MINISTRY OF HEALTH OF UKRAINE

ODESA NATIONAL MEDICAL UNIVERSITY

Faculty of Medicine

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.

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 Presentation of lecture material according to the plan:		Slide presentation of lecture material	85% (75 min)

	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	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:

Classification of epilepsy, etiology, pathogenesis, clinical picture (the types of attacks), differential treatment of epilepsy. Epileptic status, the first aid. Non - epileptic paroxysmal states (with spasm s-spasmophilia, febrile and toxic convulsions, hysteric paroxysms; without convulsions vegetative paroxysm, syncopes). Differential diagnosis of epilepsy and nonepileptic paroxysmal states.» - 2 hours

According to definition of WHO experts (1975), epilepsy is a chronic disease of the brain of various etiology, which is characterized by repeated epileptic fits appearing as a result of excessive neurotic discharges and accompanied by diverse clinical and paraclinical symptoms.

Variety of epileptic forms had stipulated classification of epileptic fits (1981), epilepsies and epileptic syndromes by International Antiepileptic Leaque (1989).

International classification of epileptic attacks includes;

1. Partial epileptic attacks.
1.1 Common partial attacks (consciousness is not broken).
1.2 Complicated partial attacks (consciousness is broken).
1.3 Partial attacks with secondary generalization.
2. Generalized epileptic attacks.
2.1 Absentia epilepticas.
2.2 Muscular-spastic.
2.3 Clonic.
2.4 Tonic.
2.5 Tonico-clonic.
2.6 Atonic attacks.

International classification of epilepsies and epileptic syndromes includes:

1. Localized-stipulated epilepsies and syndromes,
1.1 Idiopathic (with age-dependent debut).
1.2 Symptomatic;
1.3 Cryptogenetic.
2. Generalized epilepsies and syndromes.
2.1 Idiopathic.
2.2 Cryptogenetic.

2.3 Symptomatic.

3. Epilepsies and syndromes, that have symptoms of restricted (focal) and generalized ones.

3.1 With generalized and focal attacks.

3.2 Without definite symptoms of generalization and focallity.

4. Special syndromes.

4.1 Attacks connected with certain situation.

4.2 Single attacks or epileptic status.

5. Symptomatic epilepsy is a result of known or supposed diseases of the central nervous system. Idiopathic epilepsy - is a disease that is not caused by definite reasons, with the exception of hereditary inclination. Cryptogenetic epilepsy does not correspond both, mentioned above, but most of its types can be regarded to epilepsy of symptomatic one with unknown etiology. The term "cryptogenetic" means diseases of unstated cause.

It should be noted, that in native literature epilepsy is a single disease just as in constrast to international classification the disease is used in plural - epilepsies.

Frequency of epilepsy at total is 5-10 cases per 1000.

Epilepsy's etiologic factors.

Genetic, infection, neuroinfections, craniocerebral trauma (including perinatal one), vascular diseases, brain's tumors, intoxications.

Pathogenesis of epilepsy is not dear. There are a lot of hypothetic theories. The most acknow4eged one is Kryzhanovskey'"s G. N. Theory based on the change of neurons: epileptisation of a neuron, groups of neurons wdth formation of determinative epileptic focus and subsequent brain's epileptisation.

The clinical picture of epilepsy is variable. The main symptoms of the disease:

1. Different types of attacks: the heralds of the attack, auras, partial convulsions or sensitive reactions, tonico- clonic, clonic spasms, tonic, myoclonias, absentia epilepticas. (Look at the classification of attacks).

2. Affective (dysphoric) states.

3. Dusk (crepuscular) disorders of conciou sness,

4. Availability of psychic defect of different stage of manifestation from changing of personality to psychoses.

Practically, most often one ought to deal with patients suffering only from tonico-clonic attacks and absentia epilepticas, considerably less with myoclonic attacks and their derivatives (polymorphic attacks), dysphorias, changes of personality- and psychic disorders.

The course of epilepsy without treatment little by little is progressing. More frequently the disease starts with appearance of rare tonico-clonic spasmodic attacks wdth manifestations of mood changing, feeling of surrounding change meaning "had already seen" or "had never seen", development of various auras (cardial, epigastric, vegetative, vestibular and others) and other sensations, that is difficult to explain to patients. Sometimes everything is over by then (isolated aura), but oftener the secondary generalized spasmodic attack is developed. Epileptic focus in such cases, as a rule is located in temporal portion of the brain that is confirmed on EEG.

The initial generalized spasmodic convulsions are developed without symptoms, but as usually are amnesied by patients, who may recollect about their existence according to indirect symptoms (the feeling of tiredness, bitten tongue, dirty clothes, etc.). Absentia epilepticas is a short term (1-2 minutes) lost of consciousness, as a rule, are not left in the patient's memory (common absentia epileptica) or identified partially (complex absentia epileptica).

Structures and mechanisms, that provide enlargement of epileptic discharge from the place of formation, make up the epileptic system.

The major epileptic attack is characterized not only by the loss of consciousness and vegetative disorders (mydriasis, hyperhdrosis, tachycardia etc.), but also by convulsions involving both halves of the body simultaneously. At first the tonic convulsions appear, then the clonic ones do. The convulsions can be only tonic or only clonic (so-called not organized convulsive fit) in

rarer cases. Apnea arises in connection with involving of respiratpry muscles into the process as a rule. the patients bite the tongue, enuresis is registered if there was urine in the urina bladder at the moment of the fit, me patients frequently receive traumatic damages) while falling. The lit is finished by an epileptic coma transforming into dream, after escaping from it amnesia is registered; the patients experience the feeling of breakeness, pains in muscles, weakness. Psychomotor excitement, dusk state of consciousness and other psychical disorders can be developed after the tit in other cases.

The second group of epileptic fits is partial (focal). The epileptic center is always located in cerebral cortex in partial fits, but subcortical truncal structures are involved into the process: the partial fits are subdivided into simple (without breach of consciousness) and complex (with breach of consciousness). Besides tliat the partial fits with secondary generalization are distinguished. The tits with locomotory. sensitive, vegetative, psychopathologic displays are distinguished among the partial ones.

For the secondary generalized attacks it is necessary also to relate the generalized convulsive fits with preceding aura. An aura (lat. aura "respiration") term meaning pathological sensations of various nature arising in patients at the beginning of the fit (apart from generalized for the second time, it can be registered by partial complex fits).

Usually the aura is stereotyped for one and the same patient by repetition of major epilepsy. Its nature is determined by location of the epileptic focus. The patient can feel, for example, some odor (olfactory aura), taste (gustatory aura), to see iifferent images or whole pictures (visual aura) etc. during an aura.

The following kinds of an aura are distinguished: sensory, motor, sensitive, spoken, vegetative and psychic. The visual and auditory auras are examples of sensory aura. The sensitive aura appears like various senesthopathias (dryness in the mouth, feeling of numbness, distorted perception of the body etc.). The motor a iira is a stereotyped motion done by a patient before the appearance of convulsions. The spoken aura may be motor and sensory. The motor aura is manifested by speech interruption or on the contrary, violent forced pronunciation of the determined words. In sensory spoken aura the patients either do not understand the addressed speech, or hear words, which actually nobody had delivered. There exist other types of auras, for example, various unpleasant sensations in the cardiac region (cardiac aura), organs of the abdomen (abdominal aura), sensation of the stop of breathing or shortage of air, feeling of hunger etc.

The psychical aura is especially original, during which the patient feels an exotic emotional condition, sometimes extreme delight. The example of a similar aura can be obtained from the magnificent description of an attack in prince Myshkin by Dostoevskey F. M. The writer himself suffered from epilepsy and, apparently, experienced the similar sensations. Sometimes aura may be the unique symptom of an epileptic attack.

But always after the aura. there is a loss ot consciousness and the convulsions are developed. The aura always indicates the focal beginning of the attack.

As it was already noted, epilepsy is characterized by paroxysmal manifestations and by unparoxysmal chronic changes of a patients'personaSity. Sluggishness, inertness of psychical processes, pathologic way of thinking are typical for epilepsy. The patients are verbose in talk, howeve, they can not select the main state, the basic thought, give a short answer, stop on minor details.

Excessive punctuality, pedantry, petty offence, intolerance, persistence, and at the same time tear, flattery, timidity, exaggeration, respect, tenderness in treatment, tendency to please the interlocutor (so-called defensivity are developed). Tne circle of interests is gradually narrowed, the memory becomes worse, egocenfricity, the signs of epileptic degradation are discovered.

Many factors are based on these changes: organic affections of the brain, impairment of its normal functional activity under the influence of epileptic discharges, a chronic stress state in connection with recurring fifs stipulated by family and social difficulties, hereditary inclinations and anunfavorable effect of a prolonged antiepileptic therapy.

Besides, chronic changes of the patient's personality and psychical manifestations at the moment of the tit (mnestic, ideational, affective sings), the periodic disorders of psychics as dysphorias or psychoses of different duration frequently appear in epilepsy.

Diagnosis

It is necessary to gather the anamnesis both from the wwds of the patient and his nearest relatives. Indications on the perinatal pathology, early cerebral processes give the grounds to suspect residual affection of tlie brain; and as result of it, indications on presence of the epileptic fits in the relations can be quite often in anamnesis.

The special attention should be paid to paroxysmal episodes in childhood, convulsions in the period of birth, so-called spasmophilia, for which the epileptic tits are taken quite often, to convulsive fits during the feverish states abdominal crisises, paroxysms of short-term pains in th abdomen appearing independently on meal and accompanied by vegetative disorders (paleness-nausea, changes of pulse etc.).

The role of some paroxysmal states, arising during sleep, night fears, myoclonias, tonic spasms etc, which are quite often observed in children is less studied.

Epilepsy is characterised by stereotypy and regularity of the fits, relative independence of origin of the latters from external influence quite often indipendence of the determined time of the day (night attacks "epilepsy of sleep", morning - "epilepsy of awakening", day time - "epilepsy of cheerfulness"), presence of proper changes of character and intellect in patients.

It is necessary to point out the form of epilepsy, nature of the fits, their frequency, peculiarities of their distribution in the awaking-sleep cycle, presence or absence of psychical changes, and other disorders.

The early manifestations of epilepsy have such clinical peculiarities: incomplete, rudimentary- abortive, partial forms of paroxysms, high frequency of dream talkings, muscular trembling. The fits still can have episodic character at this time, they are provoked by different external influences. However gradually, the typcal tor each patient type and rhythm of the fits are being formed. New symptoms are discovered, as the disease aggravating: gradually the fits become more frequent, quite often inclination to serial manifestation or development of the epileptic status and observed. Psychical changes can grow at this time. thus, epilepsy l]^s determined appropriateness of its course, study and analysis which are important lor diagnosis and prognosis of the disease.

Determined changes ofEEG are typical for epilepsy. The similar changes can be discovered in epileptic syndromes, however in these cases they'are combined with changes ofEEG stipulated by the basic process.

Such methods of investigation as KT and MRT permitting to earn[7] out visu-Eiiization of the cerebral structures at various levels and determine even insignificant segments of atrophy of the cerebral tissue and other changes are very important for diagnosis of epilepsy.

The differential diagnosis of the epileptic fits. The epileptic fits in spite of their variety, are characterized by paroxysmality, short time period, stepeotypy of one and the same type of fits for the given patient, in most cases by loss of consciousness. The generalized convulsive fits should be distinguished from demonstrative (hysterical) attacks and from the convulsive farms of syncope. Phasity of paroxysm: the aura (if it is present), loss of consciousness, convulsions (tonic, and clonic), postparoxysmal changes of consciousness (coma, sopor, sleep), are typical for the epileptic generalized convulsive attack, apart from the indicated above peculiarities of the epileptic attacks. It is registered not only extension of pupils, but also loss of their reaction on light, enuresis, increase of arterial pressure, cyanosis. The epileptic fit is developed in any conditions, the patients frequently receive damage by falling. The fit rises in determmed emotional significant for tlie patient situation, in the presence of other people usually in hysteria. One can tell that the hysteric fit is a performance to some degree organized and reckoned on the spectator. The patients are never broken by falling. The patients can bite a lip, cheek, but not a tongue, "the hysteric arch" is possible, the various pretentious postures are registered sometimes, the patients tear clothes on A' themselves, scratch their face, bite etc. Extension of pupils is not usually accompanied by their

areflexia on light. The dusk loss of consiousness is possible after an attack, that, however, is registered in epileptic convulsive paroxysm. Loss of consciousness and relaxation of muscles arise at the beginning of the convulsive forms of syncopes and only after some seconds the" tonic convulsions appear. The fit can be accompanied by the extension of pupils, enuresis. However, the reaction of pupils to light is preserved, paleness of the patient, reduction of arterial pressure are registered in contrast to the epileptic fit. At the same time the arterial pressure is usually increased, in tlie convulsive epileptic fit. The heart may not be listened, and electrical activity is absent on EEG, which is typical for epileptic tits. The akinetic epileptic attack should be distinguished from simple tatique. The attack arises suddenly, a patient loses consciousness and falls. The convulsions are absent, but the postural tension can be lost. Mydriasis, reduction or absence of reaction of the pupils on light are registered. The syncopes develop usually in persons suffering from vegetative dystonia; sensation of laintness, weakness. dimness of vision, tinnitus proceed to a patient's falling and loss of consciousness. If a patient has enough time to take a horizontal posture, the loss of consciousness does not arise frequently, the fit is finished quickly. The signs of the cardiovascular system's activity oppression, reduction of arterial pressure, bad filling of the pulse,, its acceleration or lowering are registered during a syncope. The extension of pupils is not accompanied by their areflexia on light. The complexes of “a peak-wave” of various frequency are registered on EEG in an atonic epileptic fit. If an epileptic fit has appeared in a patient, it is necessary first of all to exclude the current cerebral process. By this apart from typical for one or all to other cerebral diseases' symptoms (swellings, encephalitides etc.) it is necessary to take into account the peculiarities of manifestation and course of a epileptic fits. So, Jackson's attacks are registered seldom in epilepsy and, on the contrary, frequently, they are the first manifestation of tumors ofRolando's areas of large hemispheres of the brain. Dynamics of fits in case of tumor is characterized by a number of peculiarities. The attacks become more limited and less often in the process of appearance and of a hypertension syndrome, and then they disappear absolutely. The epileptic attacks in case of acute disorders of the cerebral blood circulation have, as a rule, convulsive nature, the peculiarities of their manifestation coi-respond to the zone of impairment of the cerebral blood circulation. So, the generalized attacks are registered by discirculation in the vertebrobasilar basin, and Jackson's secondary generalized ones by discirculation in the carotid basin. Then the fits are repeated owing to repeated acute disorders of the cerebral blood circulation, and also to decompensation of blood circulation in the zone of previous -foci.

Treatment

In the history of epilepsy there had been the period when the lack of effective drugs for treatment and prophylaxis of attacks (in foreign literature the term "the control of attacks" is used) led to polypharmacy, that is administration of several remedies for one patient at the same time or excessive usage of drugs.

The beginning of this period can be regarded to the 60-70 years and the first half of the 80th years. At present famous specialists in the field of epilepsy have more often become to persuade to use monotherapy. It *was* proved that the correct chosen antiepileptic epilepsy drug for one or another patient that is monotherapy had been effective in almost 70% of cases, the rest of 30% were treated with two types of drugs and only in especially resistant cases the combination of three or more antiepileptic remedies were needed. Modern methods of epilepsy treatment are first of all oriented for exposing and elimination of attacks' reasons and excluding the factors, provoking them, making up correct diagnosis of the disease's form and type of attacks, choosing of adequate medical preparations for treatment in in-patient and out patient departments, elaboration of generally-hygienic and social measures (recommendations how to choose speciality, getting education, job's conditions, rest and solving the future social problems). If we take into consideration the number of different types of attacks and a great number of antiepileptic drugs, thus their choosing and rational medical therapy may seem almost unrealizable task. That's why holding to some basic principles considerably makes this task easier. The first principle is based on the fact, that every antiepileptic drug possesses to same extent a marked action's selection (choice) as to a certain type of attacks and it is very important that drugs and the type of an attack

would correspond to each other. In connection with it all modern medical preparations can be divided into TWO main groups: the drugs that are the most effective for antispasmodic or insignificant (generalized) attacks (absentias epileptica, myoclonias, atonic attacks .and others) are applied to the first group. Etosuksimidum and Valproat, also Clonazepam and Clobasam are the main preparations of tills group. Oilier remedies can be considered as "reserved " ones. The second group of preparations is effective in partial attacks and repeated generalized attacks. The main drugs are Carbamazepin, Phenitoinum. Valproat and Phenobarbital. Vigabatrin, Clobasam and Acetazolamid may be applied to the second place. Recently a principally new antiepileptic drug-Lamictal (Lamotrizhinum) lias appeared at the world medical market, including the Ukrainian one. It prevents execessive discharge of amino acids, most of all Glutamatum, at the same time not lowering its discharge within ihe limits of a physiological norm. In contrast to all traditional antiepileptic remedies Lamictal effects on all types of paroxusmal disorders in epilepsy of various genesis. The results of approbation within the limits of multicentral tests testify that Lamictal is able considerably improve patient's adaptation to environmental conditions. Another principle is usage in cases where only one type of medical preparation is possible, that is monotherapy. There are some observations, that me evidence of administration of the only one antiepileptic drug in some patient helps to control the attacks better than in case of polypharmacy. It may be connected with lowering of medical toxicity risk and easing of individual choise of dosage as well as the regimen of drug's taking and excluding the factor ofundesirabte medical action. If monotherapy is not effective there should be made an attempt to change a drug by another one. And if in this case the therapy is not effective, there may be the problem to use a combination of medical antiepileptic preparations. Rational antiepileptic polypharmacy possesses its own peculiarities in partial and generalized attacks. Gexamidinum 0,25, Dephemin 0,117, Phenobarbital 0.05- 0,1, Benzonal 0,1, Carbamazepinum 0,2- (Phinlepsinum, Tegretol, Tymonil, Zeptoi, Stazepinum and others), Lamictal 0,1 are indicated in partial attacks. Carbamazepinum is especially indicated for a patient with temporal localization of a focus and an affective unbalanced state. Natrium valproat 150-300 mg (Depakinum, Convulex. Acediprol. Encorat, Orphiril and other preparations) and also Gexamidinum 0.25-, Dephminum 0,117, [^lenobarbital 0,05- 0,1, Glupheral are first of all indicated in generalized tonico- clonic attacks. Natrium valproat. Nitrozeram (Clonazepam, Antelepsinum),Sucilep (Etocuksimidum) 0.25 - are recommended in common absentias epileptica. In complex absentias Carbamazepinum is more preferable. Natrium valproat. Clonazepam, Antelepsinum take the tirest place in muscular-spasmodic attacks. In dystrophic disorders Carbamazepinum is considered to be the best preparation. Gexamidinum, Dephenil. Natrium valproat. Phenobarbital, Benzonal, Carbamazepinum are used in clonic and tonic attacks. *The* daily dosage of a preparation is identified according to therapeutic resistance of attacks, their frequency and patient's body weight. For lack of effective monotherapy, polytherapy is conducted, using other preparations, that are recommended for a certain type of attacks. Pathogenic treatment: resorption (Lydasa, Aloe), dehydrative ones (Diacarb, Hypotiazidum, Phurosemidum), generally-roborant ones (polyvitamins and others) are also used for epilepsy. From non- medicinal ways of treatment it is recommended: psychotherapy, ergotherapy, rational job placement, etc. In successful usage of complex medicinal measures various degree of relief may be obtained in 95 % of patients, arresting of attacks in 50%-70% of patients.

JACKSONIAN EPILEPSY

Is symptomatic partial epilepsy, that is characterized by localization of an epileptic focus in the motor or (less freguently) sensitive part of one of the cerebral cortex hemispheres, and correspondingly to that somatomotor or somatosensory epileptic attacks with the focal beginning in any part of the body on its one side. Jacksonian epilepsy is caused by an organic affection of the brain after traumas, infections, disorders of cerebral blood circulation, tumours, vascular brain's anomalies, parasitogenic diseases, perinatal pathology.

For the first time unilateral somatomotor attacks were described in 1827 by a French physician Bravae, a detailed clinical picture was given by an English neurologist J.Jackson in 1870.

Clinical picture of Jacksonian epilepsy is characterized by appearance ofclonic convulsions, less seldom clonico-tonic or tonico- clonic spasms on the background of clear consciousness, which start, depending of the focus of localization with movements corresponding to presentation of this or that part of the body in the frontal central convolution. In the process of attack's development the convulsions are spread, involving new motor areas of the same side of the body, according to their space location in the cortex ("Jacksonian march"). Most often the attack begins with the first finger of the hand with the following change to forearm, arm. then face and leg. In every patient the convulsions start with the same segment of the limb or face's muscles. Sometimes it is possible to stop an attack, starting in the hand, if hold him strong by hand. Ceasing of convulsions occurs guickly in reverse motion of fit's development. There may be an epileptic attack, restricted by one extremities, face's muscles or permanent repetition of" Jacksonian march". While broadened spreading of convulsions in the process of attack's development, their spreading on tine other side - the loss of consciousness may be observed (secondary generalized attack). It should be noted that the typical peculiarity is post- attacked short term paresis or extremity's paralysis, from which the attack has begun.

Somatosensory fits are manifested as attacks of paresthesias, break of temperature or painful sensitivity, they are spread typically for successive involving of posterior central convolution's various parts.

Diagnostics is carried out on the basis of the attack's type, availability of focal neurologic symptoms: pareses, paralyses, sensitivity's disfunctions. EEG investigations in a number of patients allow to observe focal cortical discharges of epileptic activity looking like "peak", complexes "peak-wave", sharp waves, transforming into multiplied sharp waves at the final stages of an attack, focal smooth activities corresponding to epileptic focus. It is very necessary to carry out a detailed examination of patients with epileptic attacks of " Jacksonian type", as these fits are often manifestations of such terrible cerebral diseases as tumors and arteriovenous aneurisms. Great attention should be paid to the results of EEG, KT and MEG, angiography, craniography. investigation of pressure and cerebrospinal substance's content.

Treatment of the main process that caused partial tits.

Anticonvulsive therapy: Gexamidinum, Dypheninum, Phenobarbital, Benzonal, Eamictal, and preparations of Valproid acid (Depakiniim, Convulsophinum, Oiphiril, Acediprol and others). Recently there are facts about high efficiency of partial attack' treatment with electrostimulation of a pneumogastric nerve. Pathogenetic therapy; Dehydrogenative, resolving, generally-strengthening. Prognosis is determined by the disease, caused epilepsy and efficiency of the conducted therapy.

Kozhevnikovs epilepsy

Is a disease first described by as a well known neurologist Kozhevmkov A, Ya, in 1895. The author noted that epilepsia partialis continua was characterized by combination of often permanent motor Jacksonian paroxysms with local myoclonic twitch ings (or tics). He expressed his supposition that the main reason of the disease was encephalitis, that: *was* confirmed by other investiogators later. Kozhevnikov's epilepsy occurs rather seldom.

Etiology factors: neiiroinfections, traumas, impairment of cerebral blood circulation, but the basic one is Russian tick borne encephalitis.

Clinical picture. It may appear at any age. The main symptom is one-sides partial motor (clonic) convulsions, proceeding without the lost of consciousness, they may be restricted by muscles of face, arm, leg or half of the body, while alarming paroxysms are increased. Myoclonias are stereotyped, permanent and oftener observed in those groups of muscles or parts of the body from where "Jacksonian march" starts. In most cases myoclonias appear during the first year of the disease.

In neurologic status of a patient with epilepsy (Kozhevnikov's syndrome) hemipareses, stipulated by the main disease (neuroinfections), are observed most often. The disease's progress

and pemohty's change, as a rule, are absent. Adhesions or complexes "adhesion waves" with clear localization in the central areas are registered on EEG.

Treatment: The preparations of first rank are Carbamazepinum and Natrium Valproat, more often attacks resistant to therapy are observed. Surgical correction helps to eliminate the attacks, but, according to some information, they appear c'nce more in some time. Great contribution in studying epilepsy had been done by collaborators of the department of Nervous Diseases, Odessa State Medical University. Thus original monograph "Epilepsy and its role in the history of 19th century" belongs to professor Popov N. M. This monograph had not lost its meaning until now. Professor Popadato L. L with professor Bakkalo S. A were the first who had used the tissue therapy for treatment of posttraumatic epilepsy.

Myoclonic epilepsy had been studied by professor Sokolyanskey G. G in details. First by himself together with Professor Dubovoy E. D and Curako Yu. I. radioactive phosphorus had been offered for treatment of epilepsy. Toxoplasmosic epilepsy had been described by academician Curako Yu. L for the first time.

Additional information.

This information is recommended by the Central Methodical Study of Ukraine's Ministry of Health protection of Higher Medical Education dealing with questions in Neurology for examination cards in Therapy at the State exams in 1999.

Question: Epileptic status. First aid

Brief answer: This is a series of convulsive attacks that follow one after another, without coming back of the sense of fatigue between them. Clinical picture The patients suffer from coma with frequent and permanent convulsions. If not treated, epistasis is quickly getting complicated by dehydration., electrolytic impairments, aspirated pneumonia, heart rhythm's disorder.

Treatment is carried out only at in-patient department. Till now wlien chloral hydrate and magnesium sulfate had played the main role in treatment, mortality rate in epistasis mounted to 30 %. Now' it is decreased to 34 times. Natrural passing of air should be maintained with the help of endotracheal tube, prevention of tonque retraction, phareyngeal and bronchial secretum are to be done with frequent suction. One-two ampullas of Seduxenum in 20 ml of 40% glucose solution is smoothly introduced intravenously, after that in the most cases the attacks are over. The action of Seduxenum lasts for 15-20 minutes. For lack of effect Tiopental or Gexenal are introduced intravenously. One gramme of preparation is debited with 10 ml ofisotonic solution of sodium chloride at a rate of 1 ml per 10 kg of patient's body weight. At revivication department the above mentioned barbiturates are introduced intravenously with the help of a dropper controlling pulse, pressure, respiration. Simultaneously 5 ml of 10 % Tiopentalium (Gexenalum) solution is introduced intramuscularly. Sodium Oxibutiratiim (20% solution) is olso used for intravenous anesthesia.

In resistant cases nitric and oxygen oxide with ratio 2:1 with myorelaxion drugs are used for inhalative anesthesia. At the highest degree the timely beginning of therapy is actual: delay of Seduxenum introduction for 6 hours decreases its efficiecy at 3 times. The patients are recommended the whole complex of revivicative measures: intubation, glucocortcoids. saluretics, cardiac preparations. Dexametzonum up to 12 mg (3 ampullas) is indicated for the brain's edema and increased intracranial pressure.

Additional information. This information is recommended by the Central Methodical Study Of Ukraine's Ministry of Health protection of Higher Medical Education dealing with questions in Neurology for examination cards in Therapy at the State exams in 1999.

Question: Paroxysmal non-epileptic status. Diagnosis and treatment

Brief answer:

Raynaund's disease.

Raynaund's disease is one of the forms of angiotrophoneuroses, characterized by the attacks of spastic vasomotorial disorders. In classic cases the attack is formed of three phases. The first phase is manifested by paleness and fingers'cooling, accompanied by intensive pain. Cyanotic

discoloration and intensive pain are typical for the second phase. In the third phase the fingers'skin is becoming bright red, the pain disappears.

Treatment. Adrenergic and vasodilative drugs (Dehydroergothaminum, Redergam, No-Shpa), ganglioblocking preparation (Pahicarpinum), neuroleptic remedies (Reserpinum, Gexonium-0,1-0,25 gr 3 times a day for 10 days.).

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.

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

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Lecture No. 5

Topic: Neurological aspects of brain injury.

Classification of craniocerebral traumas. Etiology and pathogenesis of cerebral concussion, contusion and compression, and subarachnoid hemorrhage. Traumas of spinal marrow: clinical course, diagnostics, and treatment. Complications of craniocerebral and spinal traumas, their treatment and prevention".

1. Actuality of the topic. Statement of the topic.

Traumas of brain and spinal marrow are very important medical problem. Specialties of their course with the presence of many complications, pathogenesis and pathomorphology of traumatic disease of nervous system dictate the necessity of providing an emergency care for the determination of correct diagnosis. That's why neurologists, neurosurgeons, experts in resuscitation, legal doctors and doctors of other specialities must know this pathology, which belongs to urgent ones, must be able to examine the victim and provide an emergency care correctly and in time.

2. Aims of the lection.

Teaching:

- To acquaint the students with the frequency of craniocerebral traumas (CCT), their complications, those occur during them. Create a conception about extraordinary actuality of traumatism in developed countries, their social character (the first degree of abstraction).
- On basis of lectural material the student must know: clinical classification of CCT, their pathogenesis, pathomorphology and clinical course of closed CCT, their pathogenesis, pathomorphology, clinical course of spinal marrow trauma (the second degree of abstraction).
- Proceeding from aforesaid the students must get skills in examination of patients with cerebral concussion, contusion and compression, determination of location of focus of lesion, appointment of adequate additional methodics of patient's investigation and appointment of emergency care (the third degree of abstraction).

Educating:

- Directed on the development of professionally significant substructure of personality
- Teaching of students of modern professional thinking
- Providing of student's mastering leading meaning of home clinical, scientific-pedagogical schools in elaboration of problems of the lecture especially schools of Odessa
- Students' mastering skills in 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)

Craniocerebral trauma is the leading cause of death among young people. Emerging after primary brain damage pathophysiological changes lead to repeated injuries. Factors such as hypoxia, hypercapnia and hypotension only increase the expressivity of these changes. Intensive therapy with craniocerebral injury aims to prevent a repeat of damage to the main brain. Therapeutic activities that are conducted in the light of pathophysiologic violations significantly improve the course of pathology with craniocerebral injury.

Epidemiology.

Craniocerebral trauma is the leading cause of disability and death among young people. Deems it in the United States brain injury occurs at a frequency of 200 cases for every 100,000 people a year. Each year, approximately 500,000 people received serious brain trauma, of which 450,000 are in the hospital and 50,000 die before a fall in hospital. Among the 450,000 people that fall in hospital, cases of significant disability there are approximately 100,000 people per year. Craniocerebral injury often occurs for all young people aged 15 to 24 years. According to statistics, men get this type of injury is two to three times more likely than women in all age groups. More than 50% of all cases of cranial trauma, and 70% of these deaths occur when craniocerebral injury were traffic accidents. In densely populated urban areas the use of firearms defines a large percentage of craniocerebral injury. The second main reason is falling from height. More than 50% of patients with severe craniocerebral injury highlights the multiple injuries that result in significant loss of blood, systemic hypotension and hypoxia. The classification of severe

craniocerebral injury done on a scale coma Glasgow, which allows to assess the extent of neurological damage, taking into account the results of tests on opening the eyes, voice tests and tests of motor function. The total maximum possible number of points is 15; a serious craniocerebral injury, say, when over 6 hours and longer total estimated number of points is 8 or less. Glasgow coma scale and the scale of its outcomes on the Glasgow allowed to compare the level of neurological damage in different patients and to predict the output of the state. In general, mortality is highly dependent on the first of scores obtained on the scale of Glasgow. Nevertheless, under the same injury and the same number of points, people old age have the worst prognosis than young people.

Pathophysiology.

Craniocerebral injury leads to brain damage, which develops in two stages. Initial damage due to the influence of biomeChanical forces that affect the skull and brain trauma at the time, and it develops over the milliseconds. At this moment there medullar concussion substance with diffuse damage to neurons and the white brain matter, as well as happens gap arteries and veins, which leads to multiple petechial hemorrhage. Primary damage included a concussion and contusion main brain vessels and education gap hematomas (epidural, subdural, subarachnoid or intracerebral).So far it has not yet been offered no means of therapy of primary damage.

Repeated injury develops in a few minutes or hours of injury and is a set of pathological changes that occur as a result of primary damage and lead to ischemia and brain edema, intracranial hemorrhage, intracranial hypertension and education hernial protrusion. By re factors that exacerbate the primary injury include hypoxia, hypercapnia, hypotension, anemia, and hyperglycemia. Preventing or correct these repeated treatment of pathological conditions improve exits, with craniocerebral injury. Cramps, sepsis and infectious complications that may arise at a later date after craniocerebral injury (a few hours or days) will further exacerbate brain damage, so they also must be careful to fight and prevent their occurrence.

After a craniocerebral injury, against a backdrop of severe generalized or focal violations occur unrecoverable neuropathological change, which is due to the emergence of one of two reasons: 1) cerebral circulation becomes inadequate, or 2) metabolic needs of the brain increases dramatically. Cerebral circulation may become inadequate in the system lowering blood pressure and increasing intracranial pressure, since these factors lead to a significant decline in cerebral perfusion pressure and cause ischemic brain damage. Metabolic needs of the brain increases, for example, hyperthermia, or epileptic status, because these states need the brain of oxygen and glucose exceeds the capacity of cardio-pulmonary system for their delivery. While not yet fully determined the reasons for brain injury raises the circulation of blood vessels in the brain, but this factor is involved in the repeated damage to the brain. Thus, an imbalance in the ratio of delivery of oxygen to the brain / needs the brain of oxygen and the failure of coping mechanisms in the cerebrovascular system is that the brain becomes more susceptible to the action of additional damaging factors such as fluctuations in blood pressure, blood rheology violation or hypoxia.

Traumatic brain injury starts a cascade of potentially harmful biochemical changes. There have been changes in intracellular calcium content, oxygen free radicals are released and vasoactive metabolites arachidonic acid, which damages vascular endothelium and membranes of neurons. In addition, there has been steady accumulation of "excitable" amino acids, such as glutamat and aspartate, and the process is so faster than heavier Injuries to the brain. Clinical studies are under way, seek to assess how Ascenders calcium channel, acceptors of free radicals and drugs from other groups might affect the period of biochemical reactions occurring during ischemia and brain damage. If these drugs prescribers in the early period after cranial trauma, whereas in theory they can use to warn pathophysiological changes and improve the output of the state.

Intracranial pressure. Brain is in the cranium, which has a fixed amount. Intracranial pressure (ICP) is increased when the amount of any component that contained in the cranium (cells, intracellular / extracellular fluid, cerebrospinal fluid, blood) increases so that the

compensatory mechanisms wiped out. In the normal value of ICP in humans is less than 10mm. Intracranial flexible structure, so the increased intracranial contents initially did not lead to a significant increase in ICP. However, comes a time when compensation arrangements are no longer possible to cope with the increasing volume, which is contained in the cranium, and then the slight increase in volume will lead to increased ICP. Increased ICP with craniocerebral injury may be due to hematoma, cerebral edema and a violation of cerebrospinal fluid intake. Intracranial hypertension can lead to two major pathological effects: cerebral ischaemia and herniation brain substance. Increased ICP leads to the main cerebral ischemia by cerebral perfusion pressure. DAP determine subtract from the mean arterial blood pressure values of ICP. The significance of DAP decreases as the value of ICP is close to the mean arterial pressure. Increased ICP has led to a decline in DAP, and when the latter figure reaches its critical value (about 50 mm), Developed cerebral ischemia chief. Intracranial hypertension requires aggressive therapy. The goal of therapy, which is carried out is to maintain the value of DAP at the same higher for the 70 mm . That indicates the adequacy of cerebral blood flow.

Cerebral blood flow.

Craniocerebral injury leads to violations of dynamic cerebral blood flow (MK) and cerebral metabolism. Children and young people flushing phase may be less short or absent altogether. Can develop and cerebral hypoperfusion. In the acute phase of craniocerebral injury cerebral autoregulation usually affected. Hypertension can cause flushing the brain that leads to brain edema and increasing the ICP. Hypotension may lead to cerebral ischemia, which leads to cytotoxic edema.) Under normal circumstances, hypocapnia accompanied by a narrowing of brain vessels, and hypercapnia leads to increased vascular brain. In patients with craniocerebral injury sensitivity of the brain to the C02 rises. In patients with craniocerebral injury must check consumption brain of oxygen consumption of brain lactate and the value of MK, as it allows the display of patients with ischemic brain damage.

In clinical conditions such monitoring, through continuous measurement of oxygen saturation of hemoglobin in the bulbar jugularis veins (Sj02) and determining the concentration of lactate in the cerebrospinal fluid brain edema.

Injury sufficient on its own strength to cause hemorrhage in the tissue that violates the integrity of the blood-brain barrier and leads to protein-rich fluid through the damaged vessel. Arterial hypertension and the use of anesthetics, which increases the BC, supporting progression of this type of edema. Cytotoxic edema caused by oxygen starvation brain cells and is accompanied by accumulation of sodium and water inside the cells. The global and focal ischemia or prolonged hypoxia - all these factors could lead to cytotoxic brain edema. Regardless of not finished great research to date has not yet developed a unified scheme of a specific therapy of brain edema with craniocerebral injury. It is thought that mannitol and other hypertensive solutions that are used to reduce the ICP and decrease brain edema, possess the ability to remove water from the brain tissue only when the blood-brain barrier is not affected. Steroid drugs reduce edema tissues around the brain abscesses, and pockets of tumors, but their use in the swelling of the brain against the backdrop of craniocerebral injury proved ineffective.

Pathogenesis of basic forms and morphological manifestations of acute closed craniocerebral injury.

The historical aspect of one of the first theories of pathogenesis need to recognize a number of mechanical theories skull injuries. These theories need to include the vibration theory of traumatic brain substance, at the time of injury. Putnem and Geyi focused on mechanical gaps brain tissue. In its place, the theory went the theory that analyzed the prevailing divisions brain stem lesion (Khoroshko, Valshonok, Gurevich, Krol, Sharapov, etc.).

There are theories of liquor Push (Dyure, 1878) on the basis of this theory came the expansion of the 3rd ventricle after skull injuries. Hydrodynamic theory - all changes in the trauma caused by hydrodynamic wave, as a result of traumatic agent with further changes in brain structures as a result of varying density brain substance.(Bush, Bruns, Solovyov, Ilyin and iOLI.).

Vasomotor theory (K.Bernard, Z.Botkin), one of the first highlighted the link between vasomotor reactions and those of the brain. Morozov (1862), one of the first brought development of the receptacle in response to injury. In the future, these issues were developed in the works Ogneva, Klossovskogo, Yegorova, Arutyunova. In the last decade, many original morphological changes predominantly vascular system of various divisions of the brain showed Y.Kurako with different forms of closed craniocerebral injury. Of particular importance is the theory asynapsia (N.Graschenkov 1946) that brings basis for a number of neuro-reflex violations of the clinical manifestations of acute craniocerebral injury.

Physical and chemical theory (Malm, Schults, Reyhard, Nasonov, etc.) Stress violation of colloidal equilibrium of cellular proteins, the development of edema – swelling of the brain substance.

In XX century, especially the widespread neuro-reflex theory. Modern research leading neurosurgical schools have greatly expanded the role of neuro-reflex and vascular changes in the development of traumatic disease (N.I.Graschenkov, Aratyan A.A., Romodanov A.P., Pedachenko A., Babchin I.L., Ugryumov B.M.,Arutyunov.).

It must be stressed that despite the large number of theories of the pathogenesis of craniocerebral injury, each of them does not exclude, but rather a complement to one another, showing the diversity of causes and mechanisms that occur during the Trauma skull and brain.

In general, for understanding the pathogenesis of clinical manifestations of acute, and later closed craniocerebral injury, remember the following: - All the pathological processes in the brain due to injury was the result of reflex- vascular changes in the body altogether. Emerging pathological processes in the brain interact with one another.

The clinical effects of closed craniocerebral injury.

Concussion - mild acute closed brain injuries. The most characteristic and the main feature of the brain need to recognize development unconscious immediately after the injury for a period of several seconds to 20-30 minutes. Only in rare cases, patients did not indicate loss of consciousness, but in these cases they are expressed headache, vegetative violations, expressed general weakness, repeated vomiting at rest and in which there are over 1-3 days after injury.

The second most informative feature of the brain is loss of memory of trauma that took place and further her events (retrograde amnesia), some memory loss and an event that preceded the trauma (anterograde amnesia). Immediately you want to emphasize two circumstances. First: the establishment of the criteria of the brain is very difficult or not possible in children, especially young children.

Second: Similar difficulties arise in determining the violations of consciousness and memory loss in people with trauma stratify the concerns of alcohol intoxication. Therefore, issues of diagnosis, evaluation of clinical characteristics of the brain, as well as other kinds of traumatic lesions of the brain must be weighed against the secular backgrounds, premorbidal of the body and especially of its vascular system.(A.P.Romodanov 1982).

In a study of patients with concussion in the acute period need to pay attention to the almost complete absence of these patients campfire symptoms of central nervous system against the backdrop of expression of a number of brain symptoms.

Among the symptoms to the fore are: headache varying degrees of terms and duration, which is sharply increasing voltage physical effort, somnolent states in the first hours after the injury. A major symptom is the presence of persisten is not systematic.During the first three days indicates weakness, sweating, poor sleep, increased irritability to bright light, sound stimuli. In the first days after the injury indicates tachycardia or bradycardia, sometimes fluctuation of blood pressure, paleness leather cover. A number of patients have a persistent vegetative violations asthenic condition, and sometimes more serious mental disorders - depression, hypochondric manifestations, emotional discord.

Concussion should be distinguished from conventional strike chapter, in which there is no loss of consciousness and patients tend to remember how and under what circumstances was injured, leading an active, they can not get manifestations. Bruising (concussion) of the brain - a

more difficult form of closed brain injury.

In this form of traumatic brain damage, as noted above, there is loss of individual sites brain, which is clinically campfire identifying symptoms of central nervous system.

There are three degrees of impact the brain: easy, medium and difficult. Mild degree of brain contusion is longer than with concussion, lost consciousness, although found surveillance when struck by the brain is a brief loss of consciousness. More than that described in the literature and we have seen cases of gunshot injuries of skull and brain penetrating nature, when patients are not noted any loss of consciousness at all (A.P.King 1968). Expressivity symptoms of varies and usually depends on the mechanism of injury (shock-shake mechanism of injury often is accompanied by a more pronounced symptoms, while a local trauma dominated focal symptoms of brain injury). A distinctive feature of brain contusion is focal development of symptoms - usually when light degree of asymmetry are reflex, sometimes a light facial nerve paresis, or nerve, which gives, a non-permanent horizontal nystagmus, one or the other vestibular failure.

By brain contusion should be treated fractures of the skull or the identification of blood in liquor even in the absence of focal neurologic symptoms. Brain contusion medium are more pronounced focal symptoms of central nervous system in the form of mono-or side hemiparesis, reflex expressive asymmetries with pathological symptoms, violations of the language, not blatantly expressed violations of the sensitivity, visual impairment, hearing loss, other signs of injury brain nerves .All these focal symptoms in the acute period of trauma can be masked expressed symptoms or condition. In clarifying, consciousness focal symptoms is more pronounced.

In light and medium brain contusion, usually no signs of welcoming the vital functions of the brain.

Brain contusion difficult degree is very dangerous to life and are characterized by a prolonged loss of consciousness, gross violations of neurological status as focal rift, a deep rift metabolism, blood circulation (BC). Actually, all these violations are accompanied by or violations occur against the backdrop of a number of vital functions of the brain stem divisions.

It is important to localization of brain contusion. For example, there are bruises mainly convexital divisions brain, which often take place over all with bone fractures of the skull. In contrast convexital contusion in the framework of skull fractures are more likely to develop basal divisions of the brain bruises. When the primary injury of brain stem divisions, especially the medulla oblongata, since trauma patients are unconscious in a deep coma with violations of breathing and cardiovascular activity.

By challenging brain contusion should also include brain injury foundation, diencefalic area, the deep divisions of the brain hemispheres. Often this injury can be multiple.

The condition of patients with this type of brain contusion difficult: they condition are at a fairly long time in a state of consciousness raised (sometimes 1-2 weeks after the injury) to the conservation corneal reflexes, swallowing. Violation of breath on the peripheral type occur later, when acceding to such factors as the accumulation of mucus, blood and liquor in the respiratory tract, glottal spasm, reducing the sensitivity trachea or bronchus, the violation of cough reflex, the development of pneumonia, etc.

Important role in the deterioration of the patients played by the development of edema, brain swelling, development of hypoxia condition, as well as bleeding in the basal cisterns or ventricles of brain.

It should be particularly emphasized that in a difficult degree of impact the brain, especially basal or deep divisions, and as a result of hypoxia and human blood, can develop signs or entire deficiency syndrome diencefalic area mesencefalo-bulbar field, etc.(Ugryumov et al.).

These syndrome, which for a traumatic disease may change, is of great significance in the choice or treatment.

Thus, when strikes the brain need to seek clarification of, except to the gravity of the state, containment of the overwhelming fire damage: convexital or basal divisions, root or stem of education, single or multiple fires strike, as well as the presence of fires impact on the opposite

side of the skull trauma, in a contrecoup . And in the end, great importance is the establishment of bleeding, which extended into the brain cell destruction, or the raging development of cerebral edema that leads to a sharp increase of intracranial pressure and the development of dislocations and life-threatening brain patient.

At the end of clinical course and diagnosis of brain attacks, given the importance of the disengagement to the gravity of attacks taking into account the ratio focal, the symptoms of brain violation of conscience and give a summary table (№ 2) submitted by the Central Institute of Neurosurgery (A.N.Konovalov et al.1982.).

Acute Epidural Hemorrhage As a rule, this condition is due to a temporal or parietal fracture with laceration of the middle meningeal artery or vein. Tess often, there is a tear in a dural venous sinus. The injury, even when it fractures the skull, may not have produced coma initially or it may be part of a devastating craniocerebral injury. A few hours or a day later (exceptionally, with venous bleeding, the interval may be several days or a week), headache of increasing severity develops, with vomiting, drowsiness, confusion, aphasia, seizures (which may be one-sided), hemiparesis with slightly increased tendon reflexes, and a Babinski sign. As coma develops, the hemiparesis may give way to bilateral spasticity of the limbs and Babinski signs. When the patient remains untreated, the respirations become deeper and stertorous, then shallow and irregular; finally, they cease. The pulse is often slow (below 60 beats per minute) and bounding, with a concomitant rise in systolic blood pressure (Cushing effect). The pupil may dilate on the side of the hematoma. It should be emphasized that lumbar puncture is contraindicated in this setting, now that CT and MRI are available. Death, which is almost invariable if the clot is not removed surgically, comes at the end of a comatose period rarely if ever in a conscious patient and is due to respiratory arrest. The visualization of a fracture line across the groove of the middle meningeal artery and knowledge of which side of the head was struck (the clot is usually on that side) are of aid in diagnosis and lateralization of the lesion. However, meningeal vessels may be torn without fracture. The CT scan reveals a lens-shaped clot with a smooth inner margin. The surgical procedure consists of placement of burr holes, a craniotomy or drainage, identification of the bleeding vessel, and ligation. The operative results are excellent except in the cases with extended fractures and laceration of the dural venous sinuses, in which the epidural hematoma may be bilateral rather than unilateral, as it ordinarily is. If coma, bilateral Babinski signs, spasticity, or decerebrate rigidity supervene before operation, it usually means that displacement of central structures and crushing of the midbrain have already occurred; prognosis for life is then poor. Small epidural hemorrhages, followed by serial CT scanning, will be seen to enlarge gradually for a week or two and then be absorbed.

Acute and Chronic Subdural Hematoma The problems created by acute and chronic subdural hematomas are so different that they need to be discussed separately. In *acute subdural hematoma*, which may be unilateral or bilateral, there may be a brief lucid interval between the blow to the head and the advent of coma. Or, more often, the patient is comatose from the time of the injury and the coma deepens progressively. Frequently the acute subdural hematoma is combined with epidural hemorrhage, cerebral contusion, or laceration. The clinical effects of these several lesions are difficult to distinguish; there are some patients in whom it is impossible to state before operation whether the clot is epidural or subdural in location. The CT scan visualizes subdural clots accurately in more than 90 % of cases. In less acute hematomas, the fluid becomes isodense with the cortex, and its presence is betrayed only by a ventricular shift or, in MRI, by a widened space between the skull and brain. If the lesion is bilateral, there may be no shift. With contrast infusion, however, both imaging procedures usually reveal the vascular and inflammatory border surrounding the clot.

Treatment consists of wide craniotomy to permit control of the bleeding and removal of the clot. As one would expect, early removal of the mass is associated with the best outcome. The surgical results are less certain than in chronic subdural hematoma. If the clot is too small to explain the coma or other symptoms, there is probably extensive contusion and laceration of the cerebrum. Exceptionally, the subdural hematoma forms in the posterior fossa and gives rise to headache,

vomiting, pupillary inequality, dysphagia, cranial nerve palsies, and, rarely, stiff neck and ataxia of the trunk and gait, if the patient is well enough to be tested for these functions.

In *chronic subdural hematoma,* the traumatic etiology is less clear. The head injury, especially in elderly persons and in those taking anticoagulant drugs, may have been trivial and may even have been forgotten. A period of weeks then follows when headaches (not invariable), giddiness, slowness in thinking, confusion, apathy and drowsiness, and rarely a seizure or two are the main symptoms. The initial impression may be that the patient has a vascular lesion or brain tumor or is suffering from drug intoxication, a depressive illness, or Alzheimer disease. As with acute subdural hematoma, the disturbances of mentation and consciousness (drowsiness, inattentiveness, incoherence of thinking, and confusion) are more prominent than focal or lateralizing signs, and they may fluctuate. The focal signs usually consist of hemiparesis and rarely of an aphasic disturbance. Homonymous hemianopia is seldom observed, probably because the geniculocalcarine pathway is deep and not easily compressed; similarly, hemiplegia, i.e., complete paralysis of one arm and leg, is usually indicative of a lesion within the cerebral hemisphere rather than a compressive lesion on its surface. An important feature of the hemiparesis is that it may be contralateral or ipsilateral, depending on whether the contralateral cerebral peduncle has been compressed against the free edge of the tentorium by horizontal displacement (Kernohan-Woltman false localizing sign). If the condition progresses, the patient becomes comatose but often with striking fluctuations of awareness.

Dilatation of the ipsilateral pupil is a more reliable indicator of the side of the hematoma than the hemiparesis, though the side of pupillary abnormality may also be misleading (in 10 % of cases). Convulsions are seen occasionally, most often in alcoholics or in patients with cerebral contusions, but they cannot be regarded as a cardinal sign of subdural hematoma. Rare cases of internuclear ophthalmoplegia and of chorea have been reported, presumably as a result of distortion of deep structures. Transient disturbances of neurologic function simulating transient ischemic attacks (TIA) may occur. In infants and children, enlargement of the head, vomiting, and convulsions are prominent manifestations of subdural hematoma.

Computed tomography with contrast infusion and MRI are the most reliable diagnostic procedures; other investigative measures, formerly used, are seldom necessary. Skull films are helpful only when there is calcification surrounding a chronic hematoma, shift of a calcified pineal to one side, or an unexpected fracture line. The EEG is usually abnormal bilaterally, sometimes with reduced voltage or electrical silence over the hematoma and high-voltage slow waves over the opposite side, because of the damping effects of the clot and displacement of the brain, respectively. In an arteriogram, the cortical branches of the middle cerebral artery are separated from the inner surface of the skull, and the anterior cerebral artery may be displaced contralaterally. The CSF may be clear and acellular, bloody, or xanthochromic, depending on the presence or absence of recent or old contusions and subarachnoid hemorrhage; the pressure may be elevated or normal. A xanthochromic fluid with relatively low protein content should always raise the suspicion of chronic subdural hematoma.

Acute, rapidly evolving subdural hematomas are due to tearing of bridging veins, and symptoms are caused by compression of the brain by an expanding clot of fresh blood. Unlike epidural arterial hemorrhage, which is steadily progressive, venous bleeding is usually arrested by the rising intracranial pressure.

Subdural hygroma (a collection of blood and CSF in the subdural space) may form after an injury as well as after meningitis (in an infant or young child). It is postulated that a tear of the arachnoid permits bacteria to enter and excite a serous reaction in the subdural space. Subdural hygromas may appear without infection, presumably due to a ball-valve effect of the arachnoidal tear. In the past, subdural hygroma also occurred as a complication of pneumoencephalography. Shrinkage of the hydrocephalic brain after ventriculoperitoneal shunting is also conducive to the formation of a subdural hematoma or hygroma. Drowsiness, confusion, irritability, and fever are relieved when the subdural fluid is aspirated or drained.

Treatment of Subdural Hematoma In most cases it is sufficient to place burr holes and

evacuate the clot before deep coma has developed. In others, of a more chronic standing, a craniotomy must be performed and an attempt made to strip the membranes that surround the clot. This diminishes the likelihood of reaccumulation of fluid but is not always successful. Other causes of operative failure are swelling of the compressed hemisphere or failure of the hemisphere to expand after removal of a large clot. Although no longer a common practice, the administration of corticosteroids is an alternative to surgical removal of chronic subdural hematoma in patients with minor symptoms or with impediments to surgery. Headache and other symptoms such as gait difficulty or limb clumsiness may satisfactorily resolve after several weeks of medication and usually remain abated when the steroids are slowly reduced. Small subdural hematomas causing no symptoms and followed by CT scans will self-absorb, leaving only a deep yellow, sometimes calcified membrane attached to the inner dural surface.

Cerebral Contusion Severe closed head injury is almost universally accompanied by cortical contusions and surrounding edema. The mass effect of contusional swelling, if sufficiently large, is a major factor in the genesis of tissue shifts and raised intracranial pressure. The CT appearance of contusion has already been described. In the first few hours after injury, the bleeding points in the contused area may appear small and innocuous. The main concern, however, is the tendency for contused areas to swell or to develop hematomas. This may give rise to delayed clinical deterioration, sometimes abrupt in onset and concurrent with enlargement of the damaged region on the CT scan. It has been claimed, on uncertain ground, that the process of acute contusional swelling is precipitated by the excessive administration of intravenous fluids (fluid management is considered further on in this chapter).

Traumatic Intracerebral Hemorrhage One or several cerebral hemorrhages may be apparent immediately after head injury or be delayed in their development by several days (*Spatapoplexie*). They may occur in the subcortical white matter of one lobe of the brain or in deeper structures, such as the basal ganglia or thalamus. The injury is nearly always severe; blood vessels as well as cortical tissue are torn.

The clinical picture is similar to that of hypertensive brain hemorrhage (deepening coma with hemiplegia, a dilating pupil, bilateral Babinski signs, stertorous and irregular respirations). It may be manifest by an abrupt rise in intracranial pressure. Of course, a slowly expanding clot due to venous bleeding might permit a relatively lucid interval of 2 to 3 days between injury and the symptoms of the cerebral hemorrhage, but this is not an apparent explanation in most of the cases. In elderly patients, a problem that sometimes cannot be solved, even at postmortem examination, is whether there had been a fall caused by the hemorrhage or a fall followed after an interval by the common variety of primary intracerebral hemorrhage or whether a *delayed hemorrhage* into traumatized brain tissue had occurred. Coma or confusion, if present from the time of the injury, may obscure the signs of the intracerebral hemorrhage. Craniotomy with evacuation of the clot has given a successful result in a few cases, but the advisability of surgery is governed by several factors, including the level of consciousness, the time from the initial injury, and the associated types of damage that are shown on the CT scan. The wider application of intracranial pressure monitoring and of CT scans at intervals after the injury should facilitate diagnosis and perhaps help to elucidate the pathogenesis of the delayed hemorrhage.

SEQUELAE OF HEAD INJURY

Posttraumatic Epilepsy

Epilepsy is the most common delayed sequela of craniocerebral trauma, with an overall incidence of about 5 % in patients with closed head injuries and 50 % in patients who had sustained a compound skull fracture and wound of the brain. The basis is nearly always a contusion or laceration of the cortex. As one might expect, the risk of developing posttraumatic epilepsy is related to the severity of the head injury. In the cohort of 2747 head-injured patients described by Annegers and colleagues, the risk of seizures after severe head injury was 7 % within 1 year and 11.5 % in 5 years. If the injury was only moderate (unconsciousness or amnesia for 30 min to 24 h), the risk fell to 0.7 and 1.6 %. After mild injury, the incidence of seizures was not significantly greater than in the general population. The likelihood of epilepsy is said to be greater in parietal

and posterior frontal lesions, but it may arise from lesions in any area of the cerebral cortex.

The interval between the head injury and the first seizure varies greatly. A small number of patients have a generalized seizure within moments of the injury *(immediate epilepsy).* Usually this amounts to a brief tonic extension of the limbs, with slight shaking movements immediately after concussion, followed by awakening with a mild confusional state. Whether this represents a true epileptic phenomenon or a reticular discharge, or, as appears more likely, is the result of arrest of cerebral blood flow, is unclear. Some 4 to 5 % of hospitalized head- injured individuals have been said to have one or more seizures within the first week of their injury *{early epilepsy),* but this is higher than in our experience. Immediate seizures have a good prognosis; on the other hand, *late epilepsy* is significantly more frequent in patients who had experienced early epilepsy (Jennett). In medical writings, the term *posttraumatic epilepsy* usually refers to late epilepsy, i.e., to seizures that develop several weeks or months after the head injury (1 to 3 months in most cases). Approximately 6 months after injury, half the patients who will develop epilepsy have had their first attack, and by the end of 2 years the figure rises to 80 % (Walker). The interval between head injury and development of seizures is said to be longer in children. The longer the interval, the less certain one is of its relation to the traumatic incident. Data derived from a 15-year study of military personnel with severe (penetrating) brain wounds indicate that patients who escape seizures for 1 year after injury can be 75 % certain of remaining seizure-free; patients without seizures for 2 years can be 90 % certain, and for 3 years, 95 % certain. For the less severely injured (mainly closed head injuries), the corresponding times are 2 to 6 months, 12 to 17 months, and 21 to 25 months (Weiss et al).

Posttraumatic seizures are either focal in type or generalized with loss of consciousness (grand mal); petit mal is rarely if ever due to trauma. The frequency of seizures in any given patient varies widely; some patients have only a few, others many, with occasional flurries of status epilepticus. The EEG is of value in diagnosis; a focus of spike or sharp waves is the characteristic finding.

Posttraumatic seizures tend to decrease in frequency as the years pass, and some patients (10 to 30 %, according to Caveness) eventually stop having them. Individuals who have early attacks (within a week of injury) are more likely to have a complete remission of their seizures than those whose attacks begin a year or so after injury. A low frequency of attacks is another favorable prognostic sign. Alcoholism has an adverse effect on this seizure state. We have observed some 25 patients with posttraumatic epilepsy in whom seizures had ceased altogether for several years but then recurred only in relation to drinking. In these patients the seizures were precipitated by a weekend or even one evening of heavy drinking and occurred, as a rule, not when the patient was intoxicated but in the sobering-up period.

Treatment and Prophylaxis Usually the seizures can be controlled by anticonvulsant medications, and relatively few are recalcitrant to the point of requiring excision of the epileptic focus. The surgical results vary according to the methods of selection and techniques of operation. Under the best of neurosurgical conditions three decades ago, with careful selection of cases, Rasmussen (also Penfield and Jasper) was able to eradicate seizures in 50 to 75 % of cases by excision of the focus.

Autonomic Dysfunction Syndrome in the Vegetative State

A troublesome consequence of severe head injury, observed in patients with the persistent vegetative state, is the occurrence of episodic violent extensor posturing, profuse diaphoresis, hypertension, and tachycardia, lasting minutes to an hour. A slight fever may accompany the spells. Families and staff are greatly disturbed by the display, particularly when accompanying grimacing suggests suffering. These spells of hypersympathetic activity and posturing may be precipitated by painful stimuli or distension of a viscus, but more often they arise spontaneously. The syndrome is often identified as a seizure and in many texts is still incorrectly referred to as "diencephalic epilepsy," but it is more likely the result of decortication, allowing the hypothalamus to function independently of normal inhibitory mechanisms. Narcotics and diazepines have a slight beneficial effect, but bromocriptine, which may be used in combination with sedatives or small

doses of morphine, has been more effective (Rossitch and Bullard).

Posttraumatic Nervous Instability (Postconcussion Syndrome)

This troublesome and frequent sequela of head injury has also been called the *posttraumatic* or *postconcussion syndrome*, *posttraumatic headache*, *traumatic neurasthenia* and *traumatic psychasthenia.* Headache is the central symptom, either generalized or localized to the part that had been struck. It is variously described as an aching, throbbing, pounding, stabbing, pressing, or band-like pain and is remarkable for its variability. The intensification of the headache and other symptoms by mental and physical effort, straining, stooping, and emotional excitement has already been mentioned; rest and quiet tend to relieve them. Such headaches may present a major obstacle to convalescence. Dizziness, another prominent symptom, is usually not a true vertigo but a giddiness or lightheadedness. The patient may feel unsteady, dazed, weak, or faint. However, a certain number of patients describe symptoms that are consonant with labyrinthine disorder. They report that objects in the environment move momentarily, and that looking upward or to the side may cause a sense of unbalance; labyrinthine tests may show hyporeactivity; far more often they disclose no abnormalities.

The patient with posttraumatic nervous instability is intolerant of noise, emotional excitement, and crowds. Tenseness, restlessness, inability to concentrate, feelings of nervousness, fatigue, worry, apprehension, and an inability to tolerate the usual amount of alcohol complete the clinical picture. The resemblance of these symptoms to those of anxiety and depression is at once apparent. In contrast to this multiplicity of subjective symptoms, memory and other intellectual functions tend to show little or no impairment on detailed testing, although this has been disputed.

The syndrome of posttraumatic nervous instability complicates all types of head injury, mild and severe. Once established, it may persist for months or even years, and it tends to resist all varieties of treatment. Eventually, the symptoms lessen. Strangely, this syndrome is almost unknown in children. Characteristic also is the augmentation of both the duration and intensity of this syndrome by compensation problems and litigation, suggesting a psychologic factor.

Extrapyramidal and Cerebellar Disorders

The question of *posttraumatic Parkinson syndrome* has been discussed many times, usually with the conclusion that it does not exist. Most such patients probably had paralysis agitans or, in earlier times, postencephalitic parkinsonism brought to light by the head injury. Cerebellar ataxia and extrapyramidal signs are rare consequences of cranial trauma unless the latter was complicated by cerebral anoxia. When cerebellar ataxia is due to the trauma itself, it is frequently unilateral and the result of injury to the superior cerebellar peduncle. An ataxia of gait may reflect the presence of a communicating hydrocephalus. The one exception to these statements is a parkinsonian syndrome in ex-boxers as a manifestation of the "punch-drunk" syndrome (see below).

"Punch-Drunk Encephalopathy" (Dementia Pugilistica) The cumulative effects of repeated cerebral injuries, observed in boxers who had engaged in many bouts over a long period of time, constitute a type of head injury that is difficult to classify. What is referred to is the development, after many years in the ring (sometimes toward the end of the boxer's career, more often a number of years after retirement), of dysarthric speech and a state of forgetfulness, slowness in thinking, and other signs of dementia. Movements are slow, stiff, and uncertain, especially those involving the legs, and there is a shuffling, wide-based gait. Often a parkinsonian syndrome emerges and sometimes a moderately disabling ataxia. The plantar reflexes may be extensor on one or both sides. The EEG contains slow waves of theta and sometimes of delta type. The clinical syndrome was analyzed by Roberts and colleagues, who found it present to some degree in 37 of 224 professional boxers they examined. More recent studies have shown that in about one-half of all professional boxers, both active and retired, the CT scan discloses ventricular dilatation and/or sulcal widening and a cavum septi pellucidi. These abnormalities had been demonstrated many years before, by pneumoencephalography, and were found to be related to the number of bouts. The pathogenesis of the punch-drunk state remains unclear. Meningeal fibrosis and hydrocephalus from repeated small subarachnoid hemorrhages is an unlikely explanation but cannot be excluded.

Posttraumatic Hydrocephalus

This is an uncommon complication of severe head injury. Intermittent headaches, vomiting, confusion, and drowsiness are the initial manifestations. Later there are mental dullness, apathy, and psychomotor retardation, by which time the CSF pressure may have fallen to a normal level (normal-pressure hydrocephalus). Postmortem examinations have demonstrated an adhesive basilar arachnoiditis. Since a similar syndrome is observed occasionally after the rupture of a saccular aneurysm with subarachnoid hemorrhage, the same mechanisms, i.e., blocking of the aqueduct and fourth ventricle by blood clot and basilar meningeal fibrosis, may also be operative in traumatic hydrocephalus. Response to ventriculoperitoneal shunt may be dramatic. Zander and Foroglou have had extensive experience with this condition and have written informatively about it.

Posttraumatic Cognitive and Psychiatric Disorders

In all patients with cerebral concussive injury, there remains a gap in memory (traumatic amnesia) spanning a variable period from before the accident to some point following it. This gap is permanent and is filled in only by what the patient is told. In addition, as stated in the introduction to this section, some degree of impairment of higher cortical function may persist for weeks (or be permanent) after moderate to severe head injuries, even after the patient has reached the stage of forming continuous memories. During the period of deranged mentation, the memory disorder is the most prominent feature, so that the state resembles the alcoholic form of the Korsakoff amnesic state. Pfeifer-Nietleben asserts that this amnesic state is a constant feature of one phase of every prolonged traumatic mental disorder, but to the authors it merely emphasizes the ease with which memory can be tested. Such patients rarely confabulate and usually have an impaired ability to register events and information, an abnormality not ordinarily observed in patients entering the chronic phases of Korsakoff psychosis. Apart from disorientation in place and time, the head-injured patient also shows a defect in perception and in the ability to synthesize perceptual data. Judgment is impaired, sometimes severely. A perseverative tendency interferes with action and thought.

The tendency is for all such symptoms to subside slowly though not always completely, even in those in whom an accident has provoked a frank outburst of psychosis (as may happen to a manic-depressive, paranoid schizophrenic, or neurotic patient). These forms of "traumatic insanity" were carefully analyzed for the first time by Adolf Meyer.

Hysterical symptoms that develop, after head injury appear to be more common than those following injury to other parts of the body.

TREATMENT

Patients with Only Transient Unconsciousness

Patients with an uncomplicated concussive injury who have already regained consciousness by the time they are seen in a hospital pose few difficulties in management. They should not be discharged until a decision about appropriate examinations (CT scans, skull films, etc.) has been made and, if further tests are done, the results prove to be negative. The patient should not be released until the capacity to make consecutive memories has been regained and arrangements have been made for observation by the family of signs of possible delayed complications (subdural and epidural hemorrhage, intracerebral bleeding, and edema).

The patients with persistent complaints of headache, dizziness, and nervousness, the syndrome that we have designated as *posttraumatic nervous instability*, are most difficult to manage. Three subgroups can be discerned:

1. A group in which the accident has provoked anxiety or an anxious- depressive reaction. Compensation factors loom large in this group, especially for the patient whose circumstances are marginal and who is uncertain of a job and must care for dependents. Such a person is often willing to persist in illness, even if compensated at a level considerably below his earning capacity, if it provides a modicum of security. Obviously, we are often dealing here with matters that have little to do with the purely physical factors involved in cerebral trauma.

2. A group of patients in whom the *premorbid personality* was of a neurotic or depressive type. The injury to the brain is but one more factor in decompensating a tenuous social and occupational adjustment.
3. A small number of patients, obviously the more severely injured, who upon close examination are found to be *still suffering from a personality change* and *subtle impairments of cognitive function,* i.e., traumatic psychosis or dementia. The anxiety and depression reflect an awareness of their inability to cope with environmental stress.

A treatment program must be planned in accordance with the basic problem. If there is mainly an anxious depression, certain drugs such as amitriptyline or imipramine or one of the newer antidepressants, such as fluoxetine or paroxetine are often useful. Simple analgesics, such as aspirin or acetaminophen or nonsteroidal anti-inflammatory drugs, should be prescribed for the headache. Litigation should be settled as soon as possible. To delay settlement usually works to the disadvantage of the patient. Long periods of observation and waiting only reinforce the patient's worries and fears and reduce the motivation to return to work.

Severe (Concussive-Contusional) Head Injury

If the physician arrives at the scene of the accident and finds an unconscious patient, a quick examination should be made before the patient is moved. First it must be determined whether the patient is breathing and has a clear airway and obtainable pulse and blood pressure, and whether there is dangerous hemorrhage from a scalp laceration or injured viscera. Severe head injuries that arrest respiration are soon followed by cessation of cardiac function. Injuries of this magnitude are usually fatal; if resuscitative measures do not restore and sustain cardiopulmonary function within 4 to 5 min, the brain is irreparably damaged. The likelihood of a cervical fracture-dislocation, which is occasionally associated with head injury, is the reason for taking the precautions in moving the patient. Bleeding from the scalp can usually be controlled by a pressure bandage unless an artery is divided, and then a suture becomes necessary. Resuscitative measures (artificial respiration and cardiac compression) should be continued until they are taken over by ambulance personnel. All such patients should be taken to a hospital and specifically to one that has an emergency and intensive care facility prepared to deal with all aspects of serious trauma.

In the hospital the first step is to clear the airway and ensure adequate ventilation by endotracheal intubation if necessary. A careful search for other injuries must be made, particularly of the abdomen, chest, spine, and long bones. Although the hypotension that follows some injuries is a vasodepressor response and usually comes under control without pressor drugs, a large, unimpeded intravenous line should be inserted. Hypertension is a more common finding immediately after severe head injury (see further on). Initially fluid infusion should be with normal saline, avoiding the administration of excessive free water because of its adverse effect on brain edema. Persistent hypotension due to head injury alone is rare and should always raise the suspicion of a ruptured viscus with internal bleeding, extensive fractures, or trauma to the cervical cord. Cervical spine films and a cranial CT scan should be obtained en route to the intensive care unit once vital functions are stable. If films of the cervical spine are negative, there is no longer a need to immobilize the neck.

A rapid survey can now be made, with attention to the depth of coma, size of the pupils and their reaction to light, ocular movements, corneal reflexes, facial movements during grimace, swallowing, vocalization, gag reflexes, muscle tone and movements of the limbs, predominant postures, reactions to pinch, and reflexes. The scalp should be carefully inspected and any wound probed with a sterile gloved finger and cleaned. Bogginess of the temporal or postauricular area (Battle sign), bleeding from the nose or ear, and extensive conjunctival edema and hemorrhage are useful signs of an underlying skull fracture. However, it should be remembered that rupture of an eardrum or a blow to the nose may also cause bleeding from these parts. Fracture of the orbital bones may displace the eye, with resulting strabismus; fracture of the jaw results in malocclusion and discomfort on attempting to open the mouth. If urine is retained and the bladder is distended, a catheter should be inserted and kept there. Temperature, pulse, respiration, blood pressure, and state of consciousness should be checked and charted every hour.

The Glasgow Coma Scale, mentioned above, provides a practical means by which the state of impaired consciousness can be evaluated at frequent intervals, but it should not be considered a substitute for a more complete neurologic examination. It registers three aspects of neurologic functions: (1) eye opening (spontaneously, in response to command, and in response to pain); (2) verbal responsiveness (in terms of orientation, confusion, inappropriateness, and incomprehensibility); (3) motor responsiveness (to command, to a localized stimulus, flexion and extension). This scale requires little training of ward personnel. It is useful in following the course and predicting the outcome of severe head injuries (a score of less than 8 is associated with a poor prognosis). A deteriorating scale dictates a change in management.

Computed tomography is of central importance at this juncture. A sizable epidural, subdural, or intracerebral blood clot is an indication for immediate surgery. The presence of contusions, brain edema, and displacement of central structures calls for measures to monitor progression of these lesions and to control intracranial pressure. These measures are best earned out in a critical care unit.

Management of Raised Intracranial Pressure It is common practice in some hospitals to insert a monitoring device that permits continuous recording of intracranial pressure (ICP). Pressure measurements through a lumbar puncture needle do not accurately reflect the ICP and increase the risk of a cerebellar or temporal lobe herniation. Nor do the neurologic signs that constitute the Glasgow Coma Scale reflect the pressure in the cranium. Our impression is that at a minimum, monitoring ICP prevents errors in fluid administration and refines other details of management, including the appropriate use of osmotic diuretics and the correct level of hyperventilation. It also warns of evolving hemorrhage and edema. Used in this way, monitoring can be helpful. However, there are few critical data to support the routine use of ICP monitoring; certainly the patient who is only drowsy or shows only minimal mass effect on CT is not likely to benefit. The current generation of ICP monitors employs fiberoptic strain gauges that can be inserted directly into the cortex without apparent damage. While the risk of infection is low,, prolonged use may be complicated by bacterial meningitis.

The first step in lowering a presumptively high ICP is to control the factors that are known to raise the pressure, such as hypoxia, hypercarbia, hyperthermia, awkward head positions, and high mean airway pressures. If the intracranial pressure exceeds 15 to 20 mmHg, other measures should be used, such as inducing hypocarbia by controlled ventilation (maintaining PC02 ^{at} 28 to 33 mmHg) and by hyperosmolar dehydration (0.25 to 1.0 g of 20 % mannitol every 3 to 6 h or 0.75 to 1 mg/kg of furosemide) to maintain serum osmolality at 290 to 300 mosmol/L. Even if ICP monitoring is not utilized, an attempt should be made to maintain this level of osmolarity for the first days. Elevations in osmolality that are due to excessive solutes such as glucose are not useful in reducing intracranial volume because they do not provide a water gradient across the cerebral vasculature. For this reason, the measurement of serum sodium is in some ways a more accurate reflection of free water depletion. An initial sodium level of 136 to 141 meq/L is adequate. Intravenous fluids with free water should be avoided so as not to intensify cerebral edema. This poses a particular danger in children who, because of inappropriate secretion of antidiuretic hormone, easily become water- intoxicated. With this exception, however, fluid restriction is less of a concern than effecting a reduction in free water. Fluids such as 5% dextrose in water, 0.5% saline, and 5% dextrose in 0.5% saline are therefore avoided; lactated Ringer solution is permissible.

Hyperventilation is effective in reducing ICP only for a limited time since the pH of the spinal fluid equilibrates over hours and returns cerebral blood volume to its previous level. A single-step reduction in PC02 typically lowers ICP for approximately 20 to 40 min. In a few cases the effect may persist for hours.

Attempts to prolong the effect of hypocarbia and alkalosis of the spinal fluid by the administration of ammonium buffers have met with mixed success. It has been suggested that hyperventilation may even be harmful to some head-injured patients, but we believe this risk to be minimal.

If the ICP continues to rise with these measures, the outlook for survival is bleak. Hypothermia and barbiturate anesthesia to reduce ICP have been used, but relatively few patients respond to such measures. Barbiturates may lower the blood pressure, which worsens the situation, though Marshall and coworkers claim a high rate of good survival even in cases where the ICP exceeded 40 mmHg. Several large controlled studies have established that the administration of high-dose steroids does not significantly affect the clinical outcome of severe head.

The management of posttraumatic hypertension can be a difficult problem. Immediately after head injury there is a sympathoadrenal response and elevation of blood pressure which, as a general rule, recedes spontaneously in a matter of a few hours or days. Unless the blood pressure elevation is severe (above 180/95 mmHg), it can be disregarded in the early stage. In animal experiments it has been found that hypertension leads to increased perfusion of the brain and an augmentation of the edema surrounding contusions and hemorrhages. This sequence reflects a failure of autoregulatory vascular mechanisms, with resulting transudative edema in damaged areas. The control of high systemic blood pressure must be balanced against the observation that even a brief period of mild hypotension may provoke a cycle of cerebral vasodilation, increased cerebral blood volume, and elevated ICP in the form of plateau waves. It has also been observed that these plateau waves can be suppressed by inducing hypertension briefly.

Since most therapies for elevated ICP dehydrate the patient or reduce cardiac filling pressures, leading to hypotension, a middle course, of avoiding both severe hypertension and hypotension seems the best compromise. In lowering high levels of blood pressure, diuretics, beta-adrenergic blocking agents, or angiotensin converting enzyme inhibitors should be used, rather than agents that dilate the cerebral vasculature (nitroglycerin and nitroprusside, hydralazine, and many calcium channel blockers). Hypotension should be corrected by vasopressor agents such as neosynephrine. The precise level of blood pressure that requires treatment must be judged in the context of the intracranial pressure and plateau waves (the goal is to maintain normal cerebral perfusion), the patient's previous blood pressure level, and evidence of end-organ failure such as cardiac ischemia.

Treatment and Labor expertise of the patients with head injury.

Treatment of acute closed craniocerebral injury must be based on an understanding of the pathological mechanisms of formation of cavities in the skull, taking into account the severity of injuries, age, state of its vascular system, condition premorbidal body.

Patients with concussion and contusion of the brain must be hospitalized in the hospital.

In the day the patient should be carried out Lumbar puncture to clarify the height of liquor pressure and the identification of traumatic subarachnoid hemorrhage.

In response to the frequent violation in the acute period of the state of enzymatic systems, patients are encouraged to nominate proteinase inhibitors. This is helping reduce the expressivity of consciousness, the normalization of blood pressure and respiration, decreased vegetative discord.

In the present level of development of medical treatment and labor expertise patients with the investigation craniocerebral injury should be based only on the outpatient survey.

Algorithm study of patients with head trauma.

The main tasks:

definition of traumatic injury and the extent of brain damage, the gravity of the state affected.

This requires:

Find a craniocerebral injury;

Determine the status of the vital functions;

determine the level of consciousness of oppression; determine the presence, localization, head of external damage; to determine the existence or absence of clinical signs deployment of the brain, localization pathologic focus in the brain.

This requires:

determine the presence of focal symptoms fallout from the cranial nerves, motor sphere (paresis), reflexes, speech function;

determine the presence of local irritation symptoms of cerebral shells; to clarify the presence or absence seizures,

volumetric determination of the nature of brain damage. Thisrequires:

set progradient developmental symptoms;

define the growing sense of depression symptoms with the accession of the brain stem; appoint and evaluate the data Echo EG, CT, etc. methods. Identification of opportunistic diseases, evaluation of clinical data and instrumental survey, the formulation of a diagnosis, the definition of therapeutic tactics.

5. Materials of activation for students during the lecture (questions, tasks).

- The modern classification of craniocerbral trauma.
- Etiopathogenesis of craniocerbral trauma.
- Clinical effects of concussion.
- Clinical effects of brain contusion.
- Clinical effects of brain treading.
- The principles of diagnosis (diagnostic algorithms for Action) head injury.
- Brain and focal symptoms CCT
- Modern survey of patients with head trauma.
- The main types of conservative and surgical treatment.
- The principles of rehabilitation of patients after head injury.

6. The general material and methodical maintenance of the lecture:

class-rooms;
technical devices;
illustrative materials.

7. Materials for self-preparation of the students:

Questions on the topic of the lecture:

1) 1 .What are the main types of brain damage?
2) What kind of trauma is a closed head injury?
3) CaII options for skull injuries.
4) What type of injuries include a fracture of the skull base to the availability of liquor?
5) Classification of head trauma.
6) What are the clinical signs of concusion of the brain?
7) What are the pathological and clinical signs of the brain contusion?
8) What could be caused by of the brain closed head injury?
9) What additional research techniques used in patients with closed head injury?
10) In which case a closed head injury lumbar puncture is performed with great care?

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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. 6

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.

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Electronic information resources

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