same time, there is a sharp local pain in the spine, accompanied by symptoms of spinal cord compression.

Treatment: those that eliminate swelling of the spinal cord (mannitol, furosemide, ethacrynic acid, glycerin), normalize heart activity and blood pressure, improve microcirculation and metabolism of brain tissue (reopoliglukin, pentoxifylline), prevent thromboembolism (heparin). Surgical intervention is indicated for compression-vascular spinal disorders and unsuccessful conservative treatment. Epidural hematomas require surgical treatment. Treatment measures for patients with aortic lesions are determined jointly with surgeons. The cause of chronic cerebral blood flow disorders is cerebral atherosclerosis, hypertension, diabetes, cervical osteochondrosis, arterial hypotension, rheumatism, etc. On the ECG, sinus arrhythmia, violation of the depolarization phase, displacement of the ST segment and T wave are often present.

Changes in biochemical indicators and an increase in the content of cholesterol, triglycerides, and lipoproteins. Clinical (permanent) variant (variant course of PPNMK).

Subjective signs of insufficient blood supply to the brain appear - headache, dizziness, noise in the head, memory disorders, reduced mental capacity, etc. These complaints intensify during mental stress, especially in conditions of hypoxia or overfatigue, poor sleep. After rest, the patient's condition improves or completely normalizes. Objectively, subcortical reflexes, abdominal asymmetry, revival of tendon and periosteal reflexes can be observed. In patients, signs of general vascular disease are found: coronary cardiosclerosis, hypertrophy of the left ventricle of the heart, changes in the vessels of the fundus (angiopathy), symptoms of atherosclerotic damage to other areas.

Slowly progressive disorders of blood supply to the brain - dyscirculatory encephalopathy . There may be dyscirculatory encephalopathy with a predominant lesion of the blood supply in the VBB or in the carotid system. The following phenomenological options are distinguished: with hypothalamic crises, syncopal states, transient disorders of cerebral blood supply, permanent or acute psychotic disorders with a predominance of intellectual-mnestic or emotional disorders. Compensated, subcompensated, decompensated dyscirculatory encephalopathy of atherosclerotic, hypertensive, spondylogenic origin is very often the background against which strokes develop.

Syndrome of damage to various vascular basins.

Damage to the middle cerebral artery, developing hemiplegia, hamianesthesia, hemianopsia, visual paresis, aphasia in the lateral focus.

The syndrome of damage to the anterior cerebral artery is manifested by the development of paresis of the leg opposite to the focus (or hemiparesis with a predominance of damage to the leg), apraxia, the occurrence of subcortical reflexes and changes in the psyche, characteristic of damage to the frontal lobe.

Syndrome of damage to the posterior cerebral artery. With cells in the basin of the posterior cerebral artery, the main diagnostic value is visual disorders - visual agnosia, hemianopsia with preservation of macular vision, square hemianopsia. A thalamic syndrome with hyperpathy, paroxysmal pain, sensitivity disorders, etc. may develop.

The syndrome of damage to the basilar artery is variable due to the peculiarities of its anatomical structure and the level of blockage of the vessel. Cells of softening capture the bridge. The initial symptoms of arterial thrombosis are malaise, nausea, dizziness, pain in the back of the head, tinnitus and headache. There may be oculomotor disorders, double vision, visual paresis, swallowing disorders. These symptoms are joined by limb paresis. With the acute development of the syndrome, general brain symptoms, deep coma, tetraplegia, hormetonia are expressed.

The syndrome of damage to the vertebral artery is characterized by short-term loss of consciousness, dizziness, vomiting, autonomic disorders, nystagmus. It is often combined with impaired blood circulation in the posterior lower cerebellar artery. At the same time, Wallenbern-Zakharchenko syndrome develops.

Syndrome of damage to the internal carotid artery . This type of pathology is characterized by remitting symptoms of ischemia in the branches of the carotid artery, the development of hemiplegia and aphasia (with a focus in the left hemisphere). Ophthalmoplegic syndrome is often present on the side of the blocked artery - reduced vision, up to blindness, on the opposite side - hemiplegia. Bernard-Horner syndrome may also occur on the affected side. Asymmetry of the pulsation of the carotid arteries on the neck (increased pulsation of vessels proximal to the blockage) and increased pulsation and increased pressure in the superficial carotid artery on the side of the cell are observed. Treatment of patients with PPNKM is of particular importance, since neurological disorders at this stage of the disease are reversible, and therefore timely therapy is simultaneously a prevention of acute disorders of cerebral circulation. A system of step-by-step treatment has been developed: polyclinic - hospital - resort - polyclinic, which includes dispensary supervision.

Prevention.

Primary - a system of measures aimed at preventing the development of vascular disease of the brain: elimination of risk factors, improvement of working and living conditions, healthy lifestyle.

Secondary - involves early identification and registration of patients with PPNKM, their timely treatment in order to prevent the progression of cerebrovascular insufficiency. It is necessary to use the possibilities of day hospitals, as well as non-drug methods of therapy. A large role in the prevention of PPNKM is played by medical examination of patients, planned neurological examinations.

Materials for student activation during lectures

1. What are transient disorders of cerebral circulation? Pathogenesis?

2. The patient developed motor aphasia and right-sided spastic hemiparesis. Where is the lesion?

3. The patient had a hemorrhage in the left leg of the brain. What symptoms are observed in this case?

4. The patient suddenly felt an unbearable headache, in a few minutes a large epileptic attack occurred, after which the patient lost consciousness. Meningeal symptoms were detected. What happened to the patient?

General material and methodological support of the lecture:

Educational premises.
lecture room
Equipment.
desks
chairs
blackboard, chalk

Equipment

Stand "Basic neurological tools"
Electrified model "Conducting pathways of the brain and spinal cord"
Electrified model "Localization of functions in the cerebral cortex"
Epidiascope, slides
Neurological tools for patient examination

Illustrative materials

Multimedia presentation "Blood supply of the brain and spinal cord"
Thematic patients

Literature

1. Neurology: study guide / [I.A. Hryhorova, L.I. Sokolova, R.D. Gerasymchuk, A.S. Son, etc.] edited by I.A. Grigorova, L. I. Sokolova - 3rd edition - Kyiv, Medical University "Medicine", 2020 - 640 p.

2. Topical diagnosis of nervous system pathology. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.

3. Methods of examination of a neurological patient: teaching. manual / edited by L. I. Sokolova, T. I. Ilyash. - 2nd edition. - Kyiv: Medicine, 2020. - 143 p.

4. Emergency medicine. Emergency medical care: textbook / I.S. Zozulya, V.I. Bobrova, H.G. Roschyn and others / edited by I.S. Cuckoos - 3rd edition, trans. and additional - Kyiv. - VSV "Medicine", 2017. - 960 p.

5. Negrych T.I., Bozhenko N.L., Matvienko Yu.Sh. Ischemic stroke: secondary inpatient care: education. manual Lviv: LNMU named after Danylo Halytskyi, 2019. – 160 p.

Additional literature

1. Bozhenko M.I., Negrych T.I., Bozhenko N.L., Negrych N.O. Headache. Study guide.- K.: Medknyga Publishing House, 2019. - 48 p.

2. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 1 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2020. - 258 p.

3. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 778 p.

4. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 642 p.

Electronic information resources

Clinical guidelines in neurology. (Order of the Ministry of Health of Ukraine No. 487 dated 17.08.2007) https://zakon.rada.gov.ua/rada/show/v0487282-07#Text

Lecture No. 4

Topic : Epilepsy and non-epileptic paroxysmal states. Neurological aspects of brain injury .

Actuality of theme. Justification of the topic. Late recognition of epilepsy and paroxysmal conditions causes disability, leads to a chronic course of the disease. Timely diagnosis makes it possible to successfully treat such diseases, to implement effective preventive measures.

Entire lectures

Educational:

To acquaint students with the social aspects of epilepsy, its frequency and adaptive capabilities of the body to this pathology (1st degree of abstraction)

Based on the lecture material , to know the classification of epilepsy and non-epileptic syndromes, etiology, pathogenesis, clinic of epileptic seizures, types of auras (2nd degree of abstraction)

To have the skills to diagnose loss of consciousness, tonic and clonic seizures, status epilepticus and to provide emergency aid for it. To be able to carry out differential diagnosis between epileptic and hysterical attacks (3rd degree of abstraction)

Educational:

Aimed at the development of a professionally significant personality substructure

Education of modern professional thinking

Ensuring the assimilation of the leading importance of domestic clinical, scientific and pedagogical schools, including the Odesa Neurological School, in the development of lecture problems

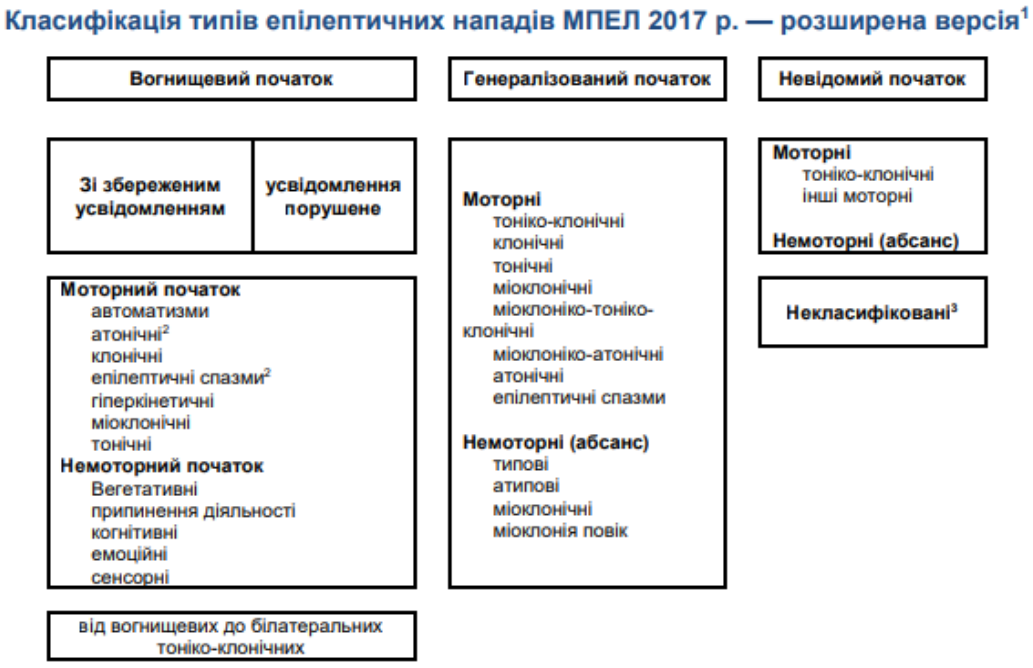
Acquisition of deontology and medical ethics skills by students

Plan and organizational structure of the lecture

No	The main stages of the lecture and their content	Goals in levels of abstraction	Type of lecture, methods and means of activation of acquirers, equipment	Time allocation
I 1. 2.	Preparatory stage Setting an educational goal Providing positive motivation	 I I	According to the publication "Methodical recommendations for planning, preparation and analysis of a lecture"	5% (5 min)
II 3.	The main stage			85% (75 min)

	Presentation of lecture material according to the plan: 1. Actuality of theme 2. Definition 3. Classification 4. Etiology and main links of pathogenesis 5. Clinical picture 6. Diagnostics 7. Leading syndromes and differential diagnosis 8. Evaluation of the severity of the course 9. Treatment 10. Prevention	II II II II II II II II II II	Slide presentation of lecture material Extracts from the medical histories of patients. Excerpts from clinical protocols of the Ministry of Health of Ukraine on providing medical care to patients.	
III 4. 5. 6.	The final stage Summary of the lecture, general conclusions Answers to possible questions Tasks for independent preparation	 III III III	List of references, questions, assignments	10% (10 min)

Content of the lecture material:



EEG diagnostics , .

Treatment: anticonvulsants , tranquilizers, dehydration, tissue preparations, hormones, diet .

Epileptic status. Emergency aid.

Epileptic status is a series of convulsive attacks that occur one after the other, without regaining consciousness between them. Without treatment, epistatus is quickly complicated by dehydration, electrolyte disturbances, aspiration pneumonia, and changes in heart rhythm. Treatment in the intensive care unit. Seduxen is slowly administered intravenously - 1-2 ampoules (20 ml) of 40% glucose solution, after which, as a rule, the attacks stop. The effect of seduxen lasts

15-20 minutes. If there is no effect, thiopental or hexenal is administered intravenously: 1 g of the drug is diluted in 10 ml of isotonic sodium chloride solution at the rate of 1 ml per 10 kg of the patient's body weight. In the intensive care unit, these barbiturates are administered intravenously, controlling pulse, pressure, and breathing. At the same time, 5 ml of a 10% solution of thiopental (hexenal) is injected intramuscularly. Sodium oxybutyrate (20% solution) is also used for intravenous anesthesia. In resistant cases, inhalation anesthesia with nitrous oxide and oxygen in a ratio of 2:1 with the use of muscle relaxants is used. It is very important to start therapy in a timely manner: delaying the administration of seduxen for 6 hours reduces the effectiveness of treatment by three times. Patients are given a full range of resuscitation measures (intubation, glucocorticoids, saluretics, cardiac drugs). With cerebral edema and increased intracranial pressure, dexamethasone up to 12 mg (3 ampoules) is administered.

Paroxysmal non-epileptic states. Diagnosis and treatment.
Classification of non-epileptic seizures /paroxysms/

Hypoxic (fainting)
Exchange (spasmophilia)
Psychogenic (neurotic)
Attacks of toxic origin (tetanus, strychnine)
Attacks of metabolic origin (hypoglycemic)
Affective respiratory attacks
Attacks of hypnotic origin (parasomnias) - night terrors,
sleepwalking, nocturnal enuresis.

Raynaud's disease . One of the forms of angiotrophoneurosis characterized by attacks of spastic vasomotor disorders. In classic cases, an attack consists of three phases. In the first phase, pallor and cooling of the fingers are detected, accompanied by intense pain. In the second phase, there is a cyanotic color, the pain intensifies. Then the skin of the fingers becomes bright red, the pain disappears. Adrenolytic and vasodilator agents (dihydroergotamine, redergam, no-shpa), ganglioblockers (pachicarpine), neuroleptics (reserpine, hexonium — 0.1-0.25 g three times a day for 10 days) are used .

Migraine. Paroxysmal pain in one half of the head, accompanied by vomiting. It often occurs in the morning, has a diffuse nature and can last from one to several days. If a migraine attack is accompanied by transient focal disturbances (hemiplegia, ophthalmoplegia, dysphasia, paralysis of the facial nerve), then it is an associated migraine. Internally or parenterally, vasoconstrictors (ergotamine, caffeine, cofetamine), analgesics, tranquilizers (if necessary, intramuscularly seduxen), antihistamines (diphenhydramine, tavegil, diazolin, suprastin). For very severe and prolonged attacks (migrainous status) — dexamethasone (8 mg intravenously).

Facial sympathalgias . Attack-like pain in one half of the face
— unbearable, burning, distending, oppressive. One-sided vegetative disorders are observed (swelling of the face, lacrimation, rhinorrhea, redness of the eyes). *Charlain's syndrome* (neuralgia of the nasopharyngeal node), *Slader's syndrome (neuralgia of the pterygopalatine node) and some others* are distinguished among facial sympathalgias .
Treatment: antihistamines, antidepressants, neuroleptics, ergotamine derivatives (ergotal, regitamine, cofetamine), anticonvulsants (stazepine, tegritol, finlepsin), lubrication of the middle nasal passage with cocaine.

Fainting There are simple fainting (syncopal) and convulsive (convulsive) fainting. Simple fainting most often develops gradually. Dizziness, nausea, darkening of the eyes occur. The patient turns pale, loses consciousness and falls. The skin is pale, the pupils are dilated, the reaction to

light is weakened, breathing is shallow. The pulse is not felt or is sharply slowed down. Pulsation of the main arteries of the head is preserved, blood pressure is reduced. Fainting lasts 30-40 seconds. Convulsive fainting is characterized by tonic convulsions against the background of low muscle tone and manifestations characteristic of simple fainting.

Vagus, vasomotor and cerebral forms of fainting are distinguished. The first form is characterized by bradycardia, asystole, a sharp drop in blood pressure; for the second — a normal pulse, a decrease in blood pressure; for the third — a sharp deterioration of the blood supply to the brain with satisfactory peripheral hemodynamic indicators. Hypersensitivity of the carotid sinus plays an important role in the pathogenesis of fainting.

Treatment. It is necessary to eliminate the factors contributing to the emergence of a state of unconsciousness. In case of arterial hypotension, agents that increase blood pressure are used. In severe cases, the patient is given a horizontal position, which is the main condition for stopping the attack. The body should be freed from tight clothing, and fresh air should be provided. The patient is allowed to inhale ammonia vapors. At caffeine or cordiamine is administered for a long time.

Meniere's syndrome . Against the background of very severe dizziness, tinnitus and hearing loss occur. A pathological process that simultaneously disrupts cochlear and vestibular functions is localized either in the inner ear or in the XIII pair of cranial nerves. Damage to the latter is rarely accompanied by severe dizziness, and the corneal reflex is reduced or absent on the affected side.

Treatment. Antihistamines (pipolfen intravenously 2 ml each, suprastin, diazolin), phenothiazine derivatives (metherazine, torecan), anticonvulsants (difenin, antilepsin, clonazepam, phenobarbital), antidepressants (better than the tricyclic series — amitriptyline, melipramine), tranquilizers (sibazone, seduxen, relanium), vasodilators agents (nicotinic acid, no-shpa), cholinolytics (atropine, belaspon, belataminal), diuretics (furosemide, diacarb). In case of acute attacks, parenterally seduxen, pipolfen, torekan, atropine, anti-nausea agents (Cerukal). Ringing in the ears is well stopped by phenobarbital, finlepsin.

Materials for students' activation during lectures

What is epilepsy?
What does the aura indicate, and what types of auras do you know?
Describe the major attack clinic
What is the reaction of the pupils to light during an epileptic attack? When hysterical?
When does Kozhevnik's epilepsy occur and where is the lesion localized?
Define status epilepticus.
What is the emergency treatment for status epilepticus?

General material and methodical provision of the lecture

Educational premises.
lecture room
Equipment.
desks
chairs
blackboard, chalk
Equipment
Electrified model "Localization of functions in the cerebral cortex"
Epidiascope, slides
Neurological tools for patient examination
Illustrative materials
A set of educational electroencephalograms
Thematic patients

List of main questions

Modern classification of epilepsy
Idiopathic and symptomatic epilepsy. Etiology, pathogenesis
Types of auras, their topic.
Clinical course of epilepsies
Kozhevnikov's epilepsy. Etiology, clinic, treatment
Epileptic status. Emergency care for him.
Non-epileptic paroxysmal conditions. Diagnosis and treatment.

literature

1. Neurology: study guide / [I.A. Hryhorova, L.I. Sokolova, R.D. Gerasymchuk, A.S. Son, etc.] edited by I.A. Grigorova, L. I. Sokolova - 3rd edition - Kyiv, Medical University "Medicine", 2020 - 640 p.

2. Topical diagnosis of nervous system pathology. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.

3. Methods of examination of a neurological patient: teaching. manual / edited by L. I. Sokolova, T. I. Ilyash. - 2nd edition. - Kyiv: Medicine, 2020. - 143 p.

4. Emergency medicine. Emergency medical care: textbook / I.S. Zozulya, V.I. Bobrova, H.G. Roschyn and others / edited by I.S. Cuckoos - 3rd edition, trans. and additional - Kyiv. - VSV "Medicine", 2017. - 960 p.

5. Negrych T.I., Bozhenko N.L., Matvienko Yu.Sh. Ischemic stroke: secondary inpatient care: training. manual Lviv: LNMU named after Danylo Halytskyi, 2019. – 160 p.

Additional literature

1. Bozhenko M.I., Negrych T.I., Bozhenko N.L., Negrych N.O. Headache. Study guide.- K.: Medknyga Publishing House, 2019. - 48 p.

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3. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 778 p.

4. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 642 p.

5. Medical Books On-line Library (Neurology) – free download
http://medbookshelf.info/category/neurology/

Electronic information resources

Clinical guidelines in neurology. (Order of the Ministry of Health of Ukraine No. 487 dated August 17, 2007)
https://zakon.rada.gov.ua/rada/show/v0487282-07#Text

Lecture No. 5

Topic. Demyelinating diseases of the nervous system

Actuality of theme. Justification of the topic:

Multiple sclerosis is a severe organic damage to the central nervous system, which belongs to the demyelinating pathology and affects the young, able-bodied part of the population. The unwavering progression of the disease, the course with and without remissions and exacerbations

leads to permanent disability due to deep paralysis, ataxia, premature death if intercurrent diseases, deep general trophic disorders and damage to the vital centers of the brain stem are added .

Entire lectures:

Educational:

To acquaint students with the epidemiology of multiple sclerosis, its pathogenesis and pathomorphology as multiple sclerosis, tka and other demyelinating diseases of the nervous system (1st degree of abstraction)

From the clinic of multiple sclerosis, diffuse encephalomyelitis, subacute sclerosing panencephalitis (2nd degree of abstraction)

To have the skills to make a diagnosis of demyelinating diseases n. p., prescribe adequate research methods for this pathology, prescribe etiopathogenetic treatment (3rd degree of abstraction)

Educational:

Aimed at the development of a professionally significant personality substructure;

Education of modern professional thinking;

Ensuring the assimilation of the leading importance of domestic clinical, scientific and pedagogical schools, including the Odesa Neurological School in the development of lecture problems

Learning the skills of deontology and medical ethics;

Plan and organizational structure of the lecture

No	The main stages of the lecture and their content	Goals in levels of abstraction	Type of lecture, methods and means of activation of acquirers, equipment	Time allocation
I 1. 2.	Preparatory stage Setting an educational goal Providing positive motivation	 I I	According to the publication "Methodical recommendations for planning, preparation and analysis of a lecture"	5% (5 min)
II 3.	The main stage Presentation of lecture material according to the plan: 1. Actuality of theme 2. Definition 3. Classification 4. Etiology and main links of pathogenesis 5. Clinical picture 6. Diagnostics 7. Leading syndromes and differential diagnosis 8. Evaluation of the severity of the course 9. Treatment 10. Prevention	 II II II II II II II II II II	 Slide presentation of lecture material Extracts from the medical histories of patients. Excerpts from clinical protocols of the Ministry of Health of Ukraine on providing medical care to patients.	85% (75 min)

III 4. 5. 6.	The final stage Summary of the lecture, general conclusions Answers to possible questions Tasks for independent preparation	 III III III	List of references, questions, assignments	10% (10 min)

Content of the lecture material:

The leading role in **multiple sclerosis** is now given by infections and immunological disorders. Sometimes a high concentration of antibodies is found in the blood and cerebrospinal fluid of patients with multiple sclerosis. Recently, there has been an assumption about the etiological connection of multiple sclerosis with slow viruses, i.e. viruses that remain in the body for a long time in a latent state, and then under the influence of some adverse factors become active and cause a chronic progressive disease.

Pathogenesis. The disease is considered as an autoimmune process with a long course and the formation of intermediate antigens that arise as a result of the vital activity of the virus in the changed brain tissue. Exogenous and endogenous factors (pregnancy, hypothermia...) play a provocative role in the development of multiple sclerosis. The main pathomorphological changes develop in the white matter and are characterized by the breakdown of the myelin sheaths of the conduction systems in various parts of the brain and spinal cord.

Clinic. Persons aged 20 to 40 are most often affected. The clinical picture is provided by extreme polymorphism and variability of pathological symptoms. The first signs are symptoms of damage to the pyramidal pathways; phylogenetically young skin reflexes (abdominal, plantar , cremasteric) disappear, tendon and periosteal reflexes increase, pathological reflexes of Babinski, Oppenheim, Rossolimo, etc. appear early. In addition, there is a feeling of heaviness in the legs, fatigue during a long walk. Later, gross movement disorders develop, such as spastic paresis and limb paralysis, which can be combined with paresis of the oculomotor muscles (diplopia, ptosis, strabismus). Pathology of the posterior columns of the spinal cord is manifested by disturbances in vibration sensitivity. Disturbances occur on the part of the visual analyzer - a fleeting feeling of fog or cloudiness in front of the eyes, a decrease in vision up to amaurosis, due to retrobulbar neuritis of the optic nerves. Cerebellar disorders are often observed: ataxia, difficulty in performing coordination tests, slurred speech, nystagmus, adiadochokinesis. Disorders of the function of the pelvic organs (retention of urine, imperative urges, urinary incontinence). Two triads are characteristic. Charcot's triad – nystagmus, slurred speech, intention tremor. Marburg's triad - absence of abdominal reflexes, pallor of the temporal halves of the optic nerve discs, lower spastic paralysis. The nature of the process is progressive – remitting.

In the remitting course of the disease, three stages are distinguished: exacerbation - the stage of the appearance of new or recovery of pathological symptoms that were already observed; remission - the stage of regression of these symptoms; steady state stage.

Prognosis - over time, deep disability occurs . Death occurs mainly from intercurrent disease.

Treatment - disease-modifying therapy drugs, corticosteroids.

Acute diffuse encephalomyelitis is an acute infectious-allergic disease of the nervous system that causes diffuse inflammatory foci of various sizes in the brain and spinal cord. The primary one arises as a result of a filtering virus entering the nervous system. Secondary develops against the background of influenza, malaria, tularemia and other acute diseases.

Pathomorphology . Against the background of edema, hyperemia and cellular infiltration of various structures of the nervous system, numerous micro necrosis and demyelination centers are observed . At the same time, sclerotic plaques are not formed.

The clinic is extremely polymorphic. The disease begins acutely with an increase in body temperature to 38-39°C. Malaise, muscle pain, headache, nausea, vomiting appear. Brain damage

is manifested by seizures, loss of consciousness, hemiparesis, hyperkinesis, ataxia, bulbar and peripheral motor disorders, etc. Damage to the spinal cord leads to the development of paraplegia, conductive and segmental sensitivity disorders, dysfunction of the pelvic organs.

Forecast. The disease progresses acutely, often with a severe general condition of the patient. The disease usually ends with recovery with moderately pronounced consequences. Sometimes more pronounced movement and other disorders remain. Remissions are not observed.

Treatment . Anti-inflammatory; antibiotics, salicylates, hexamethylenetetramine, desensitizers (diphenhydramine, pipolfen), corticosteroid hormones, vitamins, proserin, nicotinic acid, aloe, plasmol, physical therapy, exercise therapy, massage.

Amyotrophic lateral sclerosis is a chronic, progressive disease that mainly damages motor pathways. The main patho-anatomical changes are localized in the system of pyramidal tracts. Motor neurons of the medulla oblongata, anterior horns of the spinal cord, neurons of the motor cortex are affected. The fibers of the pyramidal pathways are subject to demyelination. The first signs of the disease are most often associated with damage to the muscles of the limbs, as well as muscles innervated by bulbar cranial nerves (tongue, lips, soft palate, pharynx). The process can begin with the lumbar or cervical thickening of the spinal cord, or with the medulla oblongata. Damage to motoneurons causes the appearance of fibrillar twitching of the muscles of the upper or lower limbs with degenerative muscle atrophy and an increase in their tone. At the same time, pathological pyramidal signs appear. Tendon and periosteal reflexes first increase, and then decrease, that is, mixed-type paralysis appears. Paresis and then paralysis of the muscles innervating the IX, X, XII pairs of cranial nerves are added to motor disorders of the limbs. If the process begins with lumbar thickening of the spinal cord, the course of the disease very soon takes on an ascending character and lasts up to 6-7 years.

In all cases, death occurs from damage to the medulla oblongata in cases of asphyxia, aspiration pneumonia, or cardiac arrest . treatment: riluzole.

Acute sclerosing leukoencephalitis (demyelinating leuko- and panencephalitis)

This group includes peculiar forms of chronic and subacute encephalitis with a progressive, severe course (encephalitis with Dawson's inclusions, subacute sclerosing leukoencephalitis of Van-Bogart, nodular panencephalitis of Pette-Derigna, subacute sclerosing panencephalitis of Tarishka). Since the difference between them in the clinical picture and morphology is not significant, they are treated as one disease, most often under the name *"subacute sclerosing panencephalitis"*. This group of diseases includes Schilder's periaxial encephalitis (diffuse periaxial sclerosis), which has some well-defined clinical and pathomorphological features.

Etiology and pathogenesis.

cowpox , plays a big role here . In the blood and cerebrospinal fluid of the patient, too high blood pressure titers are detected. Autoimmune mechanisms play a role in the pathogenesis of the disease, as well as an acquired or congenital defect of the immune system.

Pathomorphology.

Microscopically, diffuse demyelination and gliosis of the white matter of the cerebral hemispheres is revealed. In a number of cases, there are many glial nodules. In others, oxyphil inclusions are found in the nuclei of neurons of the cortex, subcortex, and brainstem against the background of its dystrophic changes. Axial cylinders initially remain relatively intact, then die. Moderately pronounced perivascular infiltration by lymphoid and plasma cells is noted. Schilder's leukoencephalitis is characterized by proliferation of glia with foci of sclerosis.

Clinical course.

Children and adolescents between the ages of 2 and 15 are mostly affected, but sometimes the disease also occurs in adulthood. The onset of the disease is subacute, imperceptible. Symptoms that are considered neurasthenic appear: absent-mindedness, irritability , fatigue, tearfulness. Then there are signs of personality change, deviations in behavior. Patients become indifferent, lose a sense of distance, friendship, respect, correctness of relationships, discipline. After 2-3 months from the onset of the disease, hyperkinesias in the form of myoclonia, torsional spasm, hemibalism appear in the neurological status. At the same time, convulsive epileptic

seizures, permanent partial seizures occur convulsive attacks of the Kozhevnikov type of epilepsy. Extrapyramidal disorders are associated with pronounced autonomic disorders — facial oiliness, drooling, hyperhidrosis, vasomotor lability, tachycardia, tachypnea. Uncontrollable laughter and crying are often observed. A frequent symptom is static and motor ataxia of frontal origin (the patient does not keep the body in a vertical position) . In the late stage of the disease, spastic mono-, hemi-, and tetraparesis occur, which are associated with extrapyramidal and fronto-cerebellar motor disorders. Sensory and motor aphasia, auditory and visual agnosia are revealed. Cachexia progresses.

Three stages are distinguished in the course of subacute sclerosing encephalitis

In the first stage, the preceding symptoms are personality changes, deviations in behavior, increasing defects of higher brain functions, various hyperkinesis, convulsive and non-convulsive attacks.

In the second stage extrapyramidal disorders of tone and disorders of autonomic central regulation are increasing.

The third stage characterized by cachexia and complete decoction.

Course and prognosis.

The course of sclerosing panencephalitis is relentlessly progressive and always ends fatally. Duration of illness from 6 months. up to 2-3 years. There are forms that run chronically with periodic remissions. Death occurs with complete immobility, cachexia, marasmus, most often with epileptic status, or as a result of pneumonia.

Diagnostics.

There are some difficulties in the early stage, when neurasthenia, hysteria, schizophrenia are often diagnosed.

In the future, a differential diagnosis is carried out with a brain tumor.

The diagnosis should be based on diffuseness, and not on the "unifocality" of the lesion, the absence of intracranial hypertension, the displacement of the middle structures of the brain during EchoEG, the pathognomonic pattern of EEG.

The diagnosis is confirmed by the results of immunological studies and computer tomography. The main method of diagnosis is MRI examination of the brain and spinal cord.

Research methods.

In the cerebrospinal fluid, moderate pleocytosis, increased protein content and gamma-globulin level are detected.

Lange's colloid reaction gives a paralytic curve in subacute sclerosing encephalitis, inflammatory and mixed - in Schilder's leukoencephalitis. Pathological changes in Lange's reaction and hypergammaglobulinorrhagia are early signs of leuko- and panencephalitis.

An increase in the level of oligoclonal cerebrospinal fluid is revealed. Too high (especially in subacute sclerosing encephalitis) titers of measles antibodies are found in blood serum and cerebrospinal fluid.

Periodic stereotypical regular bilaterally synchronous high-amplitude discharges of electrical activity (Rademekker complexes) are registered on the EEG. At EchoEG, which is performed in cases with a pseudotumorous course of leukoencephalitis, displacement of the middle structures is not observed.

The most informative computer axial tomography.

Treatment

Includes pathogenetic and etiotropic therapy, symptomatic drugs, as well as restorative measures.

Leukodystrophies

Leukodystrophies are progressive diseases that pass subacutely and are characterized by diffuse lesions of the white matter of the hemispheres, subcortical nodes and cerebellum with relative preservation of the cerebral cortex.

This group of diseases is best studied biochemically. In recent years, a number of data have been obtained to prove that leukodystrophies are metabolic diseases, like lipidoses, but the

enzymes responsible for their occurrence have not been identified.

This group of diseases includes leukodystrophies of Pelicius-Mertzbacher, Krabbe and Hallervorden-Spatz. The division of leukodystrophies into the specified nosological forms is conditional. Clinically, these diseases are similar, but they begin at different ages.

The clinical picture includes mental and intellectual disorders, as well as gross organic neurological symptoms. At the beginning of the disease, pyramidal disorders are noted, and at the end extrapyramidal rigidity develops. Histologically, destruction of myelin fibers with preservation of axial cylinders, as well as compensatory growth of macro- and microglia are revealed in the brain. A puncture in the cerebrospinal fluid reveals significant pleocytosis and a moderate increase in protein.

Hallervorden-Spatz leukodystrophy is the most late form of leukodystrophy. The disease begins in school years and can last for several years. It is clinically manifested in extrapyramidal hyperkinesis (choreoathetosis), which later changes to muscle stiffness, convulsions, and sometimes epileptic seizures. The disease is transmitted according to the autosomal recessive type. Persons of both sexes are ill. The disease ends with complete immobility of patients and dementia.

During the pathological examination of the brain of the deceased, deposition of iron-containing pigment, degeneration of axons, glial infiltration in the area of the globus pallidus, substantia nigra, subthalamic nucleus, thalamus, and cortex of the cerebrum and cerebellum are revealed. Biochemically, the patients have disorders of pigment and lipid metabolism. Violation of iron and catecholamine metabolism is also important.

Materials for student activation during lectures

- Pathomorphology of multiple sclerosis
- What is the feature of the course of multiple sclerosis?
- What is the Charcot triad?
- What is the Marburg Triad?
- What sensitivity and reflexes disappear first in multiple sclerosis?
- What is the difference between acute diffuse encephalomyelitis and multiple sclerosis?

General material and methodical provision of the lecture

- Educational premises.
  - lecture room
- Equipment.
  - desks
  - chairs
  - blackboard, chalk
- Equipment
  - Electrified model "Conducting pathways of the brain and spinal cord"
  - Multimedia, computer
  - Neurological tools for patient examination
- Illustrative materials
  - Thematic patients
  - Educational MRI-tomograms of the brain and spinal cord
  - Histories of diseases

List of main questions

Epidemiology of multiple sclerosis
Etiology, pathogenesis, pathomorphology of demyelinating diseases
Clinical course, treatment of multiple sclerosis
Etiology, pathogenesis, clinical course, treatment of acute diffuse encephalomyelitis
Subacute sclerosing panencephalitis. Etiology, clinical course.
Leukodystrophies: Etiology, clinical course.

Literature

1. Neurology: study guide / [I.A. Hryhorova, L.I. Sokolova, R.D. Gerasymchuk, A.S. Son, etc.] edited by I.A. Grigorova, L. I. Sokolova - 3rd edition - Kyiv, Medical University "Medicine", 2020 - 640 p.

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