MINISTRY OF HEALTH OF UKRAINE

ODESA NATIONAL MEDICAL UNIVERSITY

Faculty of Medicine №2

Department of Neurology and Neurosurgery

APPROVED BY
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TEACHING MATERIAL
FOR PRACTICAL CLASSES ON THE ACADEMIC SUBJECT

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

Approved by:
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PRACTICAL TRAINING

Practical lesson No. 1

Topic: Principles of the structure and functioning of the nervous system. The functional unit of the nervous system is a neuron. Motor system. Concept of reflex and reflex arc.

The goal: to create an idea among applicants about the extremely important role of the nervous system for the normal functioning of the body as a whole system. Applicants must have an idea of the functional purpose of reflexes, know their reflex arcs, be able to correctly interpret their lesions under the condition of correct research of reflexes. Therefore, they should master the relevant skills in order to use them in further professional activities.

Basic concepts: a significant number of diseases affecting the nervous system can be conventionally divided into two groups: organic and so-called functional.

In case of an organic disease of the nervous system, an anatomical lesion is observed in the brain or spinal cord, in the peripheral nerves and muscles connected with them, or in the autonomic nervous system. The cause of such damage can be injuries, infections, poisoning, hemorrhage, tumors, hereditary and birth defects.

Knowledge of the anatomy of the central, peripheral and autonomic nervous system is necessary for specialists in various medical disciplines: pediatricians, ophthalmologists, otolaryngologists, surgeons, therapists, etc. A change in normal reflexes and the appearance of pathological ones occurs in many diseases of the nervous system; the ability to examine reflexes, to correctly interpret violations of normal and the appearance of pathological reflexes affects the accuracy of diagnosis and the adequacy of therapy.

Equipment: classroom, furniture, equipment.

Plan:

1. 1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
    - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units).

Lesson content

The main stages of phylo- and ontogenesis of the nervous system. Structural and functional unit of the nervous system. The main anatomical and topographic divisions of the nervous system: cerebral hemispheres, subcortical nodes, brain stem, cerebellum, spinal cord, roots, spinal ganglia, plexuses, peripheral nerves. The functional unit of the nervous system is a neuron. Types of neurons, their functional significance. Neuroglia, its functional significance. Concept of reflex and reflex arc, conditional and unconditional reflexes, levels of closure of skin, tendon and periosteal reflexes. Anatomical features and neurophysiology of the system of voluntary movements, extrapyramidal system and cerebellum.

The main stages of phylo- and ontogenesis of the nervous system.

In the process of phylogenesis, the nervous system goes through a number of main stages:

I - an asynaptic network consisting of nerve cells, the processes of which are connected to each other in different directions (hydra).

II - nodal nervous system, in which nerve cells converge into separate clusters (invertebrates - worms, mollusks). In humans, it is preserved in the structure of the central nervous system.

III - the tubular nervous system is observed in vertebrates. In humans, the spinal cord has a similar structure.

Since most of the sense organs arise at the front end of the animal's body, it is separated in the form of a head and brain (cephalization).

In ontogenesis, the nervous system develops from an ectodermal sheet - a medullary tube.

The membranes covering the brain and spinal cord are formed from the mesoderm around the brain tube. The brain is formed from brain bubbles that arise as a result of uneven growth of the anterior parts of the medullary tube.

The nervous system unites a number of departments and structures, which 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 (JH 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 determine the nature of the irritation and transform it into a nerve impulse. The receptor department is the first level of analytical and synthetic activity of the nervous system, on the basis of which reactions-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 brain stem - the nuclei of the cranial nerves, as well as their roots. In the spinal cord and trunk there is white matter - 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 reflex. 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 structures, the cerebral cortex and subcortical structures are connected to the environment.

3. The subcortical integrative department includes subcortical (basal) nuclei: caudate nucleus, shell, pale layer, thalamus. It contains afferent and efferent channels of communication that connect 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. With the help of a fine device for processing signals from the surrounding and internal environment of the body, it ensures the selection of the most important information and prepares it for reception by the cortex. Other information is directed to the nuclei of the reticular formation, where it is integrated, and then enters the cortex via ascending pathways, maintaining its tone.

4. The cortical part of the brain is the third level of analysis and synthesis. The cortex receives signals of varying degrees of complexity. Information decoding, higher analysis and synthesis of nerve impulses are carried out here. The highest form of analytical and synthetic activity of the human brain ensures thinking and consciousness.

Structural and functional unit of the nervous system. The functional unit of the nervous system is a neuron. Types of neurons, their functional significance. Neuroglia, its functional significance. Neuron - (from the Greek Neuron - nerve), a neuron, a nerve cell, the main functional and structural unit of the nervous system, receives signals coming from receptors. The neuron processes them and transmits them in the form of nerve impulses to effector nerve endings that control the activity of executive organs (muscles, gland cells). The formation of a neuron occurs during the embryonic development of the nervous system: at the stage of the neural tube, neuroblasts develop, which then differentiate into neurons. In the process of differentiation, specialized parts of the neuron are formed, which ensure the performance of its functions. To perceive information, branched outgrowths - dendrites, which have selective sensitivity to certain signals and have on their surface the so-called receptor membrane. The processes of local excitation and inhibition from the receptor membrane, summing up, affect the trigger (trigger) area - the most excitable area of the surface membrane of the neuron, which is the place of origin

(generation) of bioelectric potentials. For their transmission, a long process serves as an axon, or an axial cylinder covered with a conductive membrane. Having reached the final sections of the axon, the impulse excites the secretory membrane, as a result of which a physiologically active substance - a mediator or neurohormone - is secreted from the nerve endings. In addition to the structures associated with the performance of specific functions, each neuron, like others. living cells, has a nucleus, which together with perinuclear cytoplasm forms the cell body. Macromolecules are synthesized here, some of which are transported through the axoplasm (cytoplasm of the axon) to the nerve endings. The structure, size and shape of a neuron vary greatly. Neurons of the cortex of the large hemispheres of the brain, the cerebellum, and some others have a complex structure. departments of the center, nervous system. Vertebrate brains are characterized by multipolar neurons. Neuroglia, or simply glia, is a collection of auxiliary cells of nervous tissue. Makes up about 40% of the volume of the central nervous system. The number of glial cells is on average 10-50 times more than neurons. The term was introduced in 1846 by Rudolf Virchow. Glial cells have common functions and, in part, origin (the exception is microglia). They constitute a specific microenvironment for neurons, providing conditions for the generation and transmission of nerve impulses, as well as carrying out part of the metabolic processes of the neuron itself. Neuroglia perform supporting, trophic, secretory, delimiting and protective functions.

Microglial cells, although included in the concept of "glia", are not actually nervous tissue, as they are of mesodermal origin. They are small cells scattered throughout the white and gray matter of the brain and capable of phagocytosis.

Reflex (from the Latin "reflex" - reflection) - the body's reaction to changes in the external or internal environment, carried out through the mediation of the central nervous system in response to irritation of receptors.

Types of reflexes.

All reflex acts of a whole organism are divided into unconditional and conditioned reflexes. Unconditioned reflexes are inherited, they are characteristic of every biological species; their arches are formed before birth and remain normal throughout life. However, they can change under the influence of the disease. Conditioned reflexes arise during individual development and accumulation of new skills. Production of new temporary connections depends on changes in environmental conditions. Conditioned reflexes are formed on the basis of unconditional ones and with the participation of higher brain departments. Unconditioned and conditioned reflexes can be classified into different groups according to a number of features. By biological meaning: nutritional, defensive, sexual, orienting, late-tonic (reflexes of body position in space), locomotor (reflexes of body movement in space) By location of receptors, the irritation of which is caused by this reflex act: exteroreceptive reflex – caused by irritation of receptors on the outer surface body, viscero- or interoreceptive reflex - occurs when receptors of internal organs and vessels are irritated, proprioreceptive reflex - occurs when receptors of skeletal muscles, joints, tendons are irritated.

According to the location of the neurons involved in the reflex, spinal reflexes are distinguished - neurons are located in the spinal cord, bulbar reflexes - are carried out with the obligatory participation of neurons of the medulla oblongata, mesencephalic reflexes - are carried out with the participation of midbrain neurons, diencephalic reflexes - neurons are involved diencephalon, cortical reflexes - are carried out with the participation of neurons of the cortex of the large hemispheres of the brain.

In the reflex acts carried out with the participation of neurons located in the higher departments of the central nervous system, neurons located in the lower departments - in the intermediate, middle, oblong and spinal cord - always participate. On the other hand, with reflexes that are carried out by the spinal or medulla oblongata, middle or intermediate brain, nerve impulses reach the higher departments of the central nervous system. Thus, this classification of reflex acts is somewhat conditional. According to the nature of the corresponding reaction, depending on which organs are involved in it, motor or motor reflexes - muscles serve as the

executive organ; secretory reflexes - end with gland secretion; vasomotor reflexes - manifested in the narrowing or expansion of blood vessels.

Any reflex in the body is carried out with the help of a reflex arc. The reflex arc is the path along which the excitation (signal) passes from the receptor to the executive organ. The structural basis of the reflex arc is formed by neural chains consisting of receptor, insertion and effector neurons. It is these neurons and their processes that form the path along which nerve impulses from the receptor are transmitted to the executive body during the implementation of any reflex. In the peripheral nervous system, there are reflex arcs (neuron chains) of the somatic nervous system, which innervate the skeletal muscles, and the autonomic nervous system, which innervate internal organs: the heart, stomach, intestines, kidneys, liver, etc.

The reflex arc consists of five departments: receptors that perceive irritation and respond to it with excitement. Receptors are located in the skin, in all internal organs, clusters of receptors form sense organs (eye, ear, etc.), a sensitive (afferent) nerve fiber that transmits excitation to the nerve center, where excitation is switched from sensory neurons to motor ones. The centers of most motor reflexes are located in the spinal cord. In the brain there are centers of complex reflexes, such as protective, food, orienting, etc. In the nerve center there is a synaptic connection between a sensory and a motor neuron, a motor (efferent) nerve fiber, which carries excitation from the central nervous system to the working body; A centrifugal fiber is a long process of a motor neuron. A motor neuron is called a neuron, the process of which reaches the working organ and transmits a signal to it from the center. An effector is a working organ that performs a reaction in response to receptor stimulation. Effectors can be muscles, or gland cells that secrete under the influence of nervous excitement, or other organs.

The simplest reflex arc can be schematically represented as formed by only two neurons: receptor and effector, between which there is one synapse. Such a reflex arc is called two-neuron and monosynaptic. Monosynaptic reflex arcs are quite rare. An example of them can be the arc of the myotatic reflex. In most cases, reflex arcs include not two, but a larger number of neurons: receptor, one or more insertion and effector. Such reflex arcs are called multineuronal and polysynaptic. An example of a polysynaptic reflex arc is the flexion reflex of a limb in response to a painful stimulus.

The reflex arc of the somatic nervous system on the way from the central nervous system to the skeletal muscle is not interrupted anywhere, unlike the reflex arc of the autonomic nervous system, which on the way from the central nervous system to the innervated organ is necessarily interrupted with the formation of a synapse - the autonomic ganglion. Autonomic ganglia, depending on localization, can be divided into three groups: vertebral (vertebral) ganglia - belong to the sympathetic nervous system. They are located on both sides of the spine, forming two border trunks (they are also called sympathetic chains), the prevertebral ganglia are located at a great distance from the spine, at the same time they are at some distance from the organs innervated by them. Prevertebral ganglia include ciliary node, upper and middle cervical sympathetic nodes, solar plexus. Intraorgan ganglia are located in internal organs: in the muscular walls of the heart, bronchi, middle and lower third of the esophagus, stomach, intestines, gall bladder, urinary bladder, as well as in the glands of external and internal secretion. Parasympathetic fibers are interrupted on the cells of these ganglia. Such a difference between the somatic and vegetative reflex arc is due to the anatomical structure of the nerve fibers that make up the neural chain, and the speed of nerve impulse conduction along them.

For the implementation of any reflex, the integrity of all links of the reflex arc is necessary. Violation of at least one of them leads to the disappearance of the reflex.

Methods of research of surface reflexes: skin (abdominal, plantar, cremasteric, anal), mucous membranes (corneal, conjunctival, pharyngeal, soft palate).

Cutaneous reflexes are caused by line irritation of the skin with a blunt needle.

Abdominal reflexes are caused by rapid stroke irritation of the abdominal skin from the periphery to the middle:

1) upper (Th7-Th8) - below the costal arch,

2) middle (Th9-Th10) - at the level of the navel,
3) lower (Th11-Th12) - above the Pupart ligament.

The plantar reflex (L5-S1) is caused by line irritation of the outer edge of the sole and consists in plantar flexion of all toes.

The cremasteric reflex (L1-L2) is caused by line irritation of the skin of the inner surface of the upper third of the thigh from the bottom up and consists in raising the testicle.

The anal reflex (S4-S5) is caused by irritation of the skin near the anus, and the anal sphincter is shortened in response.

Reflexes from mucous membranes are caused by:

By carefully touching a thin strip of paper or cotton wool to the cornea above the iris (corneal) or to the conjunctiva (conjunctival), the eye closes in response, the reflex arc is formed by the V and VII pairs of cranial nerves.

The pharyngeal reflex and the palatal reflex are caused by irritation of the mucous membrane with a spatula, respectively of the back wall of the pharynx and the soft palate, in response there are vomiting movements (pharyngeal reflex) or lifting of the soft palate and tongue (palatal reflex), the reflex arc is formed by the IX and X pairs of cranial nerves

The technique of studying deep reflexes: tendon (flexor ulnar and extensor ulnar, knee, Achilles) and periosteal (superior, carporadial).

Tendon reflexes.

Elbow flexion reflex from the biceps (C5—C6) are caused by a short blow of the hammer on the tendon of the biceps muscle of the shoulder in the elbow bend. In response, the forearm bends at the elbow joint

The elbow extensor reflex from the triceps (C7-C8) is caused by a hammer blow on the tendon of the triceps muscle of the shoulder above the ulnar process.

The knee reflex (L3-L4) is caused by a hammer blow on the tendon of the quadriceps muscle of the thigh, on the patellar ligament. In response, the leg flexes.

The Achilles reflex (S1-S2) occurs as a result of a hammer blow on the Achilles tendon, accompanied by plantar flexion of the foot.

Tendon reflexes are evaluated according to a 5-point international scale (0 is also taken into account):

0 points - no reflex
1 point – reduced reflex
2 points - a normal reflex
3 points - increased reflex
4 points - clonus

Periosteal reflexes:

The superbrow reflex is caused by hitting the brow arch with a hammer. The motor reaction consists in closing the eyelids. The reflex arc is formed by the trigeminal and facial nerves.

The carpal-radial reflex (C5-C8) is caused by a hammer blow on the styloid process of the radius. In response, there is flexion of the forearm in the elbow joint, flexion of the fingers and pronation of the hand, not all components of the response may be observed.

Methods of researching pathological reflexes: foot-extensor (Babinsky, Oppenheim, Gordon, Schaeffer, Pusep), flexor and their hand analogues (Rossolimo, Zhukovsky, Bekhterev). Foot extensor reflexes with extension of the big toe are early signs of organic damage to the pyramidal system.

Babinski's reflex - in case of line irritation of the outer edge of the sole, there is an extension of the big toe, which is sometimes supplemented by simultaneous fan-like separation of all other toes (fan sign). The reflex is observed normally in children up to 1-1.5 years of age, with the formation of gait it disappears and the plantar reflex is triggered.

A similar movement of the fingers can also be caused by pressing with the thumb on the front surface of the tibial crest from top to bottom (Oppenheim's reflex).

A similar movement of the fingers can be caused by squeezing the calf muscle (Gordon's reflex) or the heel tendon (Sheffer's reflex) with the hand.

Flexion foot reflexes. The Rossolimo reflex is caused by intermittent blows of a hammer or fingertips on the plantar surface of the distal phalanges of the patient's toes. There is rapid plantar flexion of II-V toes. A similar reaction occurs with light blows of a hammer on the back surface of the patient's foot (Bekhterev's reflex) and on the middle of the sole near the base of the fingers (Zhukovsky's reflex).

Study of reflexes of oral automatism (subcortical): sucking, proboscis, distance-oral, palmar, chin.

Reflexes of oral automatism are automatic, involuntary movements performed by the mouth muscles in response to mechanical irritation of various parts of the face. In the case of tapping with a hammer on the root of the nose, lip stretching occurs (naso-lip reflex). The same reaction is observed as a result of a light blow of the hammer on the upper or lower lips (oral reflex) and when the hammer approaches the patient's mouth (distance-oral reflex). The palmar-chin reflex of Marinescu-Radovich is caused by line irritation of the palmar area of the thumb (thenar) elevation. In response, there is a contraction of the chin muscles. Reflexes are normally observed in infants.

Pyramid system.

According to the traditional point of view, the main mechanism that implements arbitrary movements; starts from the Betz motor cells located in the V layer of the motor cortex (4th field), continues in the form of a corticospinal tract, or pyramidal tract, which passes to the opposite side in the area of the pyramids and ends at the motoneurons of the spinal cord (at 2- mu neuron of the pyramidal pathway), innervates the corresponding group of muscles. New data of modern anatomy and physiology were added to these ideas about the pyramidal system as the main efferent mechanism of voluntary movements.

First, not only the 4th field is motor. This is the primary motor field of the cortex, different areas of which are associated with the innervation of different groups of muscles. The primary motor field of the cortex of the large hemispheres is characterized by the strong development of the V layer, which contains the largest cells of the human brain (Betz cells). These cells ("giant pyramids") have a specific structure and have the longest axon in the human nervous system (its length can reach two meters), which ends at the motoneurons of the spinal cord. Motor cells of the pyramidal type are found not only in the 4th field, but also in the 6th and 8th fields of the precentral cortex, and in the 2nd, 1st and even in the 3rd fields of the postcentral cortex (and in a number of other areas of the cortex). So, the pyramidal path begins not only from the 4th field, as it was previously assumed, but from much larger areas of the cortex of the large hemispheres. According to P. Duus, only 40% of all fibers of the pyramidal path begin in the 4th field, about 20% - in the postcentral gyrus; the rest - in the premotor zone of the cortex of the large hemispheres. Irritation of the 4th field causes contraction of the corresponding muscle groups on the opposite side of the body.

Secondly, it was established that the pyramidal path contains fibers of different types (by diameter and degree of myelination). Well-myelinated fibers make up no more than 10% of all pyramidal fibers that go from the cortex to the periphery. With their help, the phasic (directly executive) component of voluntary movements is carried out. The vast majority of weakly myelinated fibers of the pyramidal pathway probably have other functions and primarily regulate the tonic (background, tuning) components of voluntary movements.

Thirdly, if earlier it was assumed that there is a single pyramidal, or cortico-spinal, path (lateral), which goes with a cross in the pyramidal zone from the cortex of the large hemispheres to the motoneurons of the spinal cord, now another cortico-spinal path has been identified (ventral), which goes without crossing as part of the pyramids on the same side. These two pathways have different functional significance. Finally, the pyramidal pathway does not end directly on the motoneurons located in the anterior horns of the spinal cord, as was previously believed, but mainly on the intermediate (or insertion) neurons, with the help of which the excitability of the main motoneurons is modulated and thus the influence on the final result is revealed - voluntary

movements . In addition to the 4th motor field, a number of motor zones have also been identified in humans, when stimulated, motor effects also occur. These zones, called additional motor zones, were described by H. Jasper, U. Penfield and other physiologists.

There are two main supplementary motor areas of the cortex. One of them is located along the edge of the Sylvian furrow; its stimulation causes movements of arms and legs (both ipsilateral and contralateral). The other is located on the inner medial surface of the hemispheres in front of the motor zone, in the precentral parts of the brain. Irritation of this zone also leads to various motor acts. It is important to note that when additional motor zones are irritated, not elementary contractions of individual muscle groups occur, but integral complex movements, which speaks of their special functional importance. Other additional motor areas of the cortex are also described. All these data indicate that modern knowledge about the cortical organization of voluntary movements is still far from complete.

In addition to the motor areas of the cortex of the large hemispheres, the irritation of which causes movements, there are also such areas of the cortex, the irritation of which stops the movement that has already begun (suppressive areas of the cortex). They are located in front of the 4th field (field 4s) on the border of the 4th and 6th fields; ahead of the 8th field (field 8s); behind the 2nd field (field 2s) and in front of the 19th field (field 19s). The 24s field is mainly located on the inner surface of the hemisphere. Fields 8s, 4s, 2s and 19s are narrow strips demarcating the main areas of the cortex associated with the projections of the nuclei of the optic tubercle to the cortex of the large hemispheres. As is known, the posterior parts of the cortex (fields 17, 18, 19) are the projection zone of the lateral geniculate body; sensorimotor region - projection zone of ventral thalamic nuclei; the prefrontal region is the projection zone of the DM-nucleus of the visual tubercle. Thus, the inhibitory strips of the cortex demarcate the spheres of influence of different relay nuclei of the thalamus. There are also special aversive zones in the cortex of the large hemispheres. These areas of the cortex are well known to neurosurgeons and neuropathologists. Their irritation (by electric current or a painful process) causes aversive epileptic seizures (beginning with turning the trunk, eyes, head, arms and legs in the direction opposite to the location of the exciting agent). Epilepsy with seizures of this type is known as "Jackson's epilepsy." There are two aversive zones of the cortex: premotor and parietal-occipital (fields 6, 8 and 19 on the border with the 37th and 39th fields). It is assumed that these fields of the cortex participate in complex reactions associated with attention to a stimulus, that is, in the organization of complex motor acts that mediate attention to a certain stimulus.

The extrapyramidal system is the second efferent mechanism for the implementation of voluntary movements and actions. All motor pathways that do not pass through the pyramids of the medulla are called the extrapyramidal system. The pyramidal system is not the only mechanism by which voluntary motor acts are performed. The extrapyramidal system is older than the pyramidal system. It is very complex in its composition. To this day, there are disputes about which structures to attribute to the extrapyramidal system and, most importantly, what are the functions of these structures. Cortical and subcortical divisions are distinguished in the extrapyramidal system. The cortical department of the extrapyramidal system includes the same fields that are included in the cortical nucleus of the motor analyzer. These are the 6th and 8th, as well as the 1st and 2nd fields, that is, the sensorimotor area of the cortex. The exception is the 4th field, which is the cortical link of only the pyramid system. The 6th and 8th fields by structure belong to the motor-type fields, in the III and V layers of these fields there are pyramidal motor cells, but they are smaller than Betz cells. The structure of the subcortical part of the extrapyramidal system is quite complex. It consists of a number of formations. First of all, this is the striopallidary system - the central group of formations inside the extrapyramidal system, which includes the caudate nucleus, shell and pale layer (or pallidum). This system of basal nuclei is located inside the white matter (deep in the premotor zone of the brain) and is characterized by complex motor functions. It should be noted that, according to modern data, the striopallidary system is involved in the implementation of not only motor, but also a number of other complex functions. The extrapyramidal system also includes other structures: cingulate cortex, substantia nigra, Lewis

body, anterior ventral, intralaminar nuclei of the thalamus, subthalamic nuclei, red nucleus, cerebellum, various departments of the reticular formation, reticular structures of the spinal cord. The ultimate instance of extrapyramidal influences are the same motoneurons of the spinal cord, to which the impulses of the pyramidal system are addressed.

There is no clear anatomical boundary between the pyramidal and extrapyramidal systems. They are separated anatomically only in the area of the pyramids, in the medulla oblongata. However, the functional differences between these systems are quite distinct. And they are especially clearly manifested in the clinic of local lesions of the brain.

Functions of the extrapyramidal system:

The extrapyramidal system is the material substance of complex reflex movements

1. Prepares muscles, muscle tone for performing movements with the least expenditure of energy and time;

a) creates preliminary readiness of the muscular apparatus for voluntary movements;

b) creates the most favorable posture for performing the planned action;

c) provides the necessary ratio of muscle tone of agonists and antagonists;

d) gives smoothness and dimensionality to our voluntary movements;

e) the extrapyramidal system ensures the "tuning" of the motor apparatus, their readiness for action and creates the necessary muscle tone for rapid movements;

f) the extrapyramidal system automatically creates the background, preliminary readiness, on which fast, accurate, differentiated movements are carried out due to the activity of the cortex.

2. With the help of the extrapyramidal system, the following types of movements are formed:

3. Protective movements are formed, or they are called start reflexes (shivering, turning the head and eyes, etc.).

4. Reactive movements - our voluntary movements are possible thanks to the extrapyramidal system.

Reactive movements accompany all our other movements (facial expressions, gestures).

5. The extrapyramidal system forms muscle tone.

For any movement, it is necessary to prepare a pose-muscle tone. Correct posture of the body gives appearance to the movements. For example: artists often widely use mimicry, the pose of their characters.

II. The extrapyramidal system takes part in the execution of involuntary (automated) movements.

III. Influences external manifestations of emotional and volitional acts.

Our facial expressions reflect the state of movements, mood, facial reactions, suffering face, etc. This is done thanks to the connections of the striopallidar system with the nuclei of the hypothalamic part of the diencephalon.

IU Affects the cerebral cortex, activates it.

The extrapyramidal system together with the pyramidal system carries out tonic and phasic activity. This is a mandatory condition of movement. Disconnection of these mechanisms leads to the development of hyperkinesis - involuntary movements, changes in muscle tone.

When subcortical nodes or their pathways are affected, dysfunction of the extrapyramidal system develops. The clinical picture of this pathology depends on which part of the striopallidary system will be more affected, if the pallidum is affected, the inhibitory effect of the striatum will be excessive, and vice versa, if the striatum is affected, the activating role of the pallidum will be excessive, without control and inhibition.

In this regard, the extrapyramidal system, in addition to anatomical, phylogenetic and histological division, is divided into different syndromes depending on whether it is the striatum or the pallidum.

Lesions of the striopallidary system are manifested mainly by changes in muscle tone (rigidity or hypotonia) and changes in movement activity (hypokinesis - poor movement or hyperkinesis - forced movements).

Changes in muscle tone and movement activity can be combined. Thus, hypokinesis is often combined with high muscle tone - muscle stiffness, and hyperkinesis is often combined with muscle hypotonia.

When pallidum is damaged, hypokinesia and hypomimia occur, striatum - involuntary movements occur - hyperkinesis. Therefore, movement disorders with damage to the extrapyramidal system are believed to be the result of dysfunction of the cerebral cortex and pyramidal pathways in connection with a violation of the regulatory influence of the formations of the extrapyramidal system on the cortex of the large hemispheres of the brain.

Methods of detecting extrapyramidal disorders (hyperkinetic-hypotonic and hypokinetic-hypertonic syndromes).

Visually assess the presence of involuntary movements (hyperkinesis in hyperkinetic-hypotonic syndrome), the speed of voluntary movements (slowed down in hypokinetic-hypertonic syndrome), the patient's posture, facial expressions, expressiveness of speech.

Muscle tone is evaluated by palpating them in a state of rest and complete relaxation, as well as by examining passive movements in the joints under conditions of complete muscle relaxation. To detect hidden extrapyramidal hypertension, the Neuke-Ganev test is performed - during passive movement in the elbow joint, the patient is asked to simultaneously raise the leg (bend at the hip joint), while an increase in tone in the muscles of the upper limb is observed.

The main importance of the cerebellum is to supplement and correct the activity of other motor centers. Each of the three longitudinal zones of the cerebellum has its own functions.

The cerebellar worm controls posture, tone, which support body movements and balance.

The intermediate part of the cerebellum is involved in the mutual coordination of late and purposeful movements and in the correction of movements.

Unlike other parts of the cerebellum, signals do not come directly from the peripheral organs, but from the associative zones of the cerebral cortex. Information about the intention of the movement, transmitted by afferent pathways to the motor systems, is transformed in the cerebellar hemispheres and its dentate nucleus into a movement program, which is sent to the motor areas of the cortex mainly through the nuclei of the thalamus. After that, movement becomes possible. In this way, very fast movements are carried out, which cannot be controlled through somatosensory feedback. Thus, the cerebellum is a large part of the brain that is part of the brain. It consists of the superficially located cortex of the cerebellum and nuclei lying in the depth. The cerebellar cortex is divided into lobes by furrows, its surface is equal to half the surface of the cerebral cortex . The information arriving in the cerebellum is first addressed to the cells of the cortex, from there it is transmitted to the nuclei of the cerebellum and only then to other parts of the brain. The functional value of the cerebellum is to ensure the correspondence of movements to incoming sensory information. It plays a leading role in maintaining body balance and coordination of movements. According to research conducted in recent years using invasive methods, the cerebellum is involved in cognitive processes. Lesions of the cerebellum lead to impaired muscle tone, balance, inability to perform complex and subtle movements, changes in speech and handwriting. Cerebellar function examination technique. Checking the coordination of movements. Asynergy is detected using the Babinsky test: the patient, lying on his back with his arms crossed on his chest, is offered to sit down without the help of his hands. A patient with asynergia will not be able to sit down, because he does not have a synergistic contraction of the gluteal muscles, which fix the legs and pelvis to the surface of the couch. The Stuart-Holmes test, or the symptom of the absence of a return impulse: the patient is asked to clench the hand into a fist, bend the arm at the elbow joint and resist the doctor who is trying to unbend the patient's forearm. During the sudden termination of the countermeasure by the doctor, the patient's fist hits the chest due to the lack of timely contraction of the antagonist muscles.

To detect static ataxia, Romberg's position is used: the patient stands with closed legs, arms extended forward, fingers spread, the test is performed with open and closed eyes. To detect minor violations, a complicated Romberg pose is used - the feet are on the same line, the toe touches the

heel. Finger-nose test: the patient stands with closed legs, arms extended forward, fingers spread, the patient should touch the tip of the nose with the index finger of the outstretched hand with closed eyes. Heel-knee test: the patient lies on his back, with his eyes closed, the heel of one leg touches the knee joint of the other leg, then he accurately runs the heel along the ridge of the tibial ridge from the knee to the foot.

Materials for self-control of training quality

Tests

1) Subcortical nodes are:
Answer options:
a) front horns;
b) front ropes;
c) visual hill;
d) hemispheres of the brain;
e) Varoliiv bridge.
Correct answer: c)

2) The spinal cord includes:
Answer options:
a) side pillars;
b) nerve plexuses;
c) side horns;
d) Varoliiv bridge.
e) medulla oblongata.
Correct answers: a, c)

3) What structures belong to the brain?
Answer options:
a) cerebellum;
b) front horns;
c) side horns;
d) spinal roots;
e) spinal ganglia.
Correct answer: a)

4) What structures belong to the spinal cord?
Answer options:
a) cerebellum;
b) front horns;
c) side horns;
d) spinal roots;
e) spinal ganglia.
Correct answers: b, c)

Tasks for self-control :

1. The patient has no right Achilles reflex. What nerve is affected?
Correct answer: right sciatic nerve.
2. The patient has no left knee reflex. What nerve is affected?
Correct answer: left femoral nerve.
3. A patient with thoracic spondylogenic myelopathy has increased knee and Achilles reflexes on both sides, pathological reflexes have appeared. Why did this happen and how were the reflex arcs of these reflexes affected?
Correct answer: reflex arcs are not affected. A lesion of the pyramidal tracts developed on both

sides at the thoracic level.
4. A seven-year-old girl has various pallors of the skin accompanied by tachycardia, a drop in blood pressure, tooth decay, and hyperhidrosis. Determine the localization of the process.
Correct answer: hypothalamic area.
5. A fifteen-year-old boy noticed that he could not feel the details of an object with his right hand. Determine the localization of the process.
Correct answer: parietal lobes of the brain.
6. The child does not recognize the objects that are shown to him. Determine which structures are affected.
Correct answer: occipital lobe of the brain.

Materials for classroom self-training:
List of educational practical tasks:
1. To study the functions of the frontal lobe on the right and left.
2. To study the functions of the temporal lobes.
3. To study the functions of the occipital lobes.
4. To study the functions of the parietal lobes.
5. Check autonomic skin reflexes.
6. To study corneal and conjunctival reflexes.
7. To study pharyngeal and palatal reflexes.
8. To study cranial reflexes.
9. To study the cremasteric and plantar reflexes.
10. To study flexor-elbow, extensor-elbow and carpo-radial reflexes on the hands.
11. To study the knee and Achilles reflexes on the legs.

Instructional material for mastering professional skills
1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.
3. Prescribe adequate treatment and examination of patients.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 2

Topic: Voluntary movements and their disorders. Pyramid system. Cortico-nuclear and cortico-spinal pathways.

Purpose: to acquaint students with one of the most common pathologies of the nervous system - a movement disorder - on the correct diagnosis and treatment of which depends the ability to work and the life of a person.

Basic concepts:

Movement is a universal manifestation of vital activity, which provides the possibility of active interaction of both the constituent parts of the body and the whole organism with the environment. Special arbitrary movements are carried out with the direct participation of the cerebral cortex. In humans and higher animals, the implementation of arbitrary movements is associated with a special department of the nervous system - the pyramidal system. Damage to the latter at any level leads to paralysis with subsequent long-term disability of patients.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
    - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

The pyramidal system (synonymous with the pyramidal path) is a set of long efferent projection fibers of the motor analyzer, originating mainly in the anterior central gyrus of the

cerebral cortex, ending on the motor cells of the anterior horns of the spinal cord and on the cells of the motor nuclei of the cranial nerves that carry out voluntary movements

The pyramidal path starts from the cortex, from the giant Betz pyramidal cells of layer V of field 4 as part of the radiant crown, occupying the front two-thirds of the posterior femur and the knee of the internal capsule of the brain. Then it passes through the middle third of the basal part of the peduncle of the brain into the bridge. In the medulla oblongata, the pyramidal system forms compact bundles (pyramids), some of the fibers of which cross to the opposite side at the level of the border between the medulla oblongata and the spinal cord (intersection of the pyramids). In the brainstem, fibers that cross slightly above or at the level of these nuclei depart from the pyramidal system to the nuclei of the facial and hypoglossal nerves and to the motor nuclei of the trigeminal nerve. In the spinal cord, the crossed fibers of the pyramidal system occupy the back part of the lateral cords, uncrossed - the front cords of the spinal cord. The motor analyzer receives afferent impulses from muscles, joints and ligaments. These impulses pass to the cerebral cortex through the optic tubercle, from where they approach the posterior central gyrus.

In the anterior and posterior central gyri, the distribution of cortical zones for individual muscles is observed, which coincides with the distribution of the corresponding muscles of the body. Irritation of the cortical part of the pyramidal system, for example by a scar of the brain membrane, causes seizures of Jacksonian epilepsy. Paralysis or paresis, as well as pyramidal symptoms (increased tendons and the appearance of pathological reflexes, increased muscle tone of paralyzed muscles) appear in the brain when the function of the pyramidal system fails. Damage to the cortico-nuclear pathways of the facial nerve leads to central paresis of this nerve. A lesion of the pyramidal system in the region of the internal capsule leads to hemiplegia. Damage to the pyramidal system in the brain stem results in a combination of pyramidal symptoms on the opposite side with symptoms of damage to the cranial nerve nuclei on the affected side - alternating syndromes.

The pyramidal system (PS) is a system of long efferent projection fibers of the motor analyzer that originate in the front central gyrus of the cerebral cortex (cytoarchitectonic fields 4) and partly from other fields and areas. His name p.s. received from the so-called pyramids of the medulla oblongata, formed on its ventral surface by the pyramidal tracts passing there. In lower vertebrates p.s. absent It appears only in mammals, and its importance in evolution gradually increases. In a person p.s. reaches its maximum development, and its fibers in the spinal cord occupy about 30% of the cross-sectional area (21.1% in higher monkeys, 6.7% in dogs). On behalf of p.s. in the cortex of the brain there is a core of the motor analyzer. In lower mammals, the nucleus of the motor analyzer is not spatially separated from the nucleus of the skin analyzer and has the IV granular layer (a sign of the sensitive cortex). These cores mutually overlap, becoming more and more separated from each other as the phylogeny develops. They are the most isolated in humans, although they also have remnants of overlap as fields and 3/4 5. In ontogenesis, the cortical nucleus of the motor analyzer differentiates early - at the beginning of the second half of fetal life. Before birth, field 4 preserves the IV granular layer, which is a repetition in the ontogeny of features that appear in the early stages of the phylogeny of mammals. Myelin coating of the nerve fibers of the pyramidal system is carried out during the 1st year of life. In an adult, the main cortical representation of p.s. corresponds to cytoarchitectonic fields 4 and 6 of the anterior central gyrus of the brain. Field 4 is characterized by the presence of Betz's giant pyramidal cells in layer V, agranular (absence of granular layers) and a large width of the cortex (about 3.5 mm). Field 6 has a similar structure but lacks Betz's giant pyramidal cells. From these fields, from Betz's giant pyramidal cells and from other pyramidal cells of layers V and III, and according to modern data, from other fields and regions of the cerebral cortex, the pyramidal tract originates. It is formed by fibers of a caliber from 1 to 8 μm or more, which in the white matter of the cerebral hemispheres, in a radiant crown, converge towards the internal capsule, where, forming a compact bundle, they occupy the front two-thirds of its posterior thigh and knee.

Then the fibers of the pyramidal system go in the middle third of the basal part of the brain stem. Entering the pons, they break up into separate small bundles that pass between transversely

located fibers of the frontal-pons-cerebellar pathway and the own nuclei of the pons. In the medulla, the fibers of p.s. gather again in a compact bundle and form pyramids. Here, most of the fibers go to the opposite side, forming the intersection of the pyramids. In the brain stem, the fibers to the motor cranial nerves (cortico-nuclear; tractus corticonuclearis) and to the anterior horns of the spinal cord (cortico-spinal, tractus corticospinalis) go together to the lower edge of the superior olive. Then the cortico-nuclear pathway gradually gives its fibers to the motor nuclei of the facial, hyoid, trigeminal and vagus nerves. These fibers cross at the level of the nuclei or directly above them. Cortico-spinal fibers descend into the spinal cord, where the crossed fibers of the p.s. are concentrated in the side column, occupying its rear part, and the uncrossed ones pass in the front column. Ending on the motor cells of the anterior horns (or intercalary cells) of the spinal cord, the fibers of the p.s., gradually exhausting, reach the sacral division of the spinal cord. The number of fibers p.s. exceeds 1 million. In addition to motor, there are also vegetative fibers. The cortical part of the pyramidal system, or the motor zone of the cerebral cortex, is the core of the motor analyzer. The analyzer, or afferent, nature of this nucleus is confirmed by the afferent fibers going to it from the optic tubercle. As established, the fibers of p.s. originate from a wider area of the cerebral cortex than the anterior central gyrus and p.s. closely related to the extrapyramidal system, especially in the cortical department. Therefore, with the most diverse localizations of brain lesions, the p.s. usually suffers to one degree or another.

Physiologically p.s. is a system that carries out voluntary movements, although the latter are ultimately the result of the activity of the whole brain. In the anterior central gyrus, there is a somatotopic distribution of cortical zones for individual muscles, the electrical stimulation of which causes discrete movements of these muscles. Muscles that perform the most delicate working voluntary movements are especially widely represented.

Lesions of p.s. in lower mammals do not cause significant disturbances of motor functions. The higher the mammal is organized, the more significant these violations are. Pathological processes in the cortical part of the cerebral cortex, especially in the anterior central gyrus, which irritate the cerebral cortex, cause partial (partial), or Jacksonian, epilepsy, which is manifested mainly by clonic convulsions of the muscles of the opposite half of the face, trunk and limbs on the opposite side . Loss of functions g. with. manifested by paralysis, paresis.

Lesions of p.s. it is revealed during a neurological examination of voluntary (active) movements, their volume in various joints, muscle strength, muscle tone and reflexes in combination with other neurological symptoms. Electroencephalography and electromyography acquire greater diagnostic value. With a unilateral lesion of the cerebral cortex in the area of the anterior central gyrus, monoplegia and monoparesis of the arm or leg of the opposite side of the body are most often observed. Damage to the cortico-nuclear pathways of the facial nerve is usually expressed by central paresis of the lower and middle branches of this nerve. The upper branch is usually less affected due to its bilateral innervation, although its damage can often be detected (the patient cannot close the eye on the side of the lesion in isolation). Focal lesion of p.s. in the region of the internal capsule usually leads to hemiplegia (or hemiparesis), and with bilateral damage to tetraplegia. Damage to the pyramidal system in the region of the brain stem is determined by the combination of pyramidal symptoms on the opposite side with damage to the nuclei of the cranial nerves or their roots on the side of the lesion, that is, by the presence of alternating syndromes. With pyramidal hemiplegia and hemiparesis, the distal parts of the limbs are usually the most affected. Hemiplegia and hemiparesis with damage to the ps. are usually characterized by an increase in tendon reflexes, an increase in muscle tone, a loss of skin reflexes, especially plantar reflexes, the appearance of pathological reflexes - extensor (Babinsky, Oppenheim, Gordon, etc.) and flexor (Rossolimo, Bekhtereva, etc.), as well as protective reflexes. Methods of researching pathological reflexes: foot-extensor (Babinsky, Oppenheim, Gordon, Schaeffer, Pusep), flexor and their wrist analogues (Rossolimo, Zhukovsky, Bekhterev). Foot extensor reflexes with extension of the big toe are early signs of organic damage to the pyramidal system.

Babinski's reflex - in case of line irritation of the outer edge of the sole, there is an extension of the big toe, which is sometimes supplemented by simultaneous fan-like separation of all other toes (fan sign). The reflex is observed normally in children up to 1-1.5 years of age, with the formation of gait it disappears and the plantar reflex is triggered.

A similar movement of the fingers can also be caused by pressing with the thumb on the front surface of the tibial crest from top to bottom (Oppenheim's reflex).

A similar movement of the fingers can be caused by squeezing the calf muscle (Gordon's reflex) or the heel tendon (Sheffer's reflex) with the hand.

Flexion foot reflexes. The Rossolimo reflex is caused by intermittent blows of a hammer or fingertips on the plantar surface of the distal phalanges of the patient's toes. There is rapid plantar flexion of II-V toes. A similar reaction occurs with light blows of a hammer on the back surface of the patient's foot (Bekhterev's reflex) and on the middle of the sole near the base of the fingers (Zhukovsky's reflex).

Study of reflexes of oral automatism (subcortical): sucking, proboscis, distance-oral, palmar, chin.

Reflexes of oral automatism are automatic, involuntary movements performed by the mouth muscles in response to mechanical irritation of various parts of the face. In the case of tapping with a hammer on the root of the nose, lip stretching occurs (naso-lip reflex). The same reaction is observed as a result of a light blow of the hammer on the upper or lower lips (oral reflex) and when the hammer approaches the patient's mouth (distance-oral reflex). The palmar-chin reflex of Marinescu-Radovich is caused by line irritation of the palmar area of the thumb (thenar) elevation. In response, there is a contraction of the chin muscles. Reflexes are normally observed in infants.

Tendon and periosteal reflexes are evoked from the extended zone. Cross reflexes and joint movements appear - the so-called synkinesis. In the initial stages of pyramidal hemiplegia, muscle tone (and sometimes reflexes) is reduced. An increase in muscle tone appears later - 3-4 weeks after the onset of the lesion. Most often, especially with capsular lesions, an increase in muscle tone prevails in the flexors of the forearm and extensors of the lower leg. This distribution of muscle hypertension leads to the appearance of a contracture of the Wernicke-Mann type.

Muscle strength is a quantitative measure that expresses the muscle's ability to contract when resisting an external force, including gravity. Clinical examination of muscle strength primarily reveals its decrease. A preliminary, tentative assessment of muscle strength begins with finding out whether the examinee can perform active movements in all joints and whether these movements occur in full. Having identified the limitations, the doctor performs passive movements in the corresponding joints in order to rule out local damage to the musculoskeletal system (muscle and joint contractures). Limitation of passive movements in the joint caused by bone and joint pathology does not rule out that the patient may have reduced muscle strength. At the same time, the absence or limitation of active voluntary movements with the full range of passive movements in an adequate patient cooperating with the doctor indicates that the cause of the disorder is most likely a pathology of the nervous system, neuromuscular junctions or muscles.

The term "paralysis" (plegia) refers to the complete absence of active movements due to a violation of the innervation of the corresponding muscles, and the term "paresis" refers to a decrease in muscle strength. Paralysis of the muscles of one limb is called monoplegia, paralysis of the lower facial muscles, arms and legs on the same side of the body - hemiplegia; paralysis of the muscles of both legs - paraplegia, paralysis of the muscles of all four limbs - tetraplegia. Paralysis / paresis can be the result of damage to both central (upper) and peripheral (lower) motor neurons. Accordingly, two types of paralysis are distinguished: peripheral (flabby) paralysis occurs as a result of damage to the peripheral motor neuron; central (spastic) - as a result of damage to the central motor neuron. Damage to the central motor neuron (for example, in cerebral stroke) affects the muscles of the limbs to varying degrees. The abductor (abductor muscles) and extensors (extensors) are mainly affected on the arm, and the flexors (flexors) on the leg. A lesion of the pyramidal system at the level of the internal capsule (where the axons of Betz's pyramidal cells are

very compact) is characterized by the formation of Wernicke-Mann's pathological posture: the patient's arm is bent and brought to the body, and the leg is extended and, when walking, is moved to the side so that the foot moves in an arc ("the hand begs, the foot mows"). In peripheral motor neuron pathology, each level of damage (involves the anterior horns of the spinal cord, spinal nerve root, plexus or peripheral nerve) has a characteristic type of distribution of muscle weakness (myotome, neurotome).

Muscle weakness is not only neurogenic: it occurs both in primary muscle damage (myopathy) and in pathology of the neuromuscular synapse (myasthenia). Damage to the joint can be accompanied by a significant limitation of movements in it due to pain, therefore, with a pain syndrome, it is necessary to judge about muscle weakness and the presence of neurological pathology with caution. To assess muscle strength, the patient is asked to perform a movement that requires the contraction of a specific muscle(s), fix a posture and hold the muscle in a position of maximum contraction, while the examiner tries to overcome the subject's resistance and stretch the muscle . Thus, when studying muscle strength in clinical practice, they are most often guided by the principle of "tension and overcoming": the doctor counteracts the muscle being tested by the patient and determines the degree of effort required for this. Different muscles or groups of muscles are examined in turn, comparing the right and left sides (this makes it easier to detect slight muscle weakness). It is important to follow certain rules of examination. So, when assessing the strength of the muscles abducting the shoulder, the doctor should stand in front of the patient and provide resistance to movement with only one hand (but do not lean over the sitting patient, exerting pressure on the patient's arm with the entire weight of the body). Similarly, when assessing the strength of the flexors of the fingers, the doctor uses only the finger equivalent to the one being tested, but does not apply the strength of the entire hand or hand as a whole. It is also necessary to make an adjustment for the child's or elderly age of the patient. Muscle strength is usually evaluated in points, most often according to a 6-point system. When examining the neurological status, it is necessary to find out the strength of the following muscle groups.

Neck flexors: m. sternodeidomastoideus (n. accessories, C2-C3 - pp. cervicales).

Neck extensors: mm. profundi solli (C2-C4 – nn cervicales.).

Shrug: m. trapezius (n. accessories, C2-C4 - nn. cervicales).

Abduction of the shoulder: m. deltoideus (C5-C6 - n. axillaris).

Bending of the hand in the elbow joint: m. biceps brachii (C5-C6 - n. musculocutaneus).

Extension of the hand in the elbow joint: m. triceps brachii (C6-C8 - n. radialis).

Extension in the radiocarpal joint: mm extensores of the wrist (C5-C6 – n. radialis). ulnar extensor of the wrist (C7-C8 - n. radialis).

Opposition of the thumb: m. opponens pollicis (C8-T1 -. n. Medianus).

Abduction of the little finger: m. abductor digiti minimi (C8-T1 - n. ulnaris).

Extension of the main phalanges of II-V fingers: m. extensor digitorum communis, m. extensor digiti minimi, m. extensor indicis (C7-C8 - n. profundus n. radialis).

Flexion of the thigh in the hip joint: m. iliopsoas (L1-L3- n.femoralis).

Extension of the leg in the knee joint: m. quadricepsfemoris (L2-L4 - n.femoralis).

Bending of the leg in the knee joint: m. biceps femoris, m. semitendinosus, m. semimembranosus (L1-S2 - n. ischiadicus).

Extension (back bending) of the foot in the ankle joint: m. tibialis anterior (L4-L5 - n. peroneus profundus).

Plantar flexion of the foot in the ankle joint: m. triceps surae (S1-S2 - n. tibialis).

The above muscle groups are assessed using the following tests.

Bending the neck is a test to determine the strength of the sternoclavicular-mastoid and stair muscles. The patient is asked to tilt (but not stick out) his head to the side, and turn his face to the side opposite to the tilt of the head. The doctor opposes this movement.

Neck extension is a test that allows you to determine the strength of the extensors of the head and neck (the vertical portion of the trapezius muscle, strap muscles of the head and neck, muscles

that raise the shoulder blades, semispinous muscles of the head and neck). The patient is asked to tilt his head back, resisting this movement.

Shrugging is a test used to determine the strength of the trapezius muscle. The patient is offered to "shrug his shoulders", overcoming the opposition of the doctor.

Abduction of the shoulder is a test for determining the strength of the deltoid muscle. At the request of the doctor, the patient moves the shoulder to the side to the horizontal; at the same time, it is not recommended to bend the arm at the elbow joint. They resist the movement, trying to lower his hand. It should be taken into account that the ability of the deltoid muscle to hold the shoulder in the abducted position is impaired not only when this muscle is weak, but also when the functions of the trapezius, serratus anterior and other muscles that stabilize the shoulder girdle are impaired.

Flexion of the supinated arm in the elbow joint is a test designed to determine the strength of the biceps brachii muscle. The biceps muscle of the shoulder is involved in flexion and simultaneous supination of the forearm. To study the function of the biceps muscle of the shoulder, the doctor asks the subject to supinate the hand and bend the arm at the elbow joint, resisting this movement.

Arm extension at the elbow joint is a test used to determine the strength of the triceps brachii muscle. The doctor stands behind or to the side of the patient, asks him to unbend his arm at the elbow joint and prevents this movement.

Flexion in the radiocarpal joint is a test that helps determine the strength of the radial and ulnar extensors of the hand. The patient extends and adducts the hand with straightened fingers, and the doctor prevents this movement.

Opposing the thumb of the brush - a test to determine the strength of the muscles opposing the thumb. The examinee is asked to firmly press the distal phalanx of the thumb to the base of the proximal phalanx of the little finger of the same hand and to resist the attempt to unbend the main phalanx of the thumb.

They also use a test with a strip of thick paper: they offer to squeeze it between the I and V fingers and feel the pressure.

Abduction of the little finger is a test to determine the strength of the muscle that abducts the little finger. The doctor tries to bring the abducted little finger of the patient to the other fingers against his resistance.

Extension of the main phalanges of the II-V fingers is a test used to determine the strength of the general extensor of the fingers of the hand, the extensor of the little finger and the extensor of the index finger. The patient stretches the main phalanges of the II-V fingers of the hand, when the middle and nail fingers are bent; the doctor overcomes the resistance of these fingers, and fixes his carpal joint with the other hand.

Flexion of the thigh in the hip joint is a test that allows you to determine the strength of the iliac, large and small lumbar muscles. They ask the sitting patient to bend the thigh (bring it to the stomach) and at the same time, resisting this movement, affect the lower third of the thigh. It is possible to examine the strength of hip flexion in the position of the patient lying on his back. To do this, they suggest him to raise the straightened leg and hold it in this position, overcoming the downward pressure of the doctor's palm resting on the middle of the patient's thigh. A decrease in the strength of this muscle is considered one of the early symptoms of a defeat of the pyramidal system.

Extending the leg in the knee joint is a test to determine the strength of the quadriceps muscle. The research is carried out in the position of the patient lying on his back, the leg is bent in the hip and knee joints. They ask him to unbend his leg, raising his shin. At the same time, a hand is brought under the patient's knee, holding his thigh in a semi-bent position, with the other hand, pressure is applied to the shin in a downward direction, preventing its extension. To test the strength of this muscle, a patient sitting on a chair is asked to bend the leg at the knee joint. With one hand, resist this movement, with the other - palpate the contracting muscles.

Bending the leg in the knee joint is a test necessary to determine the strength of the muscles of the back surface of the thigh. The research is carried out in the position of the patient lying on his back, the leg is bent at the hip and knee joints, the foot is firmly in contact with the couch. They try to straighten the patient's leg, having previously given him the task not to tear his foot off the couch.

Extension (dorsiflexion) of the foot in the ankle joint is a test that helps determine the strength of the tibialis anterior muscle. The patient, who is lying on his back with straight legs, is asked to pull the feet towards him, slightly bringing the inner edges of the feet, while the doctor provides resistance to this movement.

Plantar flexion of the foot in the ankle joint is a test used to determine the strength of the triceps muscle of the lower leg and the plantar muscle. The patient, lying on his back with straightened legs, performs plantar flexion of the feet, against the opposition of the doctor's palms, which exert pressure on the feet in the opposite direction.

The above techniques for assessing muscle strength should be supplemented with some simple functional tests, designed to a greater extent to check the function of the entire limb than to measure the strength of individual muscles. These tests are important for detecting minor muscle weakness, which is difficult for the doctor to notice when focusing on individual muscles.

To detect weakness in the muscles of the shoulder, forearm, and hand, the patient is asked to squeeze three or four fingers of the doctor's hand as hard as possible, and during the squeeze, they try to release their fingers. The test is performed simultaneously on the right and left hand to compare their strength. It should be taken into account that the strength of the handshake largely depends on the preservation of the muscles of the forearm, therefore, if the small muscles of the hand are weak, the handshake can remain quite strong. You can accurately measure the compression force of the brush using a dynamometer. The brush compression test allows you to detect not only the weakness of the muscles of the hand, but also the phenomenon of action myotonia, observed in such hereditary neuromuscular diseases as dystrophic and congenital myotonia. After a strong squeeze of his hand into a fist or a strong handshake of someone else's hand, a patient with the phenomenon of action myotonia cannot quickly open his hand.

To detect weakness in the proximal parts of the legs, the examinee should get up from a squatting position without the help of hands. Children should be observed how they rise from a sitting position on the floor. For example, with Duchenne myodystrophy, the child resorts to auxiliary techniques when standing up ("getting up on its own").

To detect weakness in the distal parts of the legs, the patient is offered to stand up and walk on the heels and "toes".

Central (pyramidal) paresis of the hands can be detected by asking the patient with closed eyes to hold straight arms with almost tangential palmar surfaces slightly above the horizontal level (barre test for the upper limbs). The hand on the side of the paresis begins to descend, while the hand bends in the radiocarpal joint and rotates inward ("pronator drift"). These postural disorders are considered to be very sensitive signs of central paresis, which allow it to be detected even when a direct examination of muscle strength does not reveal any abnormalities.

In patients with suspected myasthenia gravis, it is important to determine whether weakness in the muscles of the head, trunk, and limbs increases with exercise. To do this, they stretch their arms in front of them and look at the ceiling. Normally, a person can stay in this position for at least 5 minutes. Other tests provoking muscle fatigue are also used (squatting, counting loudly to 50, repeatedly opening and closing the eyes). Myasthenic fatigue can be detected most objectively with the help of a dynamometer: the force of squeezing the brush into the fist is measured, then the patient quickly performs 50 intensive compressions of both hands into the fist, after which the dynamometry of the hands is carried out again. Normally, the force of compression of the hands remains practically the same before and after such a series of compressions of the hands into a fist. In myasthenia gravis, after physical exertion of the muscles of the hand, the compression force of the dynamometer decreases by more than 5 kg.

Assessment of muscle strength according to a 6-point system (from 0 to 5 points).

The following scale is used to assess muscle strength:

0 points – no muscle contraction.

1 point - there is muscle contraction, but there is no movement in the joint.

2 points - there is movement in the joint, but it is impossible to overcome the force of gravity.

3 points - it is possible to overcome the force of gravity, but movement is impossible when overcoming the resistance.

4 points - it is possible to overcome some resistance.

5 points – muscle strength is normal.

Methodology of examination of muscle tone and determination of spastic and plastic muscle hypertension.

Muscle tone is assessed by palpating them in a state of rest and complete relaxation, as well as by examining passive movements in the joints also under conditions of complete muscle relaxation.

With spastic muscle hypertonia, the tone increases according to the spastic type, or the "complex knife" type, that is, the resistance felt by the muscles is the greatest at the beginning of the movement, and then it decreases.

With plastic muscular hypertonia, the resistance felt by the muscles is constant throughout the entire movement in the form of plastic or waxy stiffness, and intermittent, stepwise stretching of the muscles can also be observed, which is called the "cogwheel symptom."

Hypotonia of the muscles mainly occurs when the peripheral motor neuron is damaged.

Spasticity is assessed according to the Ashforth spasticity scale

Modified Ashforth spasticity scale

Points	Muscle tone
0	No promotion
1	A slight increase in tone, which is felt when bending or extending a segment of the limb in the form of a slight contraction at the end of the movement
2	A slight increase in tone in the form of resistance, which occurs after performing at least half of the volume of movement
3	A moderate increase in tone, which is evident during the entire movement, but does not make it difficult to perform passive movement
4	A significant increase in tone, which makes it difficult to perform passive movements
5	The affected segment of the limb is fixed in the position of flexion or extension

Materials for self-control of training quality

Tests

1) All paths pass through the legs of the cerebrum, except:

Answer options:

a) fronto-bridge;

b) tuberospinal;
c) cortico-spinal;
d) occipital-parietal-temporal-bridge
e) visual
Correct answer: b)

2) Where is the peripheral neuron of the cortico-muscular pathway located?
Answer options:
a) within the vertebral ganglia;
b) in the front central gyrus;
c) in the posterior central gyrus;
d) in the anterior horns of the spinal cord;
e) in the visual hill.
Correct answers: d)

3) Specify where the fibers of the cortical-nuclear pathway end?
Answer options:
a) in the lateral columns of the spinal cord;
b) in the front columns of the spinal cord;
c) in the back columns of the spinal cord;
d) in motor nuclei of cranial nerves;
e) in sensitive nuclei of cranial nerves.
Correct answer: d)

4) Specify where the fibers of the cortical-spinal pathway end?
Answer options:
a) in the lateral columns of the spinal cord;
b) in the front columns of the spinal cord;
c) in the back columns of the spinal cord;
d) in motor nuclei of cranial nerves;
e) in sensitive nuclei of cranial nerves.
Correct answer: a)

5) Indicate what condition characterizes the lesion of the 1st neuron of the motor pathway?
Answer options:
a) muscle hypertension;
b) ataxia;
c) muscle hypotonia;
d) anesthesia;
e) sensitive ataxia.
Correct answer: a)

6) Indicate what condition characterizes the damage of the II neuron of the motor pathway?
Answer options:
a) muscle hypertension;
b) ataxia;
c) muscle hypotonia;
d) anesthesia;
e) sensitive ataxia.
Correct answer: c)

Tasks for self-control :

1. A patient with thoracic spondylogenic myelopathy has increased knee and Achilles reflexes on both sides, pathological reflexes have appeared. Why did this happen and how were the reflex arcs of these reflexes affected?
   Correct answer: reflex arcs are not affected. A lesion of the pyramidal tracts developed

on both sides at the thoracic level.

2. What symptoms develop when the anterior horns of the spinal cord are affected?
   Correct answer: atony, atrophy, areflexia.
3. What kind of paralysis occurs when the lateral column of the spinal cord is affected?
   The correct answer is: central.
4. The patient developed a hemorrhage in the right internal capsule, what are the symptoms will you watch it?
   Correct answer: contralateral central hemiplegia, hemianesthesia, hemianopsia.

Materials for classroom self-training:

List of educational practical tasks:

1. Master research methods:
   a) gait of the patient;
   b) volume of arbitrary movements;
   c) muscle strength;
   d) muscle trophics;
   e) muscle tone;
   f) pathological reflexes:
   - Babinsky;
   - Oppenheim;
   - Zhukovsky;
   - Rossolimo;
   - Bekhtereva.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 3

Topic: Automated involuntary movements. Coordination of movements. Extrapyramidal system and syndromes of its damage.

Purpose: to acquaint students with the various effects of the extrapyramidal system on human motor function, on the balanced functioning of which normal life depends.

Basic concepts: damage to subcortical nodes is observed in infectious processes, injuries. Most often, the internal capsule and the optic hill are affected during strokes. The ability to diagnose the symptoms of damage to the subcortex and the internal capsule helps to correctly prescribe treatment and ensure a faster recovery of the patient.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units).

Content

A separate finished motor act, no matter how primitive it may be, requires the coordinated participation of many muscles. The simplest movement - raising the hand - is ensured by the contraction of the muscles of the shoulder girdle, but at the same time the muscles of the trunk and lower limbs, which restore the correct position of the body's center of gravity. The quality of movement depends not only on the type and number of muscles that implement it. Often the same muscles are involved in different movements; the same movement can be performed faster or slower, with more or less force, depending on the conditions. Thus, to perform the movement, the

participation of mechanisms regulating the sequence, force and duration of muscle contractions and regulating the selection of the necessary muscles is necessary. In other words, the motor act is formed as a result of the sequential, consistent strength and duration of the inclusion of individual neurons and fibers of the cortico-muscular pathway, which gives orders to the muscles. This inclusion is provided with the participation of almost all motor systems of the brain, and first of all - the extrapyramidal system and its striopallidal division.

The extrapyramidal system includes structures of the cortex of the large hemispheres, subcortical ganglia, cerebellum, reticular formation, descending and ascending pathways. Arbitrarily performing this or that action, a person does not think about which muscle should be activated at the right moment, does not keep in his conscious memory a consistent working scheme of the motor act. Habitual movements are produced mechanically, imperceptibly for attention, the change of some muscle contractions by others is involuntary, automated. Motor automatisms guarantee the most economical consumption of muscle energy during movement. A new, unfamiliar motor act is always more wasteful in terms of energy than a familiar, automated one. The swing of the scythe of the mower, the blow of the hammer of the blacksmith, the running of the fingers of the musician - refined to the limit, energy-saving and rational automated movements. Improvement of movements - in their gradual economization, automation, are ensured by the activity of the striopallidar system.

The striopallida system is divided into striatum and pallidum according to its functional significance and morphological features. The caudal core and shell are combined into a striatal system. The pallidum, substantia nigra, red nucleus, and subthalamic nucleus make up the pallidary system. The pallidum contains a large number of nerve fibers and relatively few large cells. The caudate core and shell include many small and large cells and a small number of nerve fibers. There is a somatotopic distribution in the striatal system: in the oral sections - the head, in the middle sections - the arms and trunk, in the caudal sections - the leg. There is a close relationship between the striarate and pallidary systems. The striate system is "younger" than the pallidary system, both phylogenetically and ontogenetically. It first appeared only in birds and is formed in humans by the end of the fetal period, somewhat later than the pallidum. The pallidary system in fish and the striopallidary system in birds are higher motor centers that determine the animal's behavior. Striopallidar devices ensure diffuse, massive body movements, coordinated work of all skeletal muscles in the process of movement, swimming, flight, etc. The life activity of higher animals and humans requires a finer differentiation of the work of motor centers. The extra-pyramidal system can no longer satisfy the needs of movements of a purposeful, productive nature. In the cortex of the forebrain, a higher apparatus is created in the process of evolution, coordinating the coordinated function of the pyramidal and extrapyramidal systems that control the execution of complex movements. However, having moved to a subordinate, "subordinate" position, the striopallidary system did not lose its inherent functions.

The difference in the functional value of the striatum and pallidum is also determined by the complication of the nature of movements in the process of phylogenesis. "Palidar" fish, moving in a state suspended in water with jerking, powerful movements of the body, should not "care" about saving muscle energy. The needs of such a motor act are completely satisfied by the work of the pallidar system, which ensures powerful and relatively accurate movements, but energy wasteful and excessive. A bird forced to perform enormous work in flight and unable to suddenly interrupt it in the air must have a more complex motor apparatus that carefully regulates the quality and quantity of movements - the striopallidar system.

The development and inclusion of motor systems in human ontogenesis has the same sequence. Myelination of striatal pathways ends only by the 5th month of life, therefore, in the first months, the pallidum is a higher motor organ. The motility of newborns has clear "palidaric" features. The movements of a child up to 3-4 years of age and the movements of a young animal have a great similarity, which consists precisely in excess, freedom, generosity of movements. The richness of the child's facial expressions is characteristic, and also indicates a certain predominance of "palidarity". With age, many human movements become more and more habitual, automated,

energetically prudent, stingy. A smile ceases to be a permanent facial expression. The stateliness, solidity of adults is the triumph of the striatum over the pallidum, the triumph of the sober prudence of automated movements over the wasteful generosity of the child's still "inexperienced" striopallidal system. The process of learning a certain movement, aimed at automating a motor act, has two phases. During the first phase, which is conventionally called pallidarna, the movement is excessive, excessive in strength and duration of muscle contraction. The second phase of the rationalization of movement consists in the gradual development of an energetically rational, maximally effective (with minimal effort) mode of movement that is optimal for a given individual.

The striopallidar system is the most important tool in the development of motor automatisms, which in an adult are purposefully selected and implemented by the higher cortical centers of praxis. The child's relative "pallidarity" is due not only to the immaturity of the striatum, but also to the fact that the child is still in the stage of motor learning in its first, pallidary phase. The older the child, the more the number of motor acts is automated, i.e. they are no longer "pallidary". Along with this, the immaturity of the striatum and the predominance of "pallidarity" in newborns are, as it were, planned in advance, since it is "pallidarity" that is necessary for a child in the first period of extrauterine life. The striopallidar system has numerous connections: paths connecting parts of the striopallidar system; paths connecting the striopallidary system with the final motor path and muscle; mutual connections with various departments of the extrapyramidal system and the cortex of the large hemispheres, and, finally, the afferent pathways. There are several ways of delivering pulses of the striopallidary system to the segmental motor apparatus: 1) red nuclear-spinal tract from red nuclei; 2) the vestibulo-spinal tract from the vestibular nucleus; 3) reticulospinal tracts from the reticular formation; 4) tectospinocerebral (tyrospinal) path from tetracholium; 5) pathways to motor nuclei of cranial nerves. The striopallidar system responsible for the involuntary performance of motor acts must receive comprehensive information about the state of muscles, tendons, the position of the body in space, etc. reticular formation, corrective signals from the cortex, etc.), together with efferent pathways, create feedback loops with a continuous flow of informing and correcting, commanding signals. The circulation of impulses does not stop, combining all motor and afferent systems into a single whole.

When the nuclei of the extrapyramidal system and their connections are damaged, various symptoms appear. Hypotonic-hyperkinetic and akinetic-rigid syndromes are the main ones. Disorders of the extrapyramidal system are manifested in the form of changes in motor function, muscle tone, autonomic functions, and emotional disorders.

Symptoms of damage to the pallidum. Pallidar syndrome. The symptom complex of lesions of the globus pallidum and substantia nigra is called parkinsonism, akinetic-rigid syndrome, amiostatic syndrome, and hypertonic-hypokinetic syndrome. It is associated with a functional deficiency of the pallidum, with a change in the influence of the pallidonegral system on the reticular formation and a violation of impulses in the cortical-subcortical-stem neural circuits. The reticular formation - the trunk "controller-regulator" of the flow of ascending and descending impulses - when its connections with the substantia nigra are broken, it does not prevent the passage of excess tonic signals to the muscles, as a result of which muscle stiffness develops, supported by a continuous flow of afferent impulses to the striopallidary system (a vicious circle: the affected pallidal system sends uncontrolled tonic signals that increase muscle tone and strengthen the flow of reverse afferent impulses, which in turn tones the striopallidum).

In Parkinson's disease, the structures of the extrapyramidal system are affected - the basal nuclei and the substantia nigra, the blue spot, etc. The most pronounced changes are noted in the front divisions of the substantia nigra. Symptoms typical of Parkinson's disease occur when 60-80% of the neurons of this anatomical entity die. Macroscopic changes are characterized by depigmentation of the blue spot and the area of the black substance that contains melanin . Microscopic examination of the affected areas reveals a decrease in the number of nerve cells. The presence of Lewy bodies is determined in them. There is also the death of astrocytes (types of glial cells) and the activation of microglia . Lewy bodies are formed due to the accumulation of α-

synuclein protein in the cytoplasm of cells. The presence of Lewy bodies is one of the signs of Parkinson's disease. At the same time, Lewy bodies are also detected in other neurodegenerative diseases. In this regard, they are not considered a specific marker of Parkinson's disease. In addition, in this disease, "pale bodies" - intracellular granular inclusions that replace disintegrating melanin - are found in the substantia nigra and macula.

According to the classification proposed by Braak and co-authors, in the asymptomatic stage of Parkinson's disease, Lewy bodies appear in the nerve cells of the olfactory bulb, medulla oblongata , and Varolian bridge . As the disease progresses, the presence of these pathological bodies is noted in the neurons of the substantia nigra, midbrain , basal ganglia, and at the final stages in the cells of the cerebral cortex.

Pathological physiology. The close relationship between the components of the extrapyramidal system — pallidum and striatum — is provided by numerous bundles of nerve fibers. Thanks to the connections between the thalamus and the striopallidary system, reflex arcs are formed, which ensure the performance of numerous stereotyped and automated movements (for example, walking, running, swimming, cycling, etc.). The close connection of the striopallidar system with the nuclei of the hypothalamus determines its role in the mechanisms of emotional reactions.

Normally, the extrapyramidal system sends impulses to peripheral motor neurons. These signals play an important role in ensuring myostatics by preparing the muscles for involuntary movements. The activity of this part of the central nervous system depends on the ability of a person to take the optimal posture for the planned action, the necessary ratio of the tone of agonist muscles and antagonist muscles, as well as smoothness and proportionality of voluntary movements in time and space is achieved.

The nature of the clinical manifestations of the disease depends on which part of the striopallidary system is affected - striatum or pallidum. If the inhibitory effect of the striatum is excessive, hypokinesia occurs - poor movement, amimia . Hypofunction of the striatum leads to excessive involuntary movements - hyperkinesis. The pallidum inhibits the structures of the striatum. Parkinson's disease is characterized by a decrease in the inhibitory effect of the pallidum on the striatum. Damage to the pallidum leads to "disinhibition of inhibition" of peripheral motor neurons.

The discovery of the role of neurotransmitters made it possible to explain the functions of the extrapyramidal system, as well as the causes of the clinical manifestations of Parkinson's disease and parkinsonism. There are several dopaminergic systems in the brain. One of them begins in the neurons of the substantia nigra, the axons of which reach the striatum (lat. corpus striatum) through the peduncle of the brain, the internal capsule, and the globus pallidum. The terminal sections of these axons contain a large amount of dopamine and its derivatives. Degeneration of this nigrostriatal dopaminergic pathway is a major factor in the development of Parkinson's disease. The second ascending dopaminergic system is the mesolimbic pathway. It starts from the cells of the interpeduncular nucleus of the midbrain and ends in the hypothalamus and frontal lobes of the brain. This pathway is involved in the control of mood, behavior and controls the initiation of a motor act and affective reaction movements (movements that accompany emotions).

The basis of all forms of parkinsonism is a sharp decrease in the amount of dopamine in the substantia nigra and striatum and, accordingly, a disruption in the functioning of dopaminergic pathways in the brain.

The processes of dopaminergic neurons of the compact part of the substantia nigra are directed to the striatum, exerting an excitatory effect on the direct pathway (via D1 receptors) and an inhibitory effect on the indirect pathway (via D2 receptors). The basis of all forms of parkinsonism is a sharp decrease in the amount of dopamine in the substantia nigra and striatum. Normally, the amount of dopamine in these formations is hundreds of times higher than the concentration of dopamine in other brain structures, which convincingly indicates its important role in the activity of the extrapyramidal system. Synthesis of dopamine occurs in the bodies of

dopaminergic neurons located in the substantia nigra. Here it is formed in the form of small bubbles-granules, which are transported along the axons of nigrostriatal neurons to the ends of axons in the caudate nucleus and accumulate in front of the synaptic membrane. Under the influence of incoming nerve impulses, mediator quanta are released into the synaptic cleft. The released dopamine affects the dopaminergic receptors of the postsynaptic membrane and causes its state of depolarization. Only part of the mediator is used to activate the postsynaptic membrane. It is assumed that about 80% of dopamine is absorbed back into the presynaptic space or inactivated by the enzyme KOMT (catechol-O-methyltransferase) or MAO-B. All processes of dopamine formation and transport are regulated by means of feedback mechanisms through receptors of the presynaptic membrane.

In addition to a decrease in the concentration of dopamine, the concentration of dopamine metabolites: homovanillic acid and the activity of tyrosine hydroxylase and dopamine hydroxylase - enzymes necessary for the conversion of tyrosine into DOPA and DOPA into dopamine - also noticeably decrease. These biochemical disturbances are the result of destruction of dopamine neurons and nigrostriatal connections. Disturbances in norepinephrine and serotonin metabolism in PD are less pronounced, which indicates a lower degree of damage to adrenergic and serotonergic neurons located in the macula and midline nuclei compared to nigral dopaminergic neurons. Dysfunction of the dopaminergic system causes a violation of the interaction of the striatum with other basal ganglia and the cerebral cortex. The basal ganglia are connected with each other and the cortex of the cerebrum with the help of neuronal circles that connect the pre- and postcentral sensorimotor areas, the basal ganglia, the anteroventral and ventrolateral parts of the thalamus. There are two main circles (paths) - direct and indirect. The direct path connects the scrotum and the inner segment of the globus pallidus (BC); the indirect one passes through the external segment of the BC, the subthalamic nucleus (STN) and further to the internal segment of the BC. The subthalamic nucleus exerts an activating effect on the internal segment of the brain, while all other connections of the basal ganglia, including their connection with the thalamus, are inhibitory (GABA-ergic). The direct pathway is mainly regulated by D1-dopamine receptors, and the indirect pathway is regulated by D2-receptors. Dopaminergic effects of nigrostriatal neurons facilitate the transmission of impulses in a direct way (by stimulating D1-peceptors) and suppress it in an indirect way (by stimulating D2-receptors). As a result, dopamine reduces the inhibitory effect of the basal ganglia, which leads to the facilitation of thalamocortical transmission and "disinhibition" of the motor cortex. As a result, it becomes possible to transfer the motor program from the premotor cortex to the executive precentral gyrus. The pathophysiological basis of parkinsonism is the excessive inhibitory activity of the basal ganglia, which occurs as a result of their dopaminergic denervation, which leads to the inhibition of the motor parts of the cortex and the development of akinesia. The development of these violations is opposed by significant compensatory mechanisms. It is known that in patients with parkinsonism, an increase in the ratio: homovanillic acid / dopamine is noted, which indicates that the remaining neurons work more actively than in a healthy person. This phenomenon is regarded as a compensatory mechanism that protects against the early appearance of symptoms of the disease. It has been experimentally shown that the level of dopamine in the striatum can decrease to 20% of normal before clinical manifestations of the disease appear. Increasing the sensitivity of dopamine postsynaptic receptors can also serve as a compensatory mechanism that inhibits the development of parkinsonism. The development of clinical manifestations of the disease occurs when all possibilities of compensating for the lack of the dopamine system are exhausted. In addition to dopamine, a significant concentration of another central nervous system mediator - acetylcholine - was found in the caudate nucleus. It is assumed that acetylcholine is formed in the numerous interneurons of the striatum, and does not get there, like dopamine, from other parts of the brain. As mentioned above, dopamine inhibits activity in the caudate nucleus, while acetylcholine is a facilitatory mediator. As a result, the functional state of the caudate nucleus is determined by the balance between the dopaminergic and acetylcholinergic systems with the relative predominance of the latter. At the same time, a deficiency of acetylcholine is detected in the frontal lobes, which may be one of the

causes of cognitive and behavioral disorders in PD. However, dopamine deficiency is certainly only an intermediate link in the pathological process and indicates only the loss of dopamine-producing function of nigrostriatal neurons.

The main symptoms of damage to the globus pallidus are poverty and inexpressiveness of movements (oligokinesia), their slowness (bradykinesia). Patients are immobile, inert, stiff, when moving from a position of rest to a state of movement, they often freeze in an uncomfortable position (the pose of a wax doll, mannequin - the phenomenon of catalepsy). The characteristic appearance of the patients - the body is slightly bent, the head is bent forward, the arms are bent and brought to the body, the gaze is directed forward, motionless. The beginning of a motor act is difficult - parkinsonian stomping on the spot. The patient moves with difficulty, with frequent small steps. There are no normal physiological synkinesis, acheirokinesis is noted (hands are motionless when walking). The lack of joint movements is expressed in a violation of the inertia necessary for the balance of the body. Taken out of balance, the patient cannot straighten it automatically. If the patient is pushed, he runs in the direction of the push: forward - propulsion, to the side - lateropulsion, back - retropulsion. Speech is monotonous, quiet (bradilalia). The handwriting is small, indistinct (micrograph). Diseased ligaments in the circulation, sticky (akairiya), thinking is slowed down (bradypsychia). In some cases, "paradoxical kinesias" are observed. Yes, patients sit in a chair all day, at the moment of affective outbursts and emotional stress they can run up the stairs, jump, dance. Parkinsonian resting tremor is more often localized in the fingers and manifests itself in the phenomenon of "rolling pills", "counting coins". Tremor is observed at rest and decreases with voluntary movements. A characteristic change in muscle tone of the plastic type. Resistance during the examination of tone remains uniform at the beginning and at the end of the movement. The phenomenon of the "cogwheel" appears. Postural reflexes are revealed: the Westphalia foot phenomenon and the shin phenomenon. Westphal's phenomenon - with passive dorsiflexion of the foot, there is tonic tension of the extensors of the foot and the foot stiffens in the position of dorsiflexion. The shin phenomenon - in a patient lying on his stomach with his legs bent at the knees at a right angle, with subsequent passive bending of the shin, it freezes in the flexed position.

Symptoms of damage to the striatum. Striar syndrome. When the striatal system is damaged, a hypotonic-hyperkinetic syndrome occurs, which is caused by a deficiency of the inhibitory effect of the striatum on the lower motor centers, as a result of which muscle hypotonia and excessive involuntary movements (hyperkinesis) develop. Hyperkinesis - automatic, excessive movements involving individual body parts and limbs. They arise involuntarily, disappear in sleep and intensify with arbitrary movements and excitement. Certain types of hyperkinesis are associated with damage to certain structures of the striatal system. When the oral part of the striatum (striatum) is affected, violent movements occur in the muscles of the face and neck, when the middle part is affected - in the muscles of the trunk and arms. A lesion of the caudal part of the striatum causes hyperkinesis in the legs. When studying hyperkinesis, attention is paid to the side, rhythm, character, shape, symmetry, and localization of the motor manifestation. Athetosis - slow, worm-like movements in the distal parts of the limbs (in the hands and feet). It can be observed in the musculature of the face - protrusion of the lips, distortion of the mouth, clicking of the tongue. Usually, athetosis is associated with damage to large cells of the striatal system. Its characteristic feature is the formation of temporary contractures that give the hands and fingers a peculiar position. Bilateral, double athetosis with subcortical degenerations is often observed in children. Hemiathetosis is much less common. Hemibalism - large sweeping "throwing" movements of the limbs. It is most often found in the hands in the form of a "bird's wing" wave. Violent movements with hemibalism are produced with great force, they are difficult to stop. The occurrence of hemibalism is associated with a lesion of the Lewis body located under the optic tubercle. Choreic hyperkinesis - rapid contractions of various groups of muscles of the face, trunk and limbs. Hyperkinesis is non-rhythmic, uncoordinated, extends to large groups of muscles of the distal and proximal departments. It can resemble voluntary movements, since synergistic muscles are involved in the process. Frowning of the eyebrows, forehead, protruding tongue, jerky, disorderly

movements of the limbs are noted. Hyperkinesis can cover half of the body - hemichorea. Choreic hyperkinesis occurs when the neostriatum is damaged and is observed in subcortical degenerations, rheumatic brain damage, and Huntington's hereditary chorea.

In some cases, choreic hyperkinesis is combined with athetosis (choreoathetosis). Choreoathetosis can be observed in patients both constantly and in the form of attacks - paroxysmal choreoathetosis. Several variants of the familial form of paroxysmal choreathetosis have been described. Myoclonia - short, lightning-fast clonic twitches of muscles or groups of muscle fibers, often of a rhythmic nature. Myoclonus can be both generalized and local. Their most frequent localization is the proximal parts of the limbs, trunk, and face. Local myoclonus of the tongue and soft palate (velopalatine nystagmus) is described. Myoclonus persists at rest and in motion, worsens with excitement. Unlike clonic convulsions, myoclonus usually does not cause limb movements. Myoclonic hyperkinesis accompanies inflammatory, toxic, hereditary degenerative lesions of the extrapyramidal system with predominant lesions of the dentate nuclei, inferior olives, red nuclei, substantia nigra, and striatum. Tics are fast clonic jerks of a limited group of muscles, usually of a stereotyped character, imitating voluntary movements, which is why they often give the impression of being deliberate. They are more often localized in the muscles of the face and are manifested by a rapid frowning of the forehead, raising the eyebrows, blinking, sticking out the tongue. A tic of the neck muscles is less common - turning the head to the side, nodding forward. In children, a tic often develops as a manifestation of neurosis, as a result of which a pathological conditioned reflex is formed, as an imitation of persons suffering from hyperkinesis (functional hyperkinesis). Facial muscle flow can occur with trigeminal neuralgia. Along with local forms, there can be a generalized tic characterized by the involvement of the muscles of the limbs, trunk, respiratory muscles, and facial muscles.

Muscular dystonia is a syndrome characterized by violent slow or fast repetitive movements that cause rotation, bending or extension of certain parts of the body with the formation of pathological postures. Etiology. Primary (idiopathic) dystonia is distinguished, which accounts for about 90% of dystonia cases and is often hereditary, and secondary (symptomatic) dystonia caused by the use of drugs (especially neuroleptics), injuries, encephalitis, vascular diseases, tumors, degenerative brain diseases, perinatal pathology (anoxia, nuclear jaundice), hereditary metabolic disorders (in particular, hepatolenticular degeneration). Symptoms, course. By prevalence, focal dystonia involving a small part of the body (for example, the neck), segmental dystonia involving adjacent parts of the body (for example, the neck and arm), multifocal dystonia involving non-adjacent parts of the body, and generalized dystonia, hemidystonia involving ipsilateral arm and leg At first, violent movements appear only during a certain activity (for example, a dystonic posture in the hand appears only when writing or typing on a typewriter), then - during any action, and then lose dependence on voluntary movement and become permanent, leading to to the formation of a fixed posture and joint contractures. This characteristic evolution, along with other manifestations of the dynamism of hyperkinesis (fluctuating symptoms during the day with short-term improvement after sleep, the effect of corrective gestures and changes in posture, worsening with excitement), allows us to distinguish dystonia from diseases of the musculoskeletal system that cause fixed changes in posture . In childhood, dystonia usually begins locally with the involvement of one foot, but later gradually involves other parts of the body, becoming generalized. This variant of dystonia is also referred to as torsion dystonia, which emphasizes the presence of twisting of the trunk around the axis. It can be familial (inherited in an autosomal dominant type) or sporadic (idipathic). Dystonia sensitive to levodopa (Segawa syndrome), which accounts for 10-15% of cases of childhood dystonia, is characterized by pronounced fluctuations with an increase in symptoms in the afternoon, increased hyperkinesis during physical exertion, symptoms of parkinsonism, predominant involvement of the legs (which often leads to false diagnosis of cerebral palsy). Adult-onset dystonias often remain focal or segmental. Focal dystonias include cranial dystonia (blepharospasm, oromandibular dystonia), cervical dystonia (spastic torticollis), laryngeal dystonia (spastic dysphonia), trunk and limb dystonia. A separate group consists of the so-called professional dystonias (writing spasm, typist's spasm, pianist's

spasm), induced only by specific movements.

Materials for self-control of training quality

Tests

1) What symptoms occur when motor subcortical nodes are affected?

Answer options:
a) central paralysis;
b) peripheral paralysis;
c) hyperkinesis;
d) hypokinesia
Correct answer: c, d)

2) What formations belong to the striatal system?

Answer options:
a) visual hill;
b) pale layer and black substance;
c) caudate nucleus and shell;
d) red nucleus.
Correct answers: b)

3) Name the symptoms of irritation of the optic hill?

Answer options:
a) hyperpathy on the side of the lesion;
b) hyperpathy on the opposite side from the site of irritation;
c) hemiataxia;
d) hemianopsia;
Correct answer: b)

Tasks for self-control :

1. The patient has stiffness of movements, slowness of gait, active movements, speech, shallow rhythmic tremor of the distal parts of the hands. Determine the focus of the lesion? The correct answer is: pallidar system.
2. The patient has sweeping non-rhythmic hyperkinesis, a dancing gait, and grimacing. Muscle tone is reduced. Determine a topical diagnosis? Correct answer: damage to the striatal system.

Materials for classroom self-training:

List of educational practical tasks:
1. Master the ability to identify:
   a) symptoms of pallidary disorders;
   b) symptoms of damage to the striatal system;
   c) symptoms of damage to the optic hill;
   d) symptoms of damage to the internal capsule.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 4

Topic: Cerebellum. Syndromes of damage to the cerebellum. Types of ataxia.

Goal: to be able to study cerebellar functions: coordination of movements, balance, muscle tone, synergism of muscle work; learn to diagnose and differentiate different types of ataxias for the correct diagnosis of neurological diseases in children and adults.

Basic concepts: lesions of the cerebellum and its leading pathways are observed in adults and children with a number of neurological diseases, the diagnosis of which depends on the ability

to recognize cerebellar symptoms, which is of great importance for the timely application of appropriate therapeutic measures.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
    - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units).

Content

The cerebellum is located under a duplication of the dura mater known as the cerebellar tentorium, which divides the cranial cavity into two unequal spaces - supratentorial and subtentorial. In addition to the cerebellum, the brain stem is located in the subtentorial space, the bottom of which is the posterior cranial fossa. The volume of the cerebellum is 162 cm^3 on average . Its weight varies between 136 and 169 g. The cerebellum is located above the pons and the medulla oblongata. Together with the upper and lower brain sails, it forms the roof of the IV ventricle of the brain, the bottom of which is the so-called rhomboid fossa. Above the cerebellum are the occipital lobes of the cerebrum, separated from it by the tent of the cerebellum.

There are two hemispheres in the cerebellum (cerebellar hemisphere). Between them, in the sagittal plane above the IV ventricle of the brain, the phylogenetically most ancient part of the cerebellum is located - its worm (cerebellar worm). The cerebellum and cerebellar hemispheres are fragmented into lobes by deep transverse furrows. The cerebellum consists of gray and white matter. Gray matter forms the cortex of the cerebellum and paired nuclei of the cerebellum are located in its depth. The largest of them - dentate nuclei (n. dentatus) - are located in the hemispheres. In the central part of the worm there is a tent nucleus (fastigii), between them and the toothed nuclei there are spherical and cork-like nuclei (nuclei. globosus and emboliformis). Considering the fact that the cortex covers the entire surface of the cerebellum and penetrates into the depth of its furrows, on a sagittal section of the cerebellum, its tissue has the pattern of a leaf, the veins of which are formed by white matter, which makes up the so-called tree of life of the cerebellum. At the base of the tree of life is a wedge-shaped recess, which is the upper part of the cavity of the IV ventricle; the edge of this notch forms his tent. The roof of the tent is the cerebellar worm, and its front and back walls are made up of thin brain plates, known as the front and back brain sails.

The cerebellum has three pairs of legs: lower, middle and upper. The lower leg connects it with the medulla oblongata, the middle one with the pons, the upper one with the midbrain. The peduncles of the brain are the pathways that carry impulses to and from the cerebellum. The cerebellar worm provides stabilization of the body's center of gravity, its balance, stability, regulation of the tone of reciprocal muscle groups, mainly of the neck and trunk, and the emergence of physiological cerebellar synergies that stabilize the body's balance.

To successfully maintain body balance, the cerebellum constantly receives information passing through spinocerebellar pathways from proprioceptors of various body parts, as well as from the vestibular nuclei, inferior olives, reticular formation, and other formations involved in controlling the position of body parts in space. Most of the afferent pathways going to the cerebellum pass through the lower cerebellar peduncle, some of them are located in the upper cerebellar peduncle.

Impulses of proprioceptive sensitivity going to the cerebellum, like other sensitive impulses, following the dendrites of the first sensitive neurons, reach their bodies located in the spinal nodes. Further, the impulses going to the cerebellum along the axons of the same neurons are directed to the bodies of other neurons, which are located in the inner parts of the base of the posterior horns,

forming the so-called Clark's columns. Their axons enter the lateral parts of the lateral cords of the spinal cord, where they form spinocerebellar tracts, while part of the axons enter the lateral column of the same side and form the posterior spinocerebellar tract of Flexig there (tractus spinocerebellaris posterior). The other part of the axons of the cells of the posterior horns passes to the other side of the spinal cord and enters the opposite lateral cord, forming in it the anterior spinocerebellar tract of Hovers (tractus spinocerebellaris anterior). The spinocerebellar tract, increasing in volume at the level of each spinal segment, rises to the medulla oblongata. In the medulla oblongata, the posterior spinocerebellar path deviates in the lateral direction and, passing through the lower cerebellar peduncle, enters the cerebellum. The anterior spinocerebellar path transits through the medulla oblongata, the pons of the brain and reaches the midbrain, at the level of which it makes its second crossing in the anterior cerebral sail and passes into the cerebellum through the upper cerebellar peduncle. Thus, of the two spinocerebellar pathways, one is never crossed (Flexig's pathway), and the other crosses to the opposite side twice (Govers's twice-crossed pathway). As a result, both conduct impulses from each half of the body, mainly to the homolateral half of the cerebellum. In addition to Flexig's spinocerebellar tracts, through the lower cerebellar peduncle, impulses to the cerebellum pass along the vestibulocerebellar tract (tractus vestibulocerebellaris), which begins mainly in the upper vestibular nucleus of Bekhterev, and along the olivocerebellar tract (tractus olivocerebellaris), which comes from the lower olive. Part of the axons of the cells of the thin and sphenoid nuclei, which do not participate in the formation of the bulbothalamic tract, in the form of external arcuate fibers also enter the cerebellum through the lower cerebellar peduncle. Through its middle legs, the cerebellum receives impulses from the cortex of the large hemispheres of the brain. These impulses pass through the cortical-pontine cerebellar pathway, which consists of two neurons. The bodies of the first neurons are located in the cortex of the large hemispheres, mainly in the cortex of the posterior parts of the frontal lobes. Their axons pass in the composition of the radiant crown, the front leg of the internal capsule and end in the nuclei of the bridge. Axons of the cells of the second neurons, the bodies of which are located in their own nuclei of the bridge, pass to its opposite side and form the middle cerebellar peduncle after crossing, ending in the opposite hemisphere of the cerebellum. Part of the impulses that arose in the cortex of the large hemispheres of the brain reaches the opposite hemisphere of the cerebellum, bringing information not about the developed, but only about the planned active movement program. Having received such information, the cerebellum immediately sends out impulses that correct voluntary movements, mainly by extinguishing inertia and the most rational regulation of the tone of reciprocal muscles - agonist and antagonist muscles. As a result, a kind of eumetry is created, which makes arbitrary movements clear, refined, devoid of inappropriate components. Paths leaving the cerebellum consist of axons of cells whose bodies form its nuclei. Most of the efferent pathways, including those coming from the dentate nuclei, leave the cerebellum through its upper peduncle. An intersection of efferent cerebellar pathways (the intersection of the upper cerebellar peduncles of Werneking) is formed at the level of the lower tubercles of the quadrangle. After crossing, each of them reaches the red nuclei of the opposite side of the midbrain. In the red nuclei, cerebellar impulses are switched to the next neuron and move further along the axons of cells whose bodies are embedded in the red nuclei. These axons are formed in the red nuclear-spinal tracts, Monaco's tracts, which shortly after leaving the red nuclei undergo a junction (the junction of the tire or Trout's junction), after which they descend into the spinal cord. In the spinal cord, red nuclear spinal tracts are located in the lateral cords; their constituent fibers end in the cells of the anterior horns of the spinal cord. The entire efferent path from the cerebellum to the cells of the anterior horns of the spinal cord can be called the cerebellar-red nucleus-spinal tract (tractus cerebello-rubrospinalis). It makes the intersection twice (the intersection of the upper cerebellar peduncles and the intersection of the tire) and ultimately connects each cerebellar hemisphere with peripheral motor neurons located in the anterior horns of the homolateral half of the spinal cord.

From the nuclei of the cerebellar worm, efferent pathways go mainly through the lower cerebellar peduncle to the reticular formation of the brainstem and vestibular nuclei. From here,

along the reticulospinal and vestibulospinal pathways, which passed along the anterior cords of the spinal cord, they also reach the cells of the anterior horns. Part of the impulses coming from the cerebellum, after passing through the vestibular nuclei, enters the medial longitudinal bundle, reaches the nuclei of III, IV and VI cranial nerves, which provide eyeball movements, and affects their function.

Summing up, it is necessary to emphasize the following:

1. Each half of the cerebellum receives impulses mainly a) from the homolateral half of the body, b) from the opposite hemisphere of the brain, which has cortico-spinal connections with the same half of the body.

2. From each half of the cerebellum, efferent impulses are sent to the cells of the anterior horns of the homolateral half of the spinal cord and to the nuclei of cranial nerves that provide eyeball movements.

This nature of cerebellar connections allows us to understand why, when one half of the cerebellum is damaged, cerebellar disorders occur mainly in the same, i.e. homolateral, half of the body. This is especially clear when the cerebellar hemispheres are affected.

If the cerebellum is affected, disorders of statics and coordination of movements, muscle hypotonia and nystagmus are characteristic. Damage to the cerebellum, primarily its worm, leads to violations of statics - the ability to maintain a stable position of the center of gravity of the human body, balance, stability. When this function is disturbed, static ataxia occurs. The instability of the patient is noted. Therefore, in a standing position, he spreads his legs wide, balances with his hands. Static ataxia is especially clearly manifested when the area of support is artificially reduced, in particular in Romberg's pose. The patient is asked to stand up, with his feet firmly pushed together and his head slightly raised. In the presence of cerebellar disorders, the patient's instability in this position is noted, his body sways, sometimes he is "pulled" in a certain direction, and if the patient is not supported, he may fall. In the case of damage to the cerebellar worm, the patient usually sways from side to side and more often falls back. With pathology of the cerebellar hemisphere, there is a tendency to fall mainly in the direction of the pathological focus. If the disorder of statics is expressed moderately, it is easier to detect it in the so-called complicated or sensitized Romberg pose. The patient is asked to put his feet on one line so that the toe of one foot rests on the heel of the other. The stability score is the same as in the normal Romberg pose. Normally, when a person is standing, the muscles of his legs are tense (reaction of support), when there is a threat of falling to the side, his leg on that side moves in the same direction, and the other leg comes off the floor (reaction of jumping). When the cerebellum (mainly the worm) is damaged, the patient's support and jumping reactions are impaired. Violation of the support reaction is manifested by the patient's instability in the standing position, especially in the Romberg pose. Violation of the jump reaction leads to the fact that if the doctor, standing behind the patient and securing him, pushes the patient in one direction or another, the patient falls with a small push (symptom of pushing). When the cerebellum is affected, the patient's gait is usually altered due to the development of statolocomotor ataxia. "Cerebellar" gait resembles the gait of a drunk person, so it is sometimes called "the gait of a drunk." Due to instability, the patient walks unsteadily, spreading his legs wide, while "throwing" him from side to side. And when the cerebellar hemisphere is affected, it deviates when walking from the given direction in the direction of the pathological focus. The instability during turns is especially pronounced. If the ataxia is pronounced, then the patients completely lose the ability to control their body and can not only stand and walk, but even sit. Predominant damage to the cerebellar hemispheres leads to disruption of its anti-inertial effects, in particular to the occurrence of kinetic ataxia. It is manifested by the inconvenience of movements and is especially pronounced during movements that require precision. Movement coordination tests are performed to detect kinetic ataxia.

Techniques for detecting dynamic ataxia and coordination of movements.

Finger-nose test: the patient stands with closed legs, arms stretched forward, fingers spread, the patient should touch the tip of his nose with the index finger of the outstretched hand with closed eyes.

Heel-knee test: the patient lies on his back, with his eyes closed, the heel of one leg touches the knee joint of the other leg, then he accurately runs the heel along the crest of the tibial bone from the knee to the foot.

Diadochokinesis test. The patient is asked to close his eyes, stretch his arms forward and quickly, rhythmically supine and pronate the hands. In the case of damage to the cerebellar hemisphere, the movements of the hand on the side of the pathological process are more sweeping (a consequence of dysmetria, more precisely - hypermetria), as a result, the hand begins to lag behind. This indicates the presence of adiadochokinesis.

Pointing test: The patient is asked to touch the rubber tip of the hammer in the doctor's hand several times with his index finger. In the case of cerebellar pathology, a miss due to dysmetria is noted in the patient's hand on the side of the affected cerebellar hemisphere. Tom-Giumenti symptom: If the patient picks up an object, such as a glass, he spreads his fingers excessively.

Cerebellar nystagmus. Twitching of the eyeballs when looking to the sides (horizontal nystagmus) is considered as a consequence of the intentional shaking of the eyeballs.

Speech disorder: Speech loses fluency, becomes explosive, fragmented, chanted according to the type of cerebellar dysarthria.

Change in handwriting: Due to a disorder of coordination of hand movements, the handwriting becomes uneven, the letters are deformed, excessively large (megalography).

Pronator phenomenon: The patient is asked to keep his arms stretched forward in the supination position, while spontaneous pronation soon occurs on the side of the affected cerebellar hemisphere.

Hoff-Schilder symptom: If the patient holds his arms outstretched forward, then on the side of the affected hemisphere, the arm soon moves outward.

Imitation phenomenon. The patient with closed eyes must quickly put his hand in a position similar to the one that the researcher had previously given to his other hand. When the cerebellar hemisphere is affected, the homolateral hand makes a movement that is excessive in amplitude.

Stewart-Holmes symptom. The researcher asks the patient sitting on the chair to bend the supinated forearms and at the same time, taking his hands by the wrists, resists him. If at the same time you suddenly let go of the patient's hands, then the hand on the side of the lesion, bending due to inertia, will forcefully hit him in the chest.

Muscle hypotonia. Cerebellar worm damage usually leads to diffuse muscle hypotonia. When the cerebellar hemisphere is affected, passive movements reveal a decrease in muscle tone on the side of the pathological process. Muscle hypotonia leads to the possibility of overextension of the forearm and lower leg (Olshansky's symptom) during passive movements.

Pathological cerebellar asynergies. Violations of physiological synergies during complex motor acts are revealed, in particular, during the following tests.

Babinski's asynergy in the standing position. If the patient standing with his legs apart tries to bend back, throwing his head at the same time, then normally, in this case, bending of the knee joints occurs. With cerebellar pathology, due to asynergy, this movement is absent, and the patient, losing balance, falls back.

Babinski's asynergy in the supine position. The patient, who is lying on a hard surface with extended legs, separated by the width of the upper arms, is asked to cross his arms on his chest and then sit down. In the presence of cerebellar pathology due to the absence of joint contraction of the gluteal muscles, the patient cannot fix the legs and pelvis on the support area, as a result, the legs rise and he cannot sit down. The significance of this symptom should not be overestimated in elderly patients, in people with a loose or obese abdominal wall.

Ataxia is a disorder of movement coordination, which usually causes impaired gait and balance (statics), but is not associated with paralysis. Sensitive, vestibular, cerebellar, frontal and psychogenic ataxia are distinguished.

Sensory ataxia is caused by damage to fibers of deep sensitivity that carry information about the position of the body in space. This variant of ataxia can be associated with polyneuropathy (for

example, diphtheria), diseases of the spinal cord, affecting its posterior columns, in which fibers of deep sensitivity go to the brain (for example, in multiple sclerosis, vascular or tumor diseases), or a combination of these 2- uh types of disorders (for example, with a lack of vitamin B12). Distinctive properties of sensitive ataxia: deterioration of gait in the dark, detection during examination of disturbances of deep sensitivity (joint-muscle sensation, vibration sensitivity), significant deterioration of balance in the Romberg position when eyes are closed, decrease or loss of tendon reflexes. A patient with sensitive ataxia walks cautiously, trying to keep his own step under visual control and feeling a "cotton pillow" under his foot, raises his leg high and forcefully lowers it to the floor with the entire sole ("stamping gait").

Vestibular ataxia is usually accompanied by damage to the vestibular apparatus of the inner ear or the vestibular nerve (peripheral vestibulopathy), less often to the trunk vestibular structures. It is usually accompanied by rotational vertigo, nausea or vomiting, nystagmus, and occasionally hearing loss. The expression of ataxia increases with a certain position of the head and body, when turning the eyes. Patients avoid sudden movements of the head and carefully change the position of the body. In some cases, there is a tendency to fall towards the affected labyrinth. Coordination in the hands is not impaired.

Cerebellar ataxia occurs when the cerebellum or its connections are damaged. The patient stands and walks, spreading his legs wide and swaying from side to side and back. Elimination of visual control (closing the eyes) does not significantly affect the severity of coordination disorders. Mild cerebellar ataxia can be detected by asking the patient to walk along one lane, putting the heel of one to the toe of the other (tandem gait). With the selective involvement of the middle part of the cerebellum, statics and walking are isolated in isolation, with the involvement of the hemispheres, hemiataxia develops on the side of the lesion, which is manifested by a violation of the accuracy of movements and intentional (occurs in the final stage of purposeful movement) tremor, which is detected during the thumb-nose and knee-heel test. Acute cerebellar ataxia can be caused by intoxication with pharmaceuticals (lithium products, diphenine, barbiturates, carbamazepine, ethosuximide, benzodiazepines), heart attack, viral encephalitis, cerebellar abscess. Subacute cerebellar ataxia develops with Hey-Wernicke's encephalopathy, which is also manifested by ophthalmoplegia and impaired consciousness and is associated with vitamin B1 deficiency (with alcoholism, inadequate parenteral nutrition, uncontrollable vomiting), mercury poisoning, organic solvents, synthetic glue, gasoline or cytostatics , multiple sclerosis, subdural hematoma. A prerequisite for progressive acquired cerebellar ataxia may be paraneoplastic degeneration of the cerebellum (remote effect of cancer of the lungs, breast, ovaries), hypothyroidism, alcoholic degeneration of the cerebellum, tumors of the posterior cranial fossa, Arnold-Chiari anomaly (descension of the cerebellar tonsils into the large occipital foramen), hereditary cerebellar ataxias .

Frontal ataxia is manifested by difficulties in starting movement: the patient cannot move from a place for a long time, he can hardly tear his "magnetized" legs off the floor, taking several "trial" small steps or stomping in place. Having started, he continues the movement with small steps.

Psychogenic (hysterical) ataxia is expressed in unusual gait configurations that are not usually observed in organic damage to the nervous system. Patients can walk on a broken lane, slide like a skater on an ice rink, cross their legs while walking in a "braid" style, walk on straightened and spread legs, like on stilts.

Materials for self-control of training quality

Tests

1) Flexig and Gowers pathways bring information to the cerebellum from:

Answer options:

a) occipital part of the brain;

b) the frontal part of the brain;

c) anterior horns of the spinal cord;
d) joints, muscles, tendons, periosteum, ligaments
e) parietal area of the brain.
Correct answer: d)

2) The second neurons of the frontal pathway of the bridge end:
Answer options:
a) in the cortex of the cerebellar hemisphere of its side;
b) in the cortex of the cerebellar hemisphere of the opposite side;
c) cerebellum in a worm;
d) in the Varoliev Bridge;
e) in the dentate nucleus of the cerebellum
Correct answers: b)

3) Which of the listed symptoms emphasize damage to the cerebellum?
Answer options:
a) paralysis;
b) violation of coordination of movements;
c) nystagmus;
d) dysfunction of the pelvic organs;
f) loss of deep sensitivity
Correct answer: b, c)

4) Which of the listed symptoms emphasize damage to the spinal cord?
Answer options:
a) paralysis;
b) violation of coordination of movements;
c) nystagmus;
d) dysfunction of the pelvic organs;
f) loss of deep sensitivity
Correct answer: a, d)

Tasks for self-control :

1. Patient K., 44 years old, complains of unsteady gait, uncertainty when performing precise movements, impaired speech fluency. Objectively: horizontal nystagmus, slurred speech, intentional tremor when performing finger-nose and heel-knee tests, adiadochokinesis, ataxic gait. Romberg sways with open and closed eyes. Muscular hypotonia. Deep sensitivity is not disturbed. What type of ataxia does this patient have? Where is the lesion located?

Correct answer: cerebellar system; focus - in the cerebellum, or in the cerebellar pathways.

2. Patient V., 40 years old, gradually began to feel pain in his legs, a feeling of “crawling ants” appeared in them, dull pain in the feet and lower leg muscles, he began to wobble while walking, the feeling of stiffness under his feet disappeared - he walked, like walking on a thick carpet, the legs got tired quickly. At the same time, there was a feeling that something was burning in the tongue. Objectively: cranial nerves without pathology. Romberg's symptom is positive. Toe-nose and heel-knee tests are performed with a miss. Muscle strength in the legs is reduced to 3 points. Musculoskeletal and vibrational sensitivity in the toes are impaired. Knee and Achilles reflexes are revived. Babinski's pathological reflexes are caused on both sides.

Blood analysis: hemoglobin - 60 g/l, erythrocytes - 1.7 x 10 /l, leukocytes 5.4 x 10 /l, color index 1.1; platelets 78100. Anisocytosis, poikilocytosis. RV is completely negative. Total acidity of gastric juice = 0.

Urine analysis: no pathology.

Analysis of cerebrospinal fluid: clear, colorless, protein reactions are negative. The amount of protein is 56, cytosis is 2/3. The pressure of the cerebrospinal fluid in the supine position of the patient is 180 mm. water Art.

Question:

1) How is it possible to evaluate blood test data?
2) What diagnosis can be made based on the clinical picture?
3) Make a differential diagnosis?
4) What is sensitive ataxia?
5) How to check muscle and joint sensation?

Correct answers:

1) The patient has Addison-Birmer hyperchromic anemia.
2) Funicular myelosis.
3) It is necessary to differentiate with tuberculosis of the spinal cord. The diagnosis is based on blood test data, a negative Wasserman reaction, a normal cerebrospinal fluid analysis, and the presence of pyramidal symptoms (hyperreflexia, pathological Babinski reflex).
4) Sensory ataxia is a loss of muscle-joint sensation.
5) Passive movements are performed in the joints of the hands and feet of the patient with closed eyes, he must correctly name the direction of these movements and recognize which fingers and toes the researcher is moving.

Materials for classroom self-training:

List of educational practical tasks:

1. Master the ability to research:
   - gait of the patient;
   - finger-nose test;
   - heel-knee test;
   - nystagmus;
   - chanted language;
   - a test for the presence of adiadochokinesis;
   - Babinski's cerebellar test;
   - Stewart-Holmes test.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 5

Topic: Sensitive system and symptoms of its damage. Types and types of sensitivity disorders.

Purpose: applicants should have an idea of reflex arcs, know that there are many receptors, irritations of which create various sensations: pain, touch, hot, cold, etc. Therefore, it is necessary to learn the methodology of sensitivity research, to be able to determine the level of damage to sensitive conductive pathways.

Basic concepts: the complex of afferent systems, which provide information to the central nervous system, is combined with the concept of reception. The term "sensitivity" is narrower than "reception". Only that part of the reception that is felt is attributed to sensitivity. Through the receptors, any irritations are transformed into nerve impulses, which reach the corresponding areas of the cortex with the help of conductors, as a result of which sensations arise. Thanks to sensations, concrete images are formed that reflect objective reality.

Equipment : classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).

2. Control of basic knowledge (written work, written test, frontal survey, etc.):

- Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units).

Content

The term "sensitivity" refers to a person's ability to feel pain, touch, temperature, the position of the body and its segments in space, the movements of the body and its parts, etc., that is, the ability to transform stimuli from the external and internal environment into corresponding sensations.

The function of sensitivity is provided by the nervous system - its special formation, which is called "sensitive analyzer". It has a complex structure, but for simplification, three links are distinguished: the receptor apparatus (histological structures that transform the mechanical, thermal, chemical energy of the stimulus into coded impulses for further transmission), the conductor system (which transmits impulses along the nerve chain) and the central part of the analyzer (located in the cerebral cortex, where the feeling of a certain modality directly arises and is realized by a person).

Receptors are located throughout the body, its surface and in the depth of tissues and are specific for each type of stimulus produced by human evolution.

When irritated, the receptor apparatus generates nerve impulses (in the sequence of which the parameters of the stimulus are coded), which are transmitted in the ascending direction (afferently) to the sensitive nerve cells (I sensitive neuron), which are located in the ganglia on the posterior sensitive root of the spinal cord or in the ganglia of sensitive cranial cranial nerves (for the head area – trigeminal nerve, glossopharyngeal, vagus nerves). Sensitive fibers of peripheral (spinal and cranial) nerves serve to conduct impulses.

The variety of receptors provides a person's ability to feel the effects of the most different stimuli in terms of physical properties - from the external and internal environment. But physiologically, impulse flows from different receptors are grouped and transmitted together, along separate paths. Thus, three groups of receptors are distinguished - exteroceptors (in the skin, mucous membranes - for perceiving mainly irritations from the external environment), proprioceptors (embedded in the elements of the human motor apparatus - muscles, tendons, joint capsules, etc. and signal the state of the motor apparatus , the position in space of individual segments of the body and the direction and speed of their movement) and interoceptors (embedded in internal organs, mainly the chest and abdominal cavity, the walls of blood vessels). Although the latter function constantly, providing the afferentation necessary in the processes of regulation of vital activity, they are not consciously perceived by a person (in the state of pathology – a universal feeling of pain, due to free nerve endings in tissues and organs).

For clinical purposes, all the various sensory modalities are combined into two types of sensitivity - superficial and deep. The first type (superficial) includes mainly exteroceptive types of sensitivity - tactile, pain and temperature. The second (deep sensitivity) includes proprioceptive types - joint-muscle sensation, vibration sensitivity, weight and pressure sensation. That is, receptors for surface types of sensitivity are mainly located in the skin and mucous membranes, and receptors for deep types of sensitivity are located deep in the tissues of the motor apparatus (muscles, joints, periosteum and bones, tendons). This division is caused primarily by the anatomical features of the conducting apparatus: there are two paths of central conduction of sensitivity, one of which (the spino-thalamic pathway) provides conduction of mainly superficial types of sensitivity, and the other (pathways of Goll and Burdach) – mainly deep ones.

For the purposes of neurological diagnosis and determining the localization of damage to the elements of the nervous system (including those responsible for conducting sensitive impulses), it is important to establish the type of disorders and the area where they appear. The following terms are used to quantify sensitivity disorders: noresthesia (normal sensitivity), hypoesthesia (reduced sensitivity), anesthesia (complete lack of sensitivity), hyperesthesia (increased sensitivity). Each

of these terms can be applied in relation to the sensitivity of a certain modality (for example: pain hypoesthesia, temperature anesthesia, etc.). Qualitative changes in sensitivity, complex in pathogenesis, with changes in the emotional component of the response, often spontaneous sensations related to the characteristics of pain, are indicated by the terms: allodynia, hyperpathy, dysesthesia. These terms are included in the description of the concept of "neuropathic pain", which, unlike acute and simple chronic pain, does not reflect tissue damage, but is associated with certain changes in the structure and functioning of both central and peripheral sensitive nerve elements.

The areas where sensitivity disorders are detected can be described in anatomical terms (on the arm, on the leg, on the chest, etc.). But in the final case, taking into account the somatotopic organization of the innervation of the body, the territorial type of violations should be established, which corresponds to the probable localization of the damage in one or another nervous structure.

For example, if a certain peripheral nerve is damaged, then the area of sensitivity disorder will have a pattern that corresponds to the innervation zone of this nerve. This will be a mononeuritic type of sensitivity disorder.

When a plexus or part of it is damaged, several nerves are involved (plexal type of disorder).

In case of damage to the posterior sensory root or the posterior horn of the spinal cord, the area of disorders will differ from that in case of damage to peripheral nerves - it will be a segmental type of disorders.

There is also a specific pattern of disorders in the case of damage to the conductive paths of sensitivity in the spinal cord and brain - the conductive type of disorders (will be discussed further).

To describe the territory of disorders (for example, hypo- or anesthesia), other terms are used: mono- (disorder on one limb), para- (disorder on two limbs of the same name, arms or legs), hemi- (on one half of the body), tetra- (on all four limbs and covering the body).

The method of sensitivity research consists in the fact that a certain irritation is applied to the patient by the doctor and the patient answers what he feels and where. The research methodology includes a certain set of conditions and sequences of examination. First, the patient must be conscious (otherwise we will not know what he is feeling). Second, the patient must receive instructions and an explanation of what is happening, what the doctor will do, and what he or she must answer. As a rule, when examining sensitivity, the patient should close his eyes to avoid subjective control and achieve greater objectivity. Further, the research methods of individual types of sensitivity differ, depending on their modality.

In the study of tactile sensitivity, irritation (usually with a stick or a match) is applied to the skin in the form of touch. The patient to the question "what did I do?" must answer "touched" or "not touched", "nothing" (if he did not feel irritation, or the doctor really did not apply it - eyes closed!). At the same time, the answer to the question "where did I touch?" is received. The areas of the face, arms, trunk and legs are evaluated sequentially. At the same time, the right side is compared with the left, the proximal parts of the limbs with the distal ones, as well as the upper half of the body with the lower - to identify the territory of the disorder and its pattern. The clinical value of tactile sensitivity is small due to the lack of quantitative changes (answer "yes" or "no"), as well as the fact that tactile sensitivity is carried out by both central conducting sensitive pathways and is actually preserved when one of them is damaged.

The study of pain sensitivity is more important. The irritation is applied with a needle or a pointed stick. The intensity of the irritation should be sufficient for a painful sensation ("sharp"), but without tissue damage (the doctor pre-selects the intensity for himself). A short (2-3) series of injections is usually applied. The patient must answer whether he feels the irritation as "sharp" (norm), or as a touch with a blunt object (hypesthesia), or does not feel anything at all (anesthesia). All parts of the body are examined sequentially to identify the pattern of the disorder.

Temperature sensitivity is examined by touching the skin with either two tubes of warm and cold water alternately, or the metal and rubber parts of a neurological hammer (perceived as "cold" and "warm"). The goal is to determine whether a person is able to differentiate the temperature

difference on individual areas of the skin. Temperature sensitivity, as a rule, suffers simultaneously with pain sensitivity, especially with peripheral damage.

Joint-muscular sensation (deep sensitivity) is defined as the ability of a person with closed eyes or in the dark to feel the position of his body in space, the relative position of body parts, as well as the direction and speed of movements of body parts. Based on this definition, the research method consists in applying a specific stimulus to the receptors embedded in the deep tissues of the movement apparatus and obtaining the patient's response - what does he feel or what do we do with his body parts, with which part, where do we move, etc. With the patient's eyes closed, the doctor takes his finger and passively flexes or extends it. At the same time, the patient must determine which finger the doctor took and what he is doing with it at the moment, bending or unbending, moving it to the side, etc.

In the case of a gross violation of the articular-muscular sensation in the fingers, the doctor continues the examination in larger, proximal joints (wrist, ankle-foot and further), determining the level of which joint there is a violation.

It should be emphasized that, in contrast to disorders of surface sensitivity, disorders of deep (joint-muscular sensation, vibration sensitivity) lead to obvious disorders in the motor sphere - sensitive ataxia (that is, disorders of coordination of movements, staggering when walking, etc., due to insufficient deep sensitivity).

The study of vibration sensitivity is carried out with the help of a tuning fork (with a frequency of oscillations of 64 Hz), the leg of which is installed on the areas of bones lying directly under the skin. Not only the ability to feel vibration is noted, but also to compare the sensitivity of individual segments of the body by measuring the time during which the patient still hears the vibration.

Damage levels of the sensitive analyzer and resulting syndromes

Peripheral nerve	unilaterally	Anesthesia of all types of sensitivity in the zone of nerve innervation
Posterior root of the spinal cord	unilaterally	Anesthesia of all types of sensitivity in the innervation zone of the root (segment)
Spinal ganglion	unilaterally	Anesthesia of all types of sensitivity in the innervation zone of the root (segment); herpetic rash, shooting pains
Posterior horn of the spinal cord	unilaterally	Dissociated sensitivity disorder - pain and temperature anesthesia with preservation of deep and tactile sensitivity
Anterior gray adhesion	bilaterally	Disorders of surface sensitivity, segmental, symmetrical
Lateral column of the spinal cord	contralaterally	The leading type of disorder (from the level of the lesion to the bottom) is loss of pain and temperature sensitivity
The posterior column of the spinal cord	unilaterally	The leading type of disorder is loss of deep sensitivity
Sensitive pathways in the brain stem	contralaterally	Loss of all types of sensitivity on the opposite half of the body -hemianesthesia

Visual hump	contralaterally	Loss of all types of sensitivity on the opposite half of the body - hemianesthesia, hemianopsia, sensitive hemiataxia
Inner capsule	contralaterally	Loss of all types of sensitivity on the opposite half of the body - hemianesthesia, hemianopsia
Ray crown	contralaterally	Loss of all types of sensitivity on the opposite half of the body - monoanesthesia
Extracentral gyrus	contralaterally	Loss of all types of sensitivity on the opposite half of the body is monoanesthesia. With irritation - Jackson's sensitive epilepsy

Brown-Sécart syndrome is a symptom complex observed when half of the diameter of the spinal cord is damaged: on the side of the lesion below the level of the lesion, central paralysis (or paresis) and loss of muscle-joint and vibration sensitivity are noted, on the opposite side - loss of pain and temperature sensitivity.

The most frequent sign of irritation of sensitive neurons is spontaneous pain and paresthesias. There is no usual definition of pain. According to the opinion of many doctors, biologists, psychologists, it is hardly possible to give a complete definition of pain (there is a saying - "pain and pleasure cannot be defined"). The definition of the International Association for the Study of Pain is as follows: pain is an unpleasant sensory and emotional experience associated with existing or possible tissue damage. The nature of pain can be judged only by the patient's complaints. This subjectivity of pain assessment led to the need to introduce the concept of nociceptive (lat. nocere - to harm) and antinociceptive reactions in response to the action of stimuli that can cause damage to the body. Pain receptors are stimulated by serotonin, histamine, prostaglandins, kinins, substance P and others. Enkephalins, opiates, gamma-aminobutyric acid and others inhibit the activity of pain neurotransmitters.

Pain is divided into local (local), projection, radiating and reflected (reflex). Local pain occurs in the area of the existing painful irritation, but often the localization of the pain does not coincide with the location of the existing irritation. This is often observed in radiculitis, when the posterior root suffers and as a result, sharp pain occurs in the innervation zone of this root. Such pain is called projection pain. An example can also be the so-called phantom pains in persons who have undergone limb amputation. Pain can be radiating, that is, it spreads from one branch of the nerve, which is irritated by the pathological process, to another, free from direct irritation. The result of the same irradiation of irritation is reflected pain in diseases of internal organs. Pathological impulses from the receptors of internal organs, entering the posterior horn of the spinal cord, excite the conductors of pain sensitivity of the corresponding dermatomes, where the pain spreads. This is called a viscero-sensory phenomenon, and the area of the skin where the pain is projected is the Zakharyin-Ged zone. In these zones, not only pain can be determined, but also increased sensitivity. Paresthesias are peculiar feelings of crawling ants, tingling, numbness experienced by patients. tightening, itching that occur without applying external irritants. During the examination of the patient, as a result of interruption of the conductors of sensitivity, symptoms of loss appear - loss of sensitivity, which is called anesthesia, and its decrease - hypoesthesia. The loss of pain sensitivity is defined by the term analgesia, a decrease in hypalgesia. Excessive irritation of sensitive conductors can be observed, and then the injected injections are perceived as very intense during the examination. In such cases, one speaks of hyperesthesia, and when determining pain sensitivity, one speaks of hyperalgesia. In the clinic, you can often meet with a peculiar form of pain sensitivity disorder - hyperpathy. It is characterized by an increase in the

perception threshold. The patient does not feel single injections, but a series of injections (5-6 or more) causes intense and burdensome pain. Causalgia is paroxysmal burning pain that is aggravated by light touch, negative emotions and is localized in the area of the affected peripheral nerve. Causalgia more often occurs with partial damage to the trunks of the median and tibial nerves. Pain can occur in response to compression, tension of a nerve or root, and this pain is called reactive. A disorder of pain sensitivity often coexists with a violation of temperature sensitivity. Its complete loss is defined by the term thermoanesthesia, its decrease by thermohypesthesia. Anesthesia, hypoesthesia, and hyperesthesia can also occur with disorders of tactile sensitivity. The main clinical sign of impaired deep sensitivity is the disorder of active movements (for example, the inability to walk at night, to determine the position of one's limbs), which is called sensitive ataxia. Such patients do not feel the vibration of the tuning fork. Damage to complex types of sensitivity leads to non-recognition of objects by hand in the absence of visual control.

Types of sensitivity disorders Sensitivity disorders are quantitative and qualitative. Quantitative sensitivity disorders are a decrease or increase of one or another type of sensitivity. Anesthesia is a complete loss of one or another type of sensitivity. Pain anesthesia (analgesia), temperature (thermoanesthesia), loss of localization sensation (topanesthesia), muscle and joint (bathanesthesia), etc. are distinguished. Loss of all types of sensitivity is called total anesthesia. Hypoesthesia is a decrease in one or another type of sensitivity. Hyperesthesia - increased sensitivity to various types of irritation .

Complex types of sensitivity include the ability to localize stimuli (sensation of localization), the ability to distinguish two simultaneously applied stimuli as two, and not as one (sensation of discrimination), and the ability to recognize with closed eyes geometric figures, letters, numbers drawn on the surface of the skin with a stick (spatial - two-dimensional feeling). Separately, a complex sensitive ability of a person is distinguished - stereognosis - the ability to recognize an object by touch (in the implementation of which almost all simple types of sensitivity participate). The analytical and synthetic center for this sensation lies in the upper parietal lobe of the brain, and when it is damaged, astereognosis occurs - the inability to recognize an object while retaining the ability to describe its properties (weight, shape, surface, etc.).

As indicated, superficial and deep sensitivity are carried out in two different ways. The first sensitive neuron is located on the periphery - in the sensitive ganglia of the posterior roots of the spinal cord or in similar nodes of cranial nerves. The neuron has one process that divides in a T-shape: the dendrite goes as part of the peripheral nerves to the corresponding areas of the skin and mucous membranes, and the axon goes in the direction of the spinal cord as part of the posterior sensory root. Then the paths diverge.

Fibers carrying surface sensitivity (tactile, pain, and temperature) enter the posterior horn of the spinal cord and contact (end at) the second sensitive neuron located here. The axon of the latter passes to the opposite side of the spinal cord through the anterior gray conjunctiva (in front of the central canal). Thus, the fibers cross on both sides in the adhesion.

But the crossing does not occur in the plane of the entrance of the fibers, but obliquely upwards, on 1-2 segments of the spinal cord (!). Having passed this segmental junction, fibers of surface sensitivity enter the lateral column of the spinal cord and form **the spino-thalamic pathway** , which thus carries information from the opposite half of the body. The path goes up the spinal cord, absorbing all the new fibers, passes through the brain stem (receiving the corresponding sensory fibers from the face and head, from the cranial nerves) and ends in the ventro-medial nuclei of the optic hump (thalamus), where the body lies the third sensitive neuron.

Fibers of deep sensitivity with a part of tactile (the latter goes with both paths), which go from the first neuron in the spinal ganglion, do not enter the gray matter of the spinal cord, but enter directly into the posterior column of their own side, forming the paths of Gol and Burdach. They rise up to the nuclei of the delicate and sphenoid bundles (Gol and Burdach tracts), which lie in the dorsal part of the medulla oblongata. This is already the second neuron of deep sensitivity. After the second neuron, a crossing should take place, so the fibers go to the opposite side and

form a path that is called the medial loop along the brain stem (medulla, pons, peduncles). The third neuron naturally lies in the optic hill.

Then all the sensitive fibers go together: the axons of the third neuron pass through the internal capsule (in the back third of its posterior thigh), branch widely in the radial crown and end in the extracentral gyrus and cortex of the parietal lobe of the brain.

The mechanism of pain.

Pain receptors or nociceptors are free nerve endings located in the skin, mucous membranes, muscles, joints, bones and internal organs. Sensitive endings belong either to pulpless or thin myelinated fibers, which determines the speed of signal transmission in the CNS and gives rise to the distinction between early pain, short and sharp, which occurs when impulses are conducted at a higher speed along myelin fibers, as well as late, dull and long-lasting pain pain, in the case of conducting signals along pulpless fibers. Nociceptors belong to polymodal receptors, as they can be activated by stimuli of various nature: mechanical (blow, cut, injection, pinch), thermal (action of hot or cold objects), chemical (change in the concentration of hydrogen ions, action of histamine, bradykinin and a number of other biologically active substances). The sensitivity threshold of nociceptors is high, so only sufficiently strong stimuli cause excitation of primary sensory neurons: for example, the threshold of pain sensitivity for mechanical stimuli is about a thousand times higher than the threshold of tactile sensitivity. Central processes of primary sensory neurons enter the spinal cord as part of the posterior roots and form synapses with neurons of the second order located in the posterior horns of the spinal cord. Axons of second-order neurons go to the opposite side of the spinal cord, where they form spinothalamic and spinoreticular tracts. The spinothalamic tract ends at the neurons of the lower posterolateral nucleus of the thalamus, where the leading paths of pain and tactile sensitivity converge. Neurons of the thalamus form a projection on the somatosensory cortex: this pathway ensures the conscious perception of pain, allows determining the intensity of the stimulus and its localization.

The fibers of the spinoreticular tract end on neurons of the reticular formation interacting with the medial nuclei of the thalamus. During painful excitation, neurons of the medial nuclei of the thalamus have a modulating effect on large regions of the cortex and structures of the limbic system, which leads to an increase in human behavioral activity and is accompanied by emotional and vegetative reactions. If the spinothalamic path serves to determine the sensory qualities of pain, then the spinoreticular path is intended to play the role of a general alarm signal, to exert a general excitatory effect on a person. The subjective assessment of pain is determined by the ratio of neuronal activity of both pathways and the activation of antinociceptive descending pathways depending on it, which can change the nature of transmission of signals from nociceptors. An endogenous mechanism of its reduction is built into the sensory system of pain sensitivity by regulating the threshold of synaptic switching in the posterior horns of the spinal cord ("pain gate"). The transmission of excitation in these synapses is influenced by the fibers of gray matter neurons around the aqueduct, blue spot and some nuclei of the median seam. Mediators of these neurons (enkephalins, serotonin, norepinephrine) inhibit the activity of second-order neurons in the posterior horns of the spinal cord, thereby reducing the transmission of afferent signals from nociceptors.

Antinociceptive system.

Antinociceptive system provides reduction of pain sensations inside the body. The antinociceptive system releases biologically active endogenous opioid substances - these are "internal drugs". They are called endorphins, enkephalins, dynorphins. According to their chemical structure, all of them are short peptide chains, i.e. they consist of amino acids. Hence the name: neuropeptides, opioid peptides. Opioid - i.e. similar in effect to opium poppy narcotic substances. Many neurons of the pain system have special molecular receptors for these substances. When opioids bind to these receptors, presynaptic and/or postsynaptic inhibition occurs in neurons of the pain system. The pain nociceptive system is inhibited and reacts weakly to pain.

Pathways of the antinociceptive system. The antinociceptive system acts in several ways:

Urgent mechanism.

It is excited by the action of painful stimuli, uses a system of descending inhibitory control. It quickly limits afferent nociceptive excitation at the level of the posterior horns of the spinal cord. This mechanism is involved in competitive analgesia (analgesia), i.e. the pain response is suppressed if another painful stimulus acts simultaneously.

Short-acting mechanism.

It is triggered by the hypothalamus, involves the descending inhibitory control system of the middle, medulla oblongata, and spinal cord. This mechanism limits painful excitation not only at the level of the spinal cord, but also higher, activated by stressogenic factors.

Long lasting mechanism.

It is activated during long-term pain. Its centers are located in the hypothalamus. The downward brake control system is retracted. This mechanism limits the upward flow of pain excitation at all levels of the nociceptive system. This mechanism connects the emotional evaluation and adds an emotional color to the pain.

Tonic mechanism.

Supports constant activity of the antinociceptive system. Its centers are located in the orbital and frontal regions of the cortex. Provides a constant inhibitory effect on the activity of the nociceptive structure at all levels. It is important to note that this happens even in the absence of pain. Thus, with the help of antinociceptive structures of the cortex of the large hemispheres of the brain, it is possible to prepare in advance and then, under the influence of a painful stimulus, reduce painful sensations.

Interaction of pain and anti-pain systems. So, we come to the conclusion that the strength and nature of pain sensations are the result of the work of not one system, but two systems: pain (nociceptive) and antipain (antinociceptive). Their interaction with each other determines exactly what kind of pain a person will feel.

Materials for self-control of training quality

Tests

1) All paths pass through the legs of the cerebrum, except:
Answer options:
a) fronto-bridge;
b) tuberospinal;
c) cortico-spinal;
d) occipital-parietal-temporal-bridge
e) visual
Correct answer: b)

2) Complex types of sensitivity include all of the following:
Answer options:
a) stereognosis;
b) two-dimensional spatial feeling;
c) sense of mass;
d) feeling of pressure;
e) pain sensitivity.
Correct answers: e)

3) Where is the I neuron of the surface sensitivity pathway located?
Answer options:
a) in the front central gyrus;
b) in the posterior central gyrus;
c) in spinal intervertebral ganglia;
d) in the anterior horns of the spinal cord;
e) in the posterior horns of the spinal cord.

Correct answer: c)

4) What types of sensitivity are classified as superficial?

Answer options:

a) articular-muscular sensation;
b) tactile sensitivity;
c) pain sensitivity;
d) temperature sensitivity;
e) vibration sensitivity.

Correct answer: b, c, d)

5) What types of sensitivity are classified as deep?

Answer options:

a) articular-muscular sensation;
b) tactile sensitivity;
c) pain sensitivity;
d) temperature sensitivity;
e) vibration sensitivity.

Correct answer: a, b, e)

6) Where is the I neuron of the path of deep sensitivity located?

Answer options:

a) intervertebral spinal ganglia;
b) Golya and Burdakh nuclei;
c) posterior horns of the spinal cord;
d) posterior columns of the spinal cord;
e) visual hill.

Correct answer: a)

7) Where is the II neuron of the path of deep sensitivity located?

Answer options:

a) intervertebral spinal ganglia;
b) Golya and Burdakh nuclei;
c) posterior horns of the spinal cord;
d) posterior columns of the spinal cord;
e) visual hill.

Correct answer: b)

8) What sensitivity is disturbed when the posterior horns of the spinal cord are affected?

Answer options:

a) painful;
b) temperature;
c) tactile;
d) vibrational;
e) articular-muscular sensation.

Correct answer: a, b, c)

9) What sensitivity is disturbed with a transverse lesion of the spinal cord?

Answer options:

a) painful;
b) temperature;
c) tactile;
d) vibrational;
e) articular-muscular sensation.

Correct answer: a, b, c, d, e)

Tasks for self-control :

1. What type of sensory impairment is observed when the posterior horns of the spinal cord are affected?
   Correct answer: dissociated segmental posterior horn of the half-jacket type.
2. What types of sensory disturbances occur when half of the spinal cord is damaged?
   The correct answer: Brown-Sekariv type of violation: deep sensitivity will fall on the side of the focus, and superficial - on the opposite side.
3. What will happen if the posterior columns of the spinal cord are damaged?
   The correct answer is: sensitive ataxia.
4. How will the sensitivity spread when the optic hill and internal capsule are affected?
   Correct answer: hemianaesthesia and hemiataxia will be observed contralaterally.
5. Patient V., 50 years old. He has been suffering from diabetes for 8 years. During the last 3 months, he began to feel mild pain, numbness in his legs, "crawling ants". He did not turn to the doctor. Gradually, similar sensations arose in the upper limbs.

Objectively: cranial nerve pathology was not detected. The range of motion is not limited. Tendon and periosteal reflexes on the hands are reduced. Knee reflexes are too low, Achilles reflexes are not triggered. Superficial and deep sensitivity on the hands from the level of the elbow joints, on the legs - from the upper third of the lower leg to the bottom.

Question:

1) What type of sensitivity disorder does the patient have?
2) What are the sensitivity testing rules?
3) In what cases does this type of sensitivity disorder occur?
4) What sensitivity disorders do you know?

Correct answers:

1) Distal polyneurotic, in the form of "gloves" and "stockings".
2) From top to bottom; in symmetrical sections; on the limbs - in a circle; turn off visual control.
3) With simultaneous symmetrical damage to many peripheral nerves of the limbs in the distal parts - polyneuritis, polyneuropathies.
4) Mononeural, plexus, root, segmental, dissociated, conduction, Brown-Sekarivsky, cortical, functional.

Materials for classroom self-training:

List of educational practical tasks:

1. Master research methods:
   a) pain sensitivity;
   b) temperature sensitivity;
   c) tactile sensitivity;
   d) muscle and joint sensation;
   e) vibration sensitivity;
   f) feeling of pressure and weight;
   g) stereognosis

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 6

Topic: Cranial nerves I, II, VIII and syndromes of their damage

Purpose: applicants should have an idea of the functions of special analyzers and oculomotor nerves, know their reflex arcs and symptoms of damage, which can be detected only with proper careful examination. Therefore, they should master professional skills with the aim of using them in further professional activities.

Basic concepts: damage to the conducting pathways and cortical ends of special analyzers (olfactory and visual) and auditory nerves in diseases of the nervous system is reflected in clear symptoms that make it possible to determine the topography of the focus, make a clinical diagnosis and prescribe adequate therapy.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
    - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

The first pair is the olfactory nerve (sensitive nerve): basic anatomical and physiological data.

Research of the olfactory analyzer.

Damage syndromes - hyposmia, anosmia, hyperosmia, olfactory hallucinations.

The second pair is the optic nerve (sensory nerve).

Anatomical and physiological features: departments - peripheral (rods and cones, bipolar cells, ganglion cells, the nerve itself, chiasm, optic tract), central (lateral geniculate bodies, upper tubercles of the quadrihepa, cushion of healthy tubercle (subcortical centers), bundle of Graziole, spur furrow of the occipital lobe (cortical center of the analyzer).

Symptoms of damage: amaurosis, amblyopia, homonymous and heteronymous hemianopsia (binasal, bitemporal), visual hallucinations. Changes in the optic disc (changes in the fundus).

The eighth pair is the sphenoid nerve (sensitive).

Anatomical and physiological data, cochlear and vestibular nerves. Pathology of the cochleo-vestibular apparatus: damage to the sound-receiving apparatus (hearing disorder for high tones), damage to the sound-conducting apparatus (hearing disorder for low tones); lesions of the temporal lobe (dizziness, nystagmus, impaired balance, coordination of movements, autonomic disorders, lesions of the temporal cortex (in case of irritation - auditory hallucinations).

Analyzers

Each analyzer consists of three parts: 1) peripheral (receptor), which perceives signals from the external environment and converts them into nerve impulses; 2) conductive (conductive path), along which impulses are directed to the corresponding nerve center; 3) central (the center of the analyzer in the cortex).

Olfactory nerve (n. olfactorius).

Receptor olfactory cells are scattered in the epithelium of the mucous membrane of the olfactory region of the nasal cavity. Thin central processes of these cells gather into olfactory threads, which are the olfactory nerve itself. From the nasal cavity, the nerve enters the skull cavity through the openings of the ethmoid bone and ends in the olfactory bulb. The central olfactory pathways begin from the cells of the olfactory bulb to the cortical area of the olfactory analyzer in the temporal lobe of the brain. Bilateral complete loss of smell (anosmia) or its reduction (hyposmia) is often the result of a disease of the nose or has a congenital nature (sometimes in this case it is combined with some endocrine disorders). Unilateral olfactory disorders are mainly associated with the pathological process of the anterior cranial fossa (tumor, hematoma, traumatic brain injury, etc.) Unusual paroxysmal olfactory sensations (parosmia), often of some unspecified unpleasant smell, are harbingers of an epileptic attack caused by irritation of the temporal lobe of the brain . Irritation of the cortex of the temporal lobe of the brain can cause various olfactory hallucinations.

Research methodology. Olfactory research is conducted using a special set of aromatic substances (camphor, mint, valerian, pine extract, eucalyptus oil). The examinee, with his eyes closed and one half of his nose pinched, is presented with odorous substances and asked to say what smell he feels, whether he perceives smells equally well with each nostril separately. It is not

possible to use substances with strong odors (rubbing alcohol, acetic acid), because in this case irritation of the endings of the trigeminal nerve occurs, so the results of the study will be inaccurate.

Symptoms of damage. They differ depending on the level of damage to the olfactory nerve. The main ones are loss of smell - anosmia, decrease of smell - hyposmia, increase of smell - hyperosmia, deviation of smell - dysosmia, olfactory hallucinations. A unilateral decrease or loss of smell is mainly important for the clinic, because bilateral hypo- or anosmia is caused by the phenomena of acute or chronic rhinitis.

Hyposmia or anosmia occurs when the olfactory pathways to the olfactory triangle are affected, that is, at the level of the first and second neurons. Due to the fact that the third neurons have a cortical representation both on their own side and on the opposite side, damage to the cortex in the olfactory projection field does not cause loss of smell. However, in cases of irritation of the bark of this area, the sensation of non-existent odors may occur.

The proximity of the olfactory filaments, the olfactory bulb and the olfactory tract to the base of the skull leads to the fact that the sense of smell is disturbed during pathological processes in the basal parts of the skull and brain.

Visual analyzer. The peripheral part of the visual analyzer is represented by the eyeball and the auxiliary apparatus of the eye.

The eyeball is located in the recesses of the skull - the eye socket. It is covered with three shells. The outer shell — protein — consists of collagen fibers and protects the eye from mechanical damage. In front, it passes into the transparent cornea. Under the protein layer is the vascular membrane, penetrated by a dense network of blood vessels. In front, the choroid becomes the iris, the pigments of which determine the color of the eyes. In the center of the iris there is an opening - the pupil. The diameter of the pupil changes reflexively, depending on the brightness of the light. Behind the pupil is the crystalline lens - a transparent biconvex lens with a diameter of about 9 mm. Changing the curvature of the lens — accommodation — is achieved by contraction or relaxation of the ciliary muscle.

Optic nerve (n. opticus). It is formed by the axon of neurons of the ganglion layer of the retina, which exit the eyeball through the lattice plate of the sclera through a single trunk of the optic nerve into the cavity of the skull. On the basal part of the brain in the region of the Turkish saddle, the fibers of the optic nerves converge on both sides, forming the optic junction and optic tracts. The latter go to the external geniculate body and the pillow of the thalamus, then the central visual pathway goes to the cerebral cortex (occipital lobe). The incomplete crossing of the optic nerve causes the presence of fibers from the right halves in the right optic tract, and from the left half-retinas of both eyes in the left optic tract.

Symptoms of damage. With complete damage to the conduction of the optic nerve, blindness occurs on the side of the damage with the loss of the pupil's direct response to light. When only a part of the fibers of the optic nerve is damaged, focal loss of the field of vision (scotoma) occurs. When the chiasm is completely destroyed, bilateral blindness develops. However, with many intracranial processes, the defeat of the chiasm can be partial - loss of the outer or inner half-fields of vision develops (heteronymous hemianopsia). Unilateral loss of visual fields on the opposite side (homonymous hemianopsia) occurs with unilateral damage to the visual tracts and higher visual pathways.

Lesions of the optic nerve can be inflammatory, congestive and dystrophic; are detected during ophthalmoscopy. The causes of optic neuritis can be meningitis, encephalitis, arachnoiditis, multiple sclerosis, influenza, inflammation of the paranasal sinuses, etc. Decreased acuity and narrowing of the field of vision are revealed, which is not corrected by the use of glasses. Congestive disc of the optic nerve is a symptom of increased intracranial pressure or impaired venous outflow from the eye socket. With the progression of congestive phenomena, visual acuity decreases, blindness may occur. Atrophy of the optic nerve can be primary (in case of spinal tuberculosis, multiple sclerosis, trauma to the optic nerve) or secondary (as a result of neuritis or

stagnant disc); there is a sharp decrease in visual acuity up to complete blindness, a narrowing of the field of vision.

The fundus is the part of the inner surface of the eyeball visible during ophthalmoscopic examination (optic disc, retina and choroid). The disc of the optic nerve stands out on the red posterior fundus as a rounded formation with clear borders and a pale pink color. The ratio of diameters of arteries and veins is 2:3.

Color perception is assessed using the polychromatic tables of Rabkin et al.

In the back pole of the eye is the most sensitive part of the retina - the so-called yellow spot, which has the form of a horizontally located oval of a yellowish shade. The macula consists of bulbs that provide daytime vision and participate in the accurate perception of the shape, color and details of an object. As you move away from the yellow spot, the number of bulbs decreases, and the number of rods increases. The sticks have a very high light sensitivity and provide perception of objects at dusk or at night. They find out whether there are complaints about reduced visual acuity, loss of field of vision, the appearance of sparks, dark spots, flies, etc. in front of the eyes.

Visual acuity is checked separately for each eye using special tables consisting of 12 rows of letters or open rings or contour drawings. A healthy eye can distinguish letters in 10 lines from a distance of 5 meters, this visual acuity is conditionally taken as 1. For example, if the patient's eye can distinguish letters in 6 lines, visual acuity is estimated as 0.6, if in 2 lines - 0.2.

Fields of vision are examined using a special device - a perimeter, which consists of a graduated arc rotating around a horizontal axis, an arc graduated from 0 to 90º in each direction from the center. In the center of the arch, a mark is placed on the inner surface, on which the patient fixes his gaze with the other eye closed, which is not tested at this time. Visual fields are evaluated separately for each eye. The patient informs the doctor when he notices a white mark, which is moved from the middle to the center in different planes, conducting research along the meridians every 15 degrees. The points drawn on the diagrams are combined and the boundaries of the field of view are obtained. Normally, the outer limit of the field of vision is equal to 90º, the upper and inner - 50-60º, the lower - about 70º.

An approximate assessment of the field of vision is possible using a sample with a towel, the center of which is placed in front of the eye (each eye is examined separately) in a horizontal plane, the patient should divide the towel in half, indicating its center, and a control method, when the doctor and the patient sit opposite each other, close different eyes and evaluate the perception of the object in the frontal plane, while everyone looks at each other.

Hearing analyzer.

The peripheral part of the auditory analyzer is the ear, which is divided into outer, middle and inner, the conductive part is the auditory nerve, and the central part is the auditory zone of the parietal lobe of the cortex of the large hemispheres.

The external ear includes the auricle and the external auditory canal. The auricle is formed by cartilaginous tissue. The external auditory canal is S-shaped and 30-35 mm long. In the wall of the passage there are many sebaceous glands that secrete earwax. The passage ends with the tympanic membrane, which separates the outer ear from the middle ear. The tympanic membrane is elastic because it consists of collagen fibers and is only 0.1 mm thick. The middle ear is represented by the tympanic cavity filled with air. It houses the auditory ossicles — the hammer, anvil, and stirrup, articulated with each other with the help of joints. The malleus fuses with the tympanic membrane and transmits its vibrations to the anvil, which in turn transmits its vibrations to the stirrup. The stirrup touches the septum of the oval window of the inner ear. The middle ear is connected to the nasopharynx by means of auditory (Eustachian) tubes. The inner ear is formed by semicircular canals (belonging to the vestibular apparatus) and a curl (to the organ of hearing). A coil is a spiral twisted channel. The coil channel is divided by longitudinal partitions into three parts, filled with liquid - endolymph. In the middle part of the channel there is a main membrane with hair (receptor) cells located on it. Both membranes and receptor cells make up the organ of Corti. A nerve fiber of the auditory nerve goes to each hair cell. Here, the sound signal is converted

into an electrical impulse. Electrical impulses enter the auditory nerve in the parietal part of the cortex of the large hemispheres, where information is processed and decoded. Organ of balance (vestibular apparatus). The peripheral part of the vestibular apparatus consists of three semicircular canals, oriented in mutually perpendicular planes, and two pouches — oval and round. Canals and sacs are located in the inner ear. The semicircular canals are filled with endolymph. The central processes of the cells of the parietal node form the parietal part of the eighth cranial nerve — the parietal nerve, which exits the internal auditory hole and enters the brainstem in the pons-cerebellar angle at the border between the pons and the medulla oblongata, on the outside of the facial nerve. Having penetrated into the brain, most of the central processes end in four parietal nuclei, where the second neuron of the conduction path of the statokinetic analyzer is located. Part of the central processes of the cells of the parenchymal node do not enter the parietal nuclei, but through the lower cerebellar peduncle directly reach the cerebellar vermis and the nucleus of the apex.

The second neuron is the cells of the anterior and posterior convolutional nuclei of the pons. The axons of the cells of the anterior gyrus nucleus pass to the opposite side of the bridge, participating in the formation of the trapezoidal body, most of them switch in the upper olivary nucleus and the medial additional olivary nucleus. Processes of the cells of the posterior convolutional nucleus, forming the cerebral bands of the floor of the fourth ventricle, pass to the opposite side of the bridge, sink into its cover near the median furrow of the rhomboid fossa and join the fibers of the trapezoid body. A smaller part of the axons of the posterior convolutional nucleus does not go to the opposite side of the bridge, but joins the trapezoidal body, the medial upper accessory olivary nucleus of its side and, as part of a lateral loop, contacts the neurons of the lower tubercles of the plate of the roof of the midbrain and the medial geniculate body. Thus, the lateral loop contains fibers of the conducting pathways from both auditory analyzers.

The third neuron is the nucleus of the trapezoid body and the superior olive nucleus. Nerve fibers that come out of its nuclei, as well as axons of neurons of the anterior convolutional nucleus, which pass through the trapezoid body without interruption, form a lateral loop, its fibers approach the nuclei of the lower tubercles of the plate of the roof of the midbrain and the medial geniculate body. The cells of the named formations are the fourth neurons of the conductive path of the auditory analyzer.

Methodology of research of the vestibulo-cochlear nerve (hyperacusis and hypoacusis, tests of Rinne, Weber).

The patient's complaints are investigated, hearing acuity, bone and air conduction are assessed.

Hearing acuity is tested for each ear separately using whispered speech. With closed eyes, the patient must repeat the words that are spoken in a whisper from a distance of 6 m. Hearing acuity is examined in more detail with the help of audiography.

To assess damage to the sound-receiving or sound-conducting system, a tuning fork with an oscillation frequency of 128 hertz is used.

Try Rinne. The leg of the sounding tuning fork is placed on the nipple. After the patient stops hearing the vibration through the bone, the tuning fork is brought to the ear at a distance of 1-2 cm. A healthy person perceives sound through the air for 2 times longer. Such a result is evaluated as a positive Rinne test. If the Rinne test is negative, that is, the patient does not hear the sound of the tuning fork, this indicates damage to the sound-conducting apparatus.

Weber's test. The leg of the sounding tuning fork is placed in the middle of the crown, normally the sound is equally perceived by both ears, i.e. there is no lateralization of the sound. When the sound-conducting apparatus is damaged, the sound is better felt by the affected ear (lateralization in the affected ear). When the sound-receiving apparatus is damaged, the sound is better felt by the healthy ear (lateralization in the healthy ear).

Methods of research of vestibular functions.

The examination of the function of the vestibular analyzer is carried out mainly in the clinic of otolaryngology and includes an assessment of the patient's complaints and a check for the presence of spontaneous nystagmus, balance disorders, performing coordination tests, determining

the excitability of the vestibular analyzer using caloric and rotational tests. The leading complaint is systemic vertigo - a feeling of rotation of one's own body or surrounding objects in one direction (clockwise or counterclockwise). Vestibular nystagmus is an involuntary, rapidly repeating rhythmic twitching of the eyeballs.

Research of vestibular function.

Investigations of vestibular function begin with a survey and examination of the patient, and special (vestibular) tests are also used. Usually, patients with diseases of the vestibular apparatus complain of dizziness, instability, unsteady gait, sometimes - nausea and vomiting. Spontaneous vestibular disorders are often observed in such patients, i.e. symptoms that do not normally occur. Among them, the most prominent are nystagmus, coordination and balance disorders.

Spontaneous nystagmus (rhythmic twitching of the eyes) is detected by looking at the doctor's finger, which is placed at a distance of 50-70 cm from the patient's eyes directly in front of him. With one hand, the doctor holds the patient's chin so that he does not move his head. The finger of the other hand is first placed in the center of the patient's vision, fixing his gaze on the doctor's fingers, and slowly moved: horizontally - to one side, and then to the other; vertically - first up, then down. The patient is asked to look at the doctor's finger without moving his head. At the same time, the condition of the patient's eyes is assessed both in the positions of extreme abduction and in the center.

Normally, there is only establishment nystagmus - single twitches of the eyeballs, after which the eye takes a stationary, fixed position. When the function of the vestibular analyzer is impaired, there are involuntary twitches of the eyes in horizontal or arcuate directions (horizontal or horizontal-rotatory nystagmus).

According to the source of origin, nystagmus can be induced or spontaneous. Induced nystagmus occurs as a result of external irritation (swinging on a swing, turning on a Barani chair, pouring cold water into one ear, etc.); spontaneous nystagmus occurs as a result of a pathological process in the body.

Nystagmus is distinguished by 6 characteristics:

1. According to the direction (right, left, up, down, etc.), the direction is considered to be the direction of the fast phase of eye movement;

2. By plane (horizontal, vertical, rotary, mixed). The plane is determined by the plane in which the eyeballs move.

3. By amplitude (small-scale, medium-scale, large-scale).

4. By force (I degree - nystagmus occurs only when looking in the direction of the fast phase; II degree - nystagmus appears in two eye positions - when looking in the direction of the fast phase and when looking in the center, 3 degree - nystagmus occurs when looking in all three sides).

5. The speed of movement of the eyeballs is determined by the speed (flabby and lively).

6. By duration (time during which nystagmus lasts).

Nystagmus is a cyclic eye movement that has two phases - slow and fast. Since fast eye movement is more easily visible to the observer, the side of the fast phase of eyeball movement was chosen as the direction of nystagmus. This phase is generated in the central parts of the brain and disappears if the person is put under anesthesia (the eye stops in the extreme abduction position). The slow phase of nystagmus is generated in the ampullary apparatus, therefore, in pathological conditions associated with damage to the labyrinth, the manifestations of most vestibular tests in such patients are associated with the direction of the slow phase of nystagmus.

Study of stability in the Romberg pose.

The examinee stands, toes and heels together, arms stretched out in front of him, fingers apart. The doctor stands behind and insures the examinee so that he does not fall (but does not touch him). Normally, the examined person stands confidently and almost does not sway.

If in Romberg's position the patient is not standing steady and is leaning to one side, then the patient is offered to turn his head 90° first to one side, and then to the other. When the labyrinth is affected, the direction of fall after turning the head will change in accordance with the position of

the head. When the cerebellum is damaged, the direction of fall will not change after turning the head, the patient with this pathology mainly falls towards the affected cerebellum or back.

Straight walk.

The examinee is asked to take five steps forward with closed eyes along a straight line (drawn on the floor), and without turning around and without opening his eyes, take five steps back - to the starting point (moving with his back forward).

Normally, the subject does not deviate from a straight line and almost certainly turns back. When the labyrinth is damaged, the patient deviates from a straight line in the direction of the slow phase of nystagmus, and when the cerebellum is damaged - in the direction of the lesion.

Flank march.

The examinee is asked to close his eyes and take 5 incremental steps to the right in a straight line (right side forward), and then without opening his eyes to take 5 incremental steps to the left (moving with the left side forward), trying to get to the starting point.

In a normal condition and with a lesion of the labyrinth, the examinee almost does not deviate from a straight line and turns back (flank walking is performed in both directions). When the cerebellum is damaged, the patient cannot perform a flank walk in the direction of the lesion, but the walk is performed in the healthy side.

Finger-finger test (index).

The doctor sits opposite the patient, stretches his arms forward at chest level and extends his index fingers, the remaining fingers are clenched into fists. The examinee's hands are on his knees, and his fingers are in the same position. The examinee is asked to raise his hands and simultaneously touch the doctor's fingers with the fingers of both hands. First, it should be done three times with open eyes, and then the patient is offered to close his eyes and repeat the procedure. Normally, the examinee touches the doctor's fingers with his fingers.

When the labyrinth is affected, the patient swings both hands in the direction of the slow phase of nystagmus. With a cerebellar lesion, the patient misses with one hand on the side of the lesion and in the direction of the lesion.

Finger-nose test.

The examinee is offered to move his hands to the sides and alternately touch the index finger of one hand to his own nose, and then the index finger of the other hand to his own nose. This should be done twice with open eyes, then the patient is offered to close his eyes and repeat the procedure. Normally, the examinee touches his own nose with his fingers.

When the labyrinth is affected, the patient misses with both hands in the direction of the slow phase of nystagmus (hits his own cheek). With a cerebellar lesion, the patient misses with one hand on the side of the lesion and in the direction of the lesion.

To study the function of the semicircular canals, rotary, caloric and pressure tests are performed.

Rotary test.

Necessary means:

1 Barani chair, which can rotate in the horizontal plane around its vertical axis.

The test is performed as follows: the patient is asked to sit in the Barani chair, close his eyes and tilt his head forward by 30° in order to bring the horizontal semicircular canal into the plane of rotation. To irritate the frontal channel, the patient's head is thrown back by 60°, and to irritate the sagittal channel, it is tilted to one shoulder.

After that, the chair is rotated around the vertical axis at a speed of 10 revolutions in 20 seconds. When the chair is stopped, the time is recorded with a stopwatch and the patient is asked to open his eyes and follow the doctor's finger, which is slowly moved first to the right and then to the left. At the same time, the twitching of the eyeballs, which normally perform small horizontal movements (horizontal nystagmus), is observed. Note when the nystagmus disappears. In most people with normal excitability of the vestibular analyzer, the duration of post-rotational nystagmus when the horizontal semicircular canal is irritated is on average 25-35 s, when the frontal and sagittal canals are irritated - 10-20 s.

The rotational test is based on the fact that as a result of body rotation, endolymph moves in the semicircular canals. This movement causes irritation of receptors of the ampullary apparatus, which is accompanied by the appearance of vestibular phenomena (nystagmus, nausea, dizziness, etc.).

Caloric test.

Necessary means

1. Syringe Jean
2. 100 ml of water at room temperature (20° C)
3. Renal tray
4. Water thermometer
5. Stopwatch

In this test, the right or left labyrinth is studied separately. The examinee sits on a chair with his head thrown back by 60° or lies on a couch with the head end raised so that the head is tilted to the chest by 30°. In this position, the external (horizontal) semicircular canal is in a vertical position. After that, 100 ml of cold or hot water is poured into the external auditory canal with a Jeanette syringe in 10 seconds.

Infusion of cold or hot water into the ear causes a change in the temperature of the endolymph in the external semicircular (horizontal) canal and its movement, and this, in turn, causes the emergence of a complex of vestibulo-sensory, vestibulo-somatic and vestibulo-vegetative reactions.

The infusion of cold water causes the movement of endolymph in the direction from the cooling ampoule of the external semicircular canal and the movement of endolymph in it (nausea, sinking feeling, nystagmus, etc.).

Normally, after infusing water at room temperature (20°C), nystagmus in the opposite direction is observed, which normally lasts 30-60 seconds; after infusion of warm water (44° C), nystagmus occurs in the same direction and lasts 60-90 seconds. Absence or reduction in the duration of nystagmus during a caloric test may indicate a decrease in the excitability of the vestibular apparatus (death of the labyrinth).

The caloric test is based on the fact of the movement of endolymph in the semicircular canals of the labyrinth, which occurs due to temperature changes in the outer parts of the horizontal semicircular canal under the influence of artificial heating or cooling.

In the presence of dry perforation, a caloric test should not be performed in order not to cause an exacerbation of chronic otitis media.

For visual analyzer research, visual acuity, color perception are checked and the data of the eternal bottom are analyzed (now the eternal bottom is examined by an ophthalmologist).

Visual acuity is examined using tables (Holovina, Syvtseva, Shevalova). The patient is offered to read the letters from the table in the direction from top to bottom with one eye (the other closed) from a distance of 6 meters. The line on which the examinee named all the letters is taken into account. Visual acuity (Visus) is indicated on the left of the table. Normal visual acuity is 1.0 (100%).

Fields of vision are checked using a perimeter. In bed-ridden patients and in polyclinic conditions, during an outpatient appointment, the field of vision is checked with an orienting control device. To do this, one eye of the patient is covered, and the other is offered to see in the field of vision a hammer, which is moved by the doctor from the right, left, top and bottom.

Loss of half of the field of vision is called hemianopsia.

The fundus is examined by an ophthalmologist, but every doctor must be able to correctly assess its picture. A normal picture of the eternal fundus: the papilla of the optic nerve is pink in color, its borders are clear, arteries and veins are of normal caliber, namely, arteries are twice as narrow as veins.

Smelling substances familiar to the patient, which do not cause signs of irritation, are used

for the study of smell. The patient should close his eyes and cover one nostril. An odorous substance is brought to the open nostril, the nature of which the patient must determine. Then the same manipulation is carried out with the other nostril.

Decreased sense of smell is called hyposmia, its absence is called anosmia.

Pupillary reflexes:

The afferent part of the pupillary reflex arc is part of the optic nerve, and the efferent part is part of the oculomotor nerve. When illuminated, the pupils narrow. They reveal a direct and cooperative reaction of the pupils to light.

When studying the reaction of the pupils to light, the patient's face should be superficial to the light source. The researcher tightly covers both eyes of the patient with his palms. To study the direct reaction, one palm is quickly removed from the eye - the pupil's reaction to light is recorded. The same is repeated on the other side.

To study the joint reaction, the doctor tightly covers one of the patient's eyes with his palm and watches how the pupil of the second, open eye reacts. The same thing is repeated on the other side.

The reaction of the pupil to light in one patient can be lively, in another - sluggish. Therefore, when describing the quality of the pupillary reaction (reflex), it is necessary to note the following: the reaction of the pupils to light is direct and cooperative, lively or sluggish. In addition, the pupils narrow during convergence and accommodation. The quality of this reaction is also noted - lively or sluggish.

In order to investigate the reaction of the pupils to convergence, the patient is offered to look into the distance without fixing his gaze. Note the width of the pupils. Then the patient must fix his gaze on the hammer or the researcher's fingers, which are brought closer to the patient's nose, while the pupils constrict.

Accommodation is an adaptation to clear vision at different distances. They suggest that the patient look into the distance, note the size of the pupils, and then shift his gaze to nearby objects. Pupils narrow. Since convergence and accommodation are closely related, when describing the reaction of the pupils to convergence and accommodation, its quality is noted at the same time (alive or sluggish).

During hearing tests, the patient stands at a distance of 5-6 meters from the researcher. The doctor whispers some words, and the patient must repeat them. Hearing loss - hypoacusia, absence - anacusia.

Materials for self-control of training quality

Tests

1) The peripheral department of the olfactory brain includes the following structures, with the exception of:

Answer options:
a) olfactory tract;
b) olfactory bulb;
c) olfactory triangle;
d) anterior perforated space
e) angular gyrus
Correct answer: e)

2) All of the listed nerves are sensitive except:

Answer options:
a) olfactory;
b) visual;
c) auditory;

d) vestibular;
e) diverting.
Correct answers: e)

3) Where are the I and II neurons of the visual pathway located?
Answer options:
a) retina of the eye;
b) anterior central gyrus;
c) posterior central gyrus;
d) occipital part of the cortex;
e) frontal lobe.
Correct answer: a)

4) Where are the III neurons of the visual pathway located?
Answer options:
a) retina of the eye;
b) anterior central gyrus;
c) posterior central gyrus;
d) occipital part of the cortex;
e) frontal lobe.
Correct answer: a)

5) What symptoms are associated with abductor nerve damage?
Answer options:
a) divergent strabismus;
b) coincident strabismus;
c) drooping of the upper eyelid (ptosis);
d) diplopia;
e) absence of pupillary reflex.
Correct answer: b, d)

Tasks for self-control :

1. The patient suffers from a tumor of the pituitary gland; in addition to a severe headache, he complains of impaired vision. What is the nature of the patient's visual impairment? The correct answer is bitemporal hemianopsia.

Materials for classroom self-training:

List of educational practical tasks:

1. Master research methods:
   a) visual acuity;
   b) field of vision;
   c) sense of color;
   d) pupillary reflex;
   e) picture of the fundus;
   f) sense of smell
2. Be able to make a topical diagnosis of damage to the frontal lobe and cortical ends of the olfactory analyzer.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

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- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

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- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

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1. Medical Books On-line Library (Neurology) – free download
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Practical lesson No. 7

Topic: Cranial nerves: III, IV, VI, and syndromes of their damage

Purpose: to acquaint applicants with the symptoms of damage to the oculomotor nerves in various neurological diseases, to emphasize the possible threat to the life of a patient with damage to these nerves.

Basic concepts:

Damage to the oculomotor nerves is observed in life-threatening conditions, which requires timely topical diagnosis in order to determine adequate treatment.

Equipment: classroom, furniture, equipment.

Plan:

3. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
4. Control of basic knowledge (written work, written test, frontal survey, etc.):
    - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Oculomotor nerve (lat. nervus oculomotorius) is the III pair of cranial nerves in humans and other vertebrates. The nerve is motor: it contains somatomotor fibers (to muscles) and autonomic parasympathetic (visceromotor) fibers. It innervates most of the oculomotor muscles (except the upper oblique and lateral rectus muscles of the eye), which is why it got its name, as well as the levator palpebrae muscle (lat. musculus levator palpebrae superioris), the pupil constrictor muscle (lat. musculus sphincter pupillae) and ciliary muscle (lat. musculus ciliaris). In addition, the oculomotor nerve is part of a complex system of vision stabilization (optic-kinetic nystagmus and vestibulo-ocular reflex). If it is damaged, the above functions disappear.

The pupillary reflex consists in the narrowing of the pupil when it is more illuminated and in its expansion when there is a lack of light; all this is aimed at providing greater visual acuity. The afferent link of the reflex is the optic nerve. Its fibers go to the pretectum. From this anatomical formation, the fibers go to both nuclei of Edinger-Westphal and further as part of the oculomotor nerve - to the ciliary node, where they switch to postnodal fibers that go directly to the pupillary sphincter. Since the fibers from the pretectum go to both parasympathetic nuclei of the oculomotor nerve, it does not matter whether one or both eyes are illuminated, in any case, two pupils must react (sympathetic reaction to light).

Accommodation consists in changing the curvature of the lens for better vision at near and far distances. The efferent link is the oculomotor nerve (its visceromotor component), the afferent link is the optic nerve. The path through which the signal travels from the occipital lobe (cortical visual analyzer) to the Edinger-Westphal nuclei is poorly understood. When it is necessary to see at very close distances, in addition to accommodation, convergence (bringing the eyes to the nose) occurs and the pupil narrows. The lack of reaction of the pupil to light with preservation of convergence and accommodation and the opposite situation is the basis of direct and indirect Argyle-Robertson syndromes.

The vestibulo-ocular reflex manifests itself in turning the eyes in the opposite direction to the rotation, that is, when the neck and head turn to the right, the eyes turn to the left; when the head goes down, the eyes are turned up. This ability is ensured by the complex cooperation of CNS departments, of which the main role is played by the vestibular system, the median longitudinal bundle and the III, IV and VI pairs of cranial nerves.

Changing the position of the head irritates the semicircular canals, which are connected to the VIII pair of cranial nerves. Having reached the parenchymal nuclei, several pathways lead to: a) the contralateral nucleus of the abductor nerve; b) from it through the medial longitudinal bundle to the contralateral nucleus of the oculomotor nerve, to the part that innervates the medial rectus muscle; c) there is a direct path (Deiter's path) from the parenchymal nuclei to the nucleus of the oculomotor nerve on the same side. The remaining nuclei and subnuclei are inhibited. Thus, when the head is turned to the right, the eyes will turn to the left.

Clinical examination of the condition of the oculomotor nerve is very important, as it gives an idea of the location of the pathological focus in the oculomotor nerve system. The function of the oculomotor nerve is tested according to four main criteria: movement of the eyeball, pupillary reflex, accommodation with convergence and position of the upper eyelid.

The first thing that catches your eye is the position of the upper eyelid and the location of the eyeball. If the trunk is affected on the side of the lesion, ptosis will occur (unilateral), if the central part of the oculomotor nucleus is affected, there will be bilateral ptosis, if the nucleus is affected, which does not affect this area, ptosis will be absent.

The position of the eyeball can indicate which of the nerves innervating the oculomotor muscles is affected. In the case of damage to the nerve trunk, divergent strabismus will be observed on the side of its damage. When the nucleus is damaged, on the side of its damage, it will not be possible to bring and, to a lesser extent, lower the eye, and on the opposite side, there will be an inability to raise the eye. The patient will develop diplopia.

The pupillary reflex is a reaction (narrowing of the pupil to the action of light). The reflex can be direct (when shining on the pupil being tested) and indirect (shining on the opposite pupil). In any case, the pupil on both sides must respond to the stimulus. Both in the case of damage to the nucleus and in the case of damage to the nerve on the side of the lesion, the pupil will not react to light and will be dilated.

Accommodation is tested along with convergence. When bringing the finger to the patient's nose, it is normal to observe abduction of both eyes, dilation of the pupil and (you must ask the patient) better clarity of the image of the finger in front of the nose.

Based on the existing symptoms, the approximate location of the lesion is determined; this place is confirmed by MRI, which allows you to visualize the focus, understand its nature, size, and exact localization.

Argyle-Robertson syndrome

Argyle-Robertson syndrome is understood as the following symptom complex: slightly narrowed pupils, which narrow even more during convergence, but do not narrow under illumination. It is a sign of neurosyphilis. There is also an indirect Argyle-Robertson syndrome in which the pupils will react to light but not constrict at convergence and is often seen in Economou's encephalitis. The causes of the syndrome have not been fully elucidated. There are two main opinions of scientists. Some believe that the interneuron that connects the neuron of the Edinger-Westphal nucleus with the ganglion cells of the retina is damaged (the path that is responsible for narrowing during convergence is not damaged). Another part of scientists believes that the pathological focus is localized in the ciliary node.

Paralysis of the oculomotor nerve

Paralysis of the oculomotor nerve, or neuropathy of the oculomotor nerve, is the main disease associated with the III pair of nerves. It can be caused by various factors: blood circulation disorders, congenital malformations of the nerve, infections, tumors, endocrine disorders, inflammatory processes. Congenital and acquired neuropathy are distinguished. The causes of congenital neuropathy have not been fully elucidated; any damage to the nerve during embryonic development, its hypo- or anaplasia are the causes of such neuropathy.

Paralysis of the oculomotor nerve will occur when any part of the nerve is damaged, only the manifestations will differ: starting from a barely noticeable loss of a single function, ending with a complete loss of innervation. The main cause of neuropathy is blood circulation problems (strokes), compression of the trunk, tumors, which will be manifested by a certain syndrome.

IV pair of CN - motor nerve. The cortical analyzer of the block nerve is embedded together with the cortical analyzers of other nerves involved in the innervation of the muscles that provide eye movements (back sections of the second frontal gyrus). Fibers from here go as part of the pyramidal path through the white matter of the large hemispheres, the knee of the internal capsule and end on the nuclei of the block nerves. The nuclei lie in the covers of the legs of the brain at the bottom of the sylvian aqueduct at the level of the lower tubercles of the roof of the midbrain. The intramedullary part of the roots is directed to the opposite side, exits from the substance of the brain on the dorsal surface of the brain behind the lower tubercles on two sides from the frenulum of the upper brain sail. In the region of the brain sail, the left and right block nerves make a second crossing, then bypass the pedicle of the brain from the extreme position and, passing between the temporal and parietal lobes, reach the base of the brain. Later, the block nerve passes forward and

at that time pierces the dura mater, after which it enters the outer wall of the cavernous sinus. It leaves the cranial cavity through the upper orbital fissure with the III, VI pairs of cranial nerves and the I branch of the trigeminal nerve. It innervates the upper oblique muscle, the function of which is to move the eyeball down and rotate it inwards.

Clinic for lesions of the IV pair of cranial nerves. The patient complains of double vision when looking down (for example, when going down the stairs). Some patients have a forced posture of the head: in recent cases of paresis of the block nerve, the head is tilted towards the shoulder of the opposite side. In this way, the patient compensates for doubling. When asked to look down, the eyeball on the affected side deviates downward and outward.

An isolated lesion of the IV pair is observed in diabetes mellitus, injuries, for example, a crack of the upper edge of the orbit, hemorrhages in the cerebral cortex, a tumor of the pineal gland. When the orbit is injured, especially if it is accompanied by severe bruising, hemorrhage into the conjunctiva, a crack in the upper edge of the orbit is often visible, and its possibilities are discussed only after the swelling disappears, and the double vision will continue to bother the patient, which ultimately is a reason for radiography of the orbit.

In diabetes, in addition to the IV pair of cranial nerves, other nerves that innervate the eye muscles can be involved in the process. Most often, this occurs against the background of sub or decompensation of moderate or severe forms of diabetes. However, in some patients, paralysis of the oculomotor muscles can occur in the latent stage of diabetes and serve as the first manifestations of this serious pathology. In a number of patients, muscle paresis is preceded by pain in the eye, in the forehead, eyebrow, cheek, half of the face, and pain along the course of the temporal artery. In some of them, sensitivity disorders are found on the face. Other causes of unilateral or bilateral damage to the IV pair can be nuclear aplasia, arteriovenous malformation, demyelination, subdural hematoma with compression of the brain stem, Guillain-Barre syndrome, ophthalmic herpes zoster, encephalitis, etc.

The abductor nerve has only one nucleus - the nucleus of the abductor nerve (lat. nucleus nervi abducentis). It is a motor nucleus. The nucleus is located in the pons , near its transition into the medulla oblongata, under the facial tubercle (lat. colliculus facialis) and next to the middle longitudinal bundle (lat. fasciculus longitudinalis medialis). Actually, this tubercle is formed by the fibers of the facial nerve , which bypass (this bend is called the internal knee of the facial nerve) the nucleus of the abductor nerve and separate it from the fourth ventricle of the brain. In humans, there are two types of neurons in the nucleus : motoneurons , which provide innervation of the lateral rectus muscle of the eye, and interneurons (internuclear neurons), which, as part of the median longitudinal bundle, go to the other side, to the front part of the lateral part of the nucleus of the oculomotor nerve . to ensure commonality in the movements of both eyes. In the study of animals, another population of neurons was found, which send their processes to the cerebellar flap (lat. flocculus cerebelli); the cerebellum thus provides stabilization of the gaze to the side. Analogous neurons have not yet been found in humans.

Nerve trunk. Fibers from each nucleus go forward through the substance of the bridge. Later, they leave the brain stem at the border between the pons and the pyramid of the medulla oblongata ; this area is called bulbo-bridge groove. The nerve first enters the cistern of the bridge (a component of the subarachnoid space), later pierces the dura mater and reaches the top of the temporal bone, where it passes through the Dorela canal - a structure formed by the top of the temporal bone from the side and below, the stony-sphenoid ligament above and the back of the Turkish saddle from the inside. After passing through the channel, the nerve enters the cavernous sinus (lat. sinus cavernosus). The topography of the nerve in this formation is very important:

the lateral and highest position will be occupied by the oculomotor nerve , below it will pass the block nerve (lat. nervus trochlearis), but later their mutual location will change even lower, the first and second branches of the trigeminal nerve will be placed medially from these two branches, the abductor (lat. nervus abducens) will go even more medially the internal carotid artery directs from it .

From the cavernous sinus, the nerve enters the orbit through the superior orbital fissure (lat.

fissura orbitalis superior), from the medial edge of the latter, passes through the tendinous ring (the common point of origin of all proximal ends of the oculomotor muscles) and weaves into the lateral rectus muscle of the eye . what a nerve and innervates.

Path to the central nervous system

Since the nerve is purely motor, its only path is motor and pyramidal. The path from the cortex to the nucleus of the afferent nerve belongs to the cortical-nuclear pathway (lat. corticonuclearis) and consists of two neurons:

the first neuron (upper motoneuron) lies in the precentral gyrus of the brain, the second neuron (lower motoneuron) is located in the nucleus of the afferent nerve; these neurons receive fibers both from the opposite hemisphere and from their own side.

Since the fibers from the nucleus (from the lower motoneuron) go to the lateral rectus muscle of the eye, in the literature you can find, taking into account this segment from the nucleus to the muscle, another name for the path - corticomuscular (lat. tractus corticomuscularis).

Blood supply of the nerve. The bundle and subarachnoid parts of the nerve are supplied with blood from the basin of the main artery . The key role for the abductor nerve is played by the anterior inferior cerebellar artery - the main vessel in this department. The nerve is also fed by the plexus, which is located on the slope of the occipital bone and which is formed by the division of the branches of the ascending pharyngeal artery (basin of the external carotid artery) and from the branches departing from the carotid sinus . In the area of the cavernous sinus and the upper orbital fissure, the nerve is supplied with blood from the anterolateral trunk of the internal carotid artery .

Functions. The abductor nerve is one of the oculomotor nerves. It innervates the lateral rectus muscle of the eye, which ensures removal of the eyeball. Like the rest of the oculomotor nerves, it is involved in both conscious and unconscious eye movements. In addition to direct innervation of the muscle, it also ensures coordinated eye movement when looking to the side. The latter is realized due to the presence of interneurons in the nucleus of the abductor nerve, which in turn transmit a signal to the contralateral nucleus of the oculomotor nerve, namely to the part that innervates the medial rectus muscle of the eye. These interneurons are involved in providing optokinetic and vestibulo-ocular reflexes.

Materials for self-control of training quality

Tests

1) M. ciliaris is innervated by:
Answer options:
a) abductor nerve;
b) oculomotor nerve;
c) Broca's center;
d) optic nerve
e) center of Budge
Correct answer: b)

2) All of the listed nerves are sensitive except:
Answer options:
a) olfactory;
b) visual;
c) auditory;
d) vestibular;
e) diverting.
Correct answers: e)

3) Where are the nuclei of the III pair of CN?
Answer options:
a) middle brain;

b) anterior central gyrus;
c) posterior central gyrus;
d) occipital part of the cortex;
e) bridge.
Correct answer: a)

4) Where are the nuclei of the IV pair of CN?
Answer options:
a) middle brain;
b) anterior central gyrus;
c) posterior central gyrus;
d) occipital part of the cortex;
e) bridge.
Correct answer: a)

5) What symptoms are associated with abductor nerve damage?
Answer options:
a) divergent strabismus;
b) coincident strabismus;
c) drooping of the upper eyelid (ptosis);
d) diplopia;
e) absence of pupillary reflex.
Correct answer: b, d)

Tasks for self-control :
1. The patient suffers from myasthenia gravis. What is the nature of the patient's visual impairment?
Correct answer: ptosis, diplopia.

Materials for classroom self-training:
List of educational practical tasks:
1. Master research methods:
   a) function of oculomotor nerves;
   b) pupillary reflexes;
2. Diagnose damage to the internal muscles of the eyeball;
3. Diagnose damage to the oculomotor nerves.

Instructional material for mastering professional skills
1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

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Electronic information resources

1. Medical Books On-line Library (Neurology) – free download
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Practical lesson No. 8

Topic: Cranial nerves: V, VII, and syndromes of their damage.

Purpose: to acquaint applicants with the symptoms of damage to the brain bridge in various neurological diseases, to emphasize the danger of these findings.

Basic concepts: damage to the bridging group of cranial nerves leads to violations of important functions, which requires timely topical diagnosis of the focus in the trunk with the aim of determining adequate treatment.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
    - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

The trigeminal nerve occupies a special position among other cranial nerves. It is a homologue of the spinal segment, it has sensitive and motor roots. Its peripheral branches consist of three trunks (branches).

I branch - optic nerve, II branch - maxillary nerve, III branch - mandibular nerve.

These large nerve trunks depart from the Gasserov (trigeminal) node, located inside the dura mater on the front surface of the pyramid of the temporal cyst. This formation is homologous to the spinal ganglion.

The optic nerve, the first branch of the trigeminal nerve, exits the skull cavity through the superior orbital fissure and divides into three branches:

1 Lacrimal nerve - innervates the lacrimal gland, the skin of the upper eyelid and the outer corner of the eye slit, has a connecting branch with the zygomatic nerve, through which the secretory fibers go to the lacrimal gland.

2 Frontal nerve - innervates the skin of the forehead, upper eyelid, medial corner of the eye.

3 Nasopharyngeal nerve - innervates the eyeball, nasal septum, nasal mucosa, side wall of the nasal cavity.

The maxillary nerve is the second branch of the trigeminal nerve, exits the nasal cavity through the round opening and enters the pterygopalatine fossa. In the pterygopalatine fossa, the maxillary nerve sends a branch to the dura mater, to the pterygopalatine node, from which branches including parasympathetic, sympathetic and sensitive fibers depart. Three main branches depart from it.

1 Hypoxillary nerve - a direct continuation of the maxillary nerve, gives the upper (posterior, middle, anterior) alveolar nerves. They innervate the large molars of the upper jaw, the mucous membrane of the maxillary sinus, participate in the formation of the upper dental plexus, innervate the small molars of the upper jaw, the mucous membrane of the alveolar process, the gums and sockets of the upper canines and incisors, the front parts of the nasal cavity. One of the branches (nasal) takes part in the formation of an anastomosis with the nasopalatine nerve coming from the pterygopalatine node. A branch goes from the anterior superior alveolar nerves to the mucous membrane of the anterior part of the nasal cavity.

2 The zygomatic nerve departs from the submental nerve, innervates the skin of the cheek and the front part of the temporal area.

3 The pterygopalatine nerve, the branches of which are directed to the pterygopalatine node, where the sensitive nerves of the node begin from them. Part of the fibers does not enter the node and joins its branches. The pterygopalatine nerves connect the trigeminal nerve with the pterygopalatine node.

The mandibular nerve is the third branch of the trigeminal nerve. It exits the skull cavity through the foramen ovale. It innervates the skin of the lower lip, chin, lower part of the cheek, front part of the auricle, external auditory canal, part of the outer surface of the tympanic membrane, mucous membrane of the cheek, floor of the oral cavity, front 2/3 of the tongue, teeth of the lower jaw. It provides motor innervation of the chewing muscles. Before exiting the cranial cavity, it gives off a meningeal branch to the dura mater.

Branches depart from the mandibular nerve:

1 Buccal nerve innervating the mucous membrane of the cheek.

2 Lingual nerve innervating the front 2/3 of the mucous back of the tongue. It is joined by the tympanic string, parasympathetic secretory fibers for the sublingual salivary gland, sensitive fibers from the taste buds of the anterior 2/3 of the tongue. The main branches of the lingual nerve are the branches of the pharyngeal isthmus, the hypoglossal nerve, and the lingual branches.

3 The auricular-temporal nerve innervates the skin of the middle part of the temporal area, the front part of the auricle, and the temporomandibular joint. It has branches that go to the parotid salivary gland. These are sensitive parasympathetic secretory fibers from the ear node, which is a small round body located under the oval opening. Parasympathetic secretory fibers in the small stony nerve (branches of the tympanic nerve from the glossopharyngeal nerve) fit to it. These fibers are interrupted at the node and go to the parotid gland via the auriculotemporal nerve.

4 The lower alveolar nerve is the largest branch of the mandibular nerve. Entering the mandibular canal, it gives off numerous branches, anastomosing with each other, which participate in the formation of the mandibular plexus, which forms the mandibular and gingival branches. When exiting the chin opening, they are divided into the chin nerve and the incisor branch. It innervates the lower teeth, lower gums, the walls of the sockets, the skin of the chin and lower lip.

Motor branches - masticatory nerve, deep temporal nerves (middle, front, back), lateral and medial pterygoid nerves, which tension the tympanic membrane, soft palate - go to the muscles of the same name, are axons of cells located in the brain bridge, which make up the masticatory nucleus of the trigeminal nerve.

The sensory department of the trigeminal nerve system is represented by sensitive neurons and conductors that perceive and conduct all sensory information from the skin of the face, the maxillofacial system, the mucous membrane of the paranasal sinuses, and partially from the dura mater.

The zone of sensitive innervation of the trigeminal nerve is as follows: the skin of the face, the frontal-parietal part of the scalp, the eyeball, the mucous membrane of the nasal cavities, the mouth, the front two-thirds of the tongue, the teeth, the periosteum of the facial skull, the dura mater of the anterior and middle cranial fossae. Fibers of the trigeminal nerve also approach the proprioceptors of the masticatory, eye, and facial muscles. 3 nodes of the autonomic nervous system are connected to the branches of the mandibular nerve: ear (gangl. oticum), submandibular (gangl. submandibulare), sublingual (gangl. sublinguale). Postganglionic parasympathetic fibers go from the nodes to the salivary glands. As part of the mandibular nerve, taste fibers also go to the mucous membrane of the front two thirds of the tongue.

Vegetative fibers pass as part of the optic nerve from the ciliary node located in the eye socket, as part of the maxillary nerve and its branches from the pterygopalatine node.

Thus, the system of the trigeminal nerve contains the receptor apparatus, conducting pathways, nuclei, cortical sections, as well as structural formations with which the nerve is connected in the process of functioning, both in the norm and in pathology.

Facial pain caused by damage to the trigeminal nerve system

According to the classification used by us, the most common cause of facial pain is pathology of the trigeminal nerve system. This group of prosopalgias is classified as typical neurogenic, associated with nerve lesions, which are characterized by paroxysmalness. Compression of the process of the trigeminal nerve by vessels, local inflammatory processes, as well as due to congenital narrowness of the channels of passage of the second or third branch of the trigeminal nerve indicate the tunnel process. Compression of the root or the nerve itself causes a disruption of the afferent sensory flow with subsequent formation of a paroxysmal-type algogenic system in the central structures of the affected nerve (peripheral and central factors of pathogenesis). This is determined by the manifestations of chicken zones.

Pain syndromes caused by damage to the trigeminal nerve system are diverse in clinical manifestation, have a severe course and chronic relapsing nature. As a rule, the main criteria for establishing a diagnosis are patients' complaints about the specific nature of the pain, its duration, irradiation, the possibility of remissions, and the duration of the disease. The most frequent syndromes of the trigeminal nerve system include trigeminal neuralgia (trigeminal neuralgia). In clinical neurology, this is the most persistent pain syndrome. It occurs in the population with a frequency of 4-13 cases per 100,000, moreover, 90% of cases occur in people over 40 years old, mostly women (60-70%). The first description of the pathology dates back to 1677, 1772.

Trigeminal neuralgia is a polyetiological disease based on the development of which the central or peripheral component prevails. The central component is determined by endocrine-metabolic, vascular, immunological factors that form the focus of pathological activity in the central nervous system. Changes in the functional state of sensitive nuclei of the trigeminal nerve in the facial area form trigger zones, the irritation of which provokes the occurrence of pathological

paroxysmal pain of various nature in the zone of innervation of the branches of the trigeminal nerve.

The primary chain of the disease is, as a rule, damage to the peripheral segment of the nerve - a branch or a root, most often under the influence of a compression factor, mainly in the channels of the branches, which can be destructively changed.

Prolonged compression leads to atrophy of myelin cells with subsequent thinning of the membranes around the axons, as a result of which the proximal part of the axon begins to grow and a neuroma appears. In this state, the nerve is very sensitive to direct mechanical irritation, which causes pain in the area of innervation, and also has a tendency to paroxysmal activity. As you know, unmyelinated fibers are pain conductors.

A major role in the occurrence of pain syndrome belongs to the central nervous system, and especially to the pain-conducting neurons of the trigeminal nuclei of the brain stem. The basis of paroxysmal neuralgic pain is the phenomenon of activation of the substance of the posterior horns of the spinal cord, subcortical nuclei with the development of a neuralgic attack, which stops when the neurons of the brain stem are exhausted. And it is he who explains the effectiveness of anticonvulsant drugs that affect the activity of neurons in the brain stem.

There is a division of neuralgia into two forms: idiopathic and symptomatic neuralgia. Symptomatic neuralgia is divided into central and peripheral.

When establishing the diagnosis of "trigeminal neuralgia", it is necessary to indicate the side of the lesion, the segment of the face (inner, middle, outer), the phase of the disease (aggravation, remission, including medication), the severity of the pain syndrome (moderate, medium, severe, neuralgic status), stages (early, late), state of postoperative interventions (blockade, neuroexeresis, microvascular decompression, etc.), as well as accompanying syndrome (phobic, depressive, other).

Central trigeminal neuralgia is defined as classic. This is a chronic relapsing disease, the main clinical manifestation of which is a painful short-term attack of pain. As a rule, pain occurs in the zone II and III branches of the trigeminal nerve. Neuralgia of the I branch is observed very rarely. During an exacerbation, patients complain of pain in the face of an attack-like nature, which patients describe as electric currents. The pain is very short-lived, appears suddenly and disappears just as suddenly. During the "light interval" there is no pain. Pain paroxysms are stereotyped. The intensity of the pain is very great. Patients always note that such pain cannot be endured. For the most part, the pain radiates to the posterior parts of the face, which are included in the root-segmental parts of the innervation of the trigeminal nerve. The presence of trigger areas, touching which causes pain, is characteristic.

The presence of trigger areas, touching which causes pain, is characteristic.

They are located on the skin of the face (wing of the nose, corner of the mouth or jaw) or on the mucous membrane of the oral cavity in the innervation zone of the affected branch. Characteristically, there is a very high sensitivity in these areas. Even the touch of the web causes a pain attack. If the patient is asked to show the trigger point, he will never touch it, but will stop his finger at a certain distance. The presence of trigger zones affects the psyche of the patient. The disease passes with periods of exacerbation and remission, exacerbations are more frequent in the autumn and winter periods.

Trigger factors - conditions or actions under which pain paroxysms occur. Most often, such factors are washing, eating, brushing teeth, opening the mouth, sometimes talking. Most patients, due to the fact that irritation of trigger areas occurs during eating, use the contralateral side during chewing. Therefore, over time, signs of myofascial pain syndrome may appear, which is superimposed on the neuralgic syndrome. This can make differential diagnosis difficult. In some patients, as a result of irritation of the precentral gyrus, spasms of facial facial muscles (more often - the muscle that raises the corner of the mouth) occur.

During an attack on the skin of the face, you can find a certain point that relieves pain - antalgic.

In some patients, during an attack, vegetative reactions (redness of the face, burning of the skin) are observed, which is explained by irritation of the excitation of the vegetative ganglia of the face, which are connected with each branch of the trigeminal nerve.

Absence of attacks at night is characteristic. The maximum period of pain paroxysms occurs in the morning. Trigeminal neuralgia is more common in women after the age of 45. Almost always only one side is affected (in most cases - the right). Some authors explain this by the development of age-related sclerotic processes in the anatomically narrower right suborbital or mental channels. Sometimes, patients notice signs of exacerbation in the form of fever, itching, and the appearance of red spots on the skin of the face. The development of a pain attack is preceded by paresthesias in the form of tingling, "creeping ants", dull aching pains of a constant nature. During the period of remission and exacerbation, these symptoms disappear. Remission of the disease occurs as a result of treatment, rarely - spontaneously, lasts from several months to several years. Provocative moments of exacerbation of the disease - stressful situations, viral infections, temperature factor.

In order to establish a diagnosis, a study of the sensory and motor functions of the trigeminal nerve is performed: the exit points of the branches of the trigeminal nerve are palpated (suprafoveal, infrafoveal, submental); the position of the lower jaw when opening the mouth is evaluated; the tone of the masticatory muscles and the range of movements of the lower jaw are investigated; conjunctival, corneal and mandibular reflexes are checked. Electroencephalography, X-ray of the facial skeleton with removal of the infraorbital canal, angiography, CT, MRI are used among additional research methods.

The clinical course of trigeminal neuralgia is similar to the course of some other prosopalgias. Differential diagnosis should be carried out between neuralgia of the trigeminal nerve and nasopharyngeal nerve, neuralgia of the vegetative ganglia of the face, pain dysfunction of the temporomandibular joint, dental diseases. The main symptoms indicating trigeminal neuralgia are the paroxysmal nature of the pain syndrome, the presence of trigger areas and trigger factors, the absence of nocturnal attacks, as well as objective signs of impaired sensory or motor function.

The main method of treatment is pathogenetic after examining the patient and identifying the cause of the pain.

Emergency care during a pain attack consists of local or central anesthesia. For the purpose of local anesthesia, novocaine blockades of individual branches of the trigeminal nerve are used. It is not recommended to use lidocaine for anesthesia. Local applications of anesthetic pastes (vinyl-anesthetic paste), solutions (with propolis, tea tree oil) are also recommended. For central anesthesia, anticonvulsants are used: carbamazepine (tegretol, finlepsin, carbasan, difenin, dilantin) ¼ tablet every 4 hours. If this dose does not provide an analgesic effect, it can be increased to ½ tablet every 4 hours. After the exacerbation subsides, a maintenance dose of carbamazepine (¼ tablet every 6 hours for 1 week) is recommended.

Due to the fact that swallowing is a strong trigger factor, suppositories with carbamazepine (0.1 g) are offered, which are quickly absorbed and therefore the analgesic effect occurs faster. Drugs that act on the opiate mechanism of antinociception are used: sodium oxybutyrate, phenibut ½ tablet at night; antidepressants (use a tricyclic antidepressant - amitriptyline 25 mg 3 times a day during the entire period of exacerbation); tranquilizers: relanium (diazepam, sibazone, seduxen). The use of these drugs is especially indicated in the presence of a phobic syndrome of waiting for an attack. The optimal dose is 1 tablet 2 times a day, or 2 ml of a 2% solution intravenously in an isotonic sodium chloride solution.

Apply action to trigger areas: lubricate the skin of the face with 5% anesthetic or 5% lidocaine ointment, alcohol tincture of water pepper. The nerve is decompressed in the bone canals (infraorbital, mandibular). For this purpose, dehydration is used (furosemide - 1 tablet in the morning once every three days; verospiron - 1 tablet every other day; eufilin - 5-10 ml of 2.4% solution intravenously slowly);

Neurotrophic agents (thiamine - 1 ml intramuscularly every other day No. 10; Neurovitan - 1 tablet 2 times a day for 1 month; Nootropil - 5 ml intravenously daily No. 10); To improve blood

circulation (trental - 1 tablet 3 times a day, curantyl - 1 tablet 2 times a day, nicotinic acid - 1 ml intramuscularly daily No. 10).

In case of irritation of vegetative formations of the face, use beloid, belataminal - 1 tablet 2 times a day; nicotinic acid - 1 tablet 3 times a day; spasmolitin - 0.1 g 3 times a day.

Elderly patients should be prescribed vascular therapy to improve blood circulation in cerebral vascular pools. Trental is recommended - 1 tablet 2 times a day, cavinton - 2 ml intravenous drip per 100 ml of physiological solution, nicotinic acid - starting with 0.2 ml and gradually increasing the dose to 1 ml per day, intramuscularly.

Diadynamic currents and amplipulse therapy are used as physiotherapeutic methods in the period of exacerbation. A certain effect can be achieved in some patients by using mumiyo electrophoresis on the exit points of the trigeminal nerve, phonophoresis of novocaine, novocaine or lidocaine ointment on trigger areas.

It is recommended to use a low-frequency laser with irradiation of each trigger point from 2 to 4 minutes, the total duration of the session - up to 20 minutes.

To stop pain paroxysms, acupuncture is used both as monotherapy and in combination with other treatment methods. In recent years, pharmacopuncture with novocaine in combination with plasmol, dimedrol or cerebrolysin has been successfully used.

Odontogenic neuralgia of the trigeminal nerve

Very often, the cause of pain in the face is a disease of the maxillofacial system, therefore, before establishing a diagnosis of "trigeminal neuralgia", symptomatic odontogenic pain syndrome must first be excluded. The reasons for the development of symptomatic neuralgia of the second or third branches of the trigeminal nerve can be: pulpitis, periodontitis, traumatic surgical manipulations, alveolitis, gingivitis, periodontitis, caries, irrational prosthetics, osteomyelitis of the jaws, dysfunction of the temporomandibular joint, and others, as well as a combination of these factors.

The leading clinical manifestations of odontogenic neuralgia are: pain of a constant wave-like nature, absence of trigger areas, localization of pain in the area of innervation of the main branches of the trigeminal nerve, more often night pain with no effect from anticonvulsants, presence of a positive therapeutic effect from the use of analgesics, bilateral localization of pain prevails, practically no lesion the first branch of the trigeminal nerve.

Thus, when establishing the diagnosis, it is necessary to exclude the odontogenic genesis of facial pain syndrome. Additional research methods are used.

The main differential diagnostic indicators of classical (central) neuralgia of the trigeminal nerve and peripheral (odontogenic) neuralgia

Central neuralgia is characterized by a short-term attack of pain with irradiation along the branches of the trigeminal nerve, the presence of trigger areas, and its absence at night. In treatment, effective anticonvulsant drugs are central pain relievers.

Neuralgia of peripheral genesis is characterized by a constant nature of pain, intensifies in the evening and at night. Bilateral localization of pain is possible. There are no trigger points, but pain is provoked by opening the mouth, chewing, exacerbation of an infection in the oral cavity. Anticonvulsants are not very effective. Analgesia occurs during local anesthesia, novocaine blockades, analgesics are used.

Post-herpetic lesion of the branches of the trigeminal nerve

Recently, an increase in the incidence of herpetic lesions of the nervous system has been noted. Herpes zoster virus resides in the sensitive ganglia of the nervous system, in particular, in the Gasser's ganglion in an inactive state before its activation, which may be due to unfavorable factors - a decrease in immunity, malignant neoplasms, the use of immunosuppressants, the presence of a competing infection. Acute herpetic infection is more often observed in the elderly, regardless of gender, in 45% of cases the first branch of the trigeminal nerve is affected.

The disease often begins acutely, with the development of symptoms of general intoxication (weakness, fatigue, diffuse headache, increased body temperature). This condition lasts 2-3 days and is often considered to be flu-like. Patients begin to feel a burning sensation in the area of innervation of the branches of the trigeminal nerve (more often the first). The intensity of the pain is very high, accompanied by itching and swelling of half of the face, various paresthesias on the skin of the face and mucous membrane of the oral cavity.

A few hours after the onset of the pain syndrome, a vesicular rash appears on the skin of the face and mucous membrane (corresponding to the areas of innervation of the trigeminal nerve branch), which is very itchy and throbbing pain. Initially, the vesicles are small, but have a tendency to merge into large blisters with a clear liquid. 3 sometimes there is a rash. It is very dangerous to spread the rash on the mucous membrane of the eye, which can lead to the development of bilma.

After 10 - 12 days, the vesicles begin to burst and dry with the formation of crusts, after which mostly scars remain.

During the examination of the neurological status in the acute period, late sensitivity disorders in the area of the rash (hyperesthesia, hyperpathy, hypoesthesia), as well as pain in the points of the trigeminal nerve, are revealed.

The disease can last up to 1.5 months on average, but longer in elderly patients. In most cases, the disease passes spontaneously.

Post-herpetic neuralgia is observed more often in sick women of elderly age (in 15-25% of cases) and can last long enough (up to several years). There is a direct correlation with the age of patients.

Post-herpetic neuralgia in its clinical manifestations is very similar to classic trigeminal neuralgia, but has its own characteristics, namely: a history of acute herpetic ganglioneuritis, absence of trigger areas and trigger factors, tendency to wave-like nature of the pain syndrome. The pain is neuropathic in nature. Treatment tactics for Herpes zoster have two directions: antiviral therapy and relief of neuropathic pain. Early appointment of antiviral therapy is necessary.

In the neuritic (acute) stage of the disease, antiviral drugs are prescribed: zovirax (5 - 10 mg per 1 kg of body weight intravenously by drip in 100 ml of isotonic sodium chloride solution, or 0.4 g in tablets 2 times a day for 5 - 7 days, or in the form of an ointment for external use), valtrex (1000 mg 3 times a day daily for 7 days), rimantadine (0.5 g 4 times a day for 5 - 7 days), bonafton and floreal in tablets or ointment for external use, deoxyribonuclease (0.2% solution for eye drops or inhalation for 10-12 minutes 2-3 times a day), interferon (2 drops for eye and nose drops 3-4 times a day). To stimulate the formation of endogenous interferon, curantyl (0.025 g 1 time per day), dibazol (0.05 g 2 times per day), midokalm (0.05 g 2 times per day) are used. For pain relief, dimexide with novocaine in a ratio of 1:3 is used for compresses, analgesics (2 ml of 50% analgin solution intramuscularly up to 2 times a day, baralgin). In case of pronounced pain syndrome, the introduction of lytic mixtures (2 ml of 50% analgin solution, 1 ml of 1% diphenhydramine solution, 2 ml of 0.5% novocaine solution, 1 ml of 2.5% aminazine solution) is indicated.

It is not recommended to use drugs that improve nerve conduction (vitamins, proserin) in the acute stage of the disease, as they can cause the development of post-herpetic neuralgia. It also makes no sense to use antibiotics, they suppress immunity even more and have no effect on viruses. The use of antibiotics can only be explained as prevention of secondary infections in the presence of inflammatory diseases (pneumonia, tonsillitis, etc.).

During the development of post-herpetic neuralgia, it is inappropriate to use anticonvulsants, because they are practically ineffective; quartzization, as it leads to swelling and burning of tissues, which will only worsen the condition of patients; dyes that do not have antiviral activity (methylene blue, etc.).

Compresses with dimexide, paravertebral blocks with analgesics, cyanocobalamin (1000 mg), ganglioblockers are used for pain relief. Laser therapy, diadynamic currents, and local tinctures with propolis, water pepper, and tea tree oil can be used among physiotherapeutic procedures.

In case of ineffective treatment of post-herpetic neuralgia, hormone and X-ray therapy is used. Prednisone is prescribed starting with large doses (40 - 60 mg per day according to a 5-day scheme: after every 5 days, the dose is reduced by 5 mg). It should be remembered that to prevent side effects, the entire dose is prescribed in the morning (before 11 o'clock), together with hormones, potassium preparations (panangin, asparkam) are used, as well as preparations that improve blood circulation in the gastric mucosa (xanthine nicotinate, actovegin , solcoseril) and reparatin processes (metronidazole).

In the case of recurrent herpes (more often 1 time in 3 months), a mandatory test for HIV infection is required. All patients, especially the elderly, in the presence of a herpetic infection should have an x-ray of the lungs and stomach to rule out oncology.

Neuralgia of individual branches of the trigeminal nerve

In addition to the above-mentioned neuralgia of the trigeminal nerve with a pronounced clinical syndrome, neuralgia of some of its small branches is very often observed.

Neuralgia of the nasopharyngeal nerve (Charlin's syndrome), branches of the orbital nerve. Most often, the disease occurs as a result of damage to the paranasal sinuses, dental disease, chronic infections, flu.

Neuralgia of the nasopharyngeal nerve, or Charlin's syndrome (described in 1931), is characterized by attacks of pain in the area of the eyeball, eyebrow, and half of the nose. The pain occurs in the evening and at night and is accompanied by pronounced lacrimation, hyperemia, hyperesthesia, swelling of the mucous membrane of the nasal cavity on the side of the lesion, changes in the front part of the eye in the form of keratoconjunctivitis, iridocyclitis may be detected.

Features of the course of neuralgia of the nasopharyngeal nerve are the early onset of the disease (average age of 38 years), significant duration of pain, stability of clinical manifestations, absence of trigger areas, there is almost always a clear connection with etiological factors. Bilateral damage to the branches of the nasopharyngeal nerve with a relatively favorable prognosis is more common.

During an attack, a neurotonic reaction is objectively detected on the side of the lesion, pain occurs during palpation of the inner corner of the eye.

A differential sign is the disappearance of pain after lubrication of the front part of the nasal cavity with a 5% solution of dicain or lidocaine with the addition of 5 drops of a 0.1% solution of adrenaline (or mezatone, ephedrine, naphthyzine).

Taking into account the connection of this disease with the pathology of the paranasal sinuses, during an exacerbation it is effective to prescribe antibiotics and sulfonamide, non-steroidal anti-inflammatory drugs. Vegetotropic drugs are also prescribed: beloid, belataminal. A positive therapeutic effect is observed when cyanocobalamin is administered (1000 mg intramuscularly daily for 10 days).

If there is a pronounced pain syndrome, a complex powder is prescribed (dibazole - 0.005 g, thiamine - 0.005 g, nicotinic acid - 0.03 g, ascorbic acid - 0.3 g, glutamic acid - 0.3 g, spasmolitin - 0.1 g). Take 1 powder 2 times a day or 2 powders together at night, antihistamines, tranquilizers, antipsychotics.

Among the physiotherapeutic procedures, diadynamic currents, phonophoresis of novocaine on the eyebrow area are the most effective.

Neuralgia of the ototemporal nerve, which belongs to the III branch of the trigeminal nerve, contains sensitive and secretory fibers for the ear ganglion, connected by anastomoses with the facial and other nerves (Frey's syndrome). This disease was first described by L. Frey in 1923 under the name "auriculotemporal nerve syndrome".

The disease often begins after suffering inflammatory diseases of the parotid salivary gland (parotitis, actinomycosis, etc.) or in the presence of various injuries of the parotid area (including postoperative ones).

The main manifestations of neuralgia of the ototemporal nerve are paroxysmal pain syndrome with signs of autonomic dysfunction during the consumption of solid, spicy, sour, spicy,

sweet food, and sometimes even when mentioning it, as well as in the case of emotional and physical factors (smoking, hypothermia or overheating of the body, stressful conditions, functional disorders of the nervous system).

Pain of a burning, aching, tearing nature is localized in the area of the temple, ear, temporomandibular joint, sometimes radiates to the lower jaw.

The pain attack is accompanied by pronounced vegetative effects: hyperemia of the skin around the ear and the appearance of drops of sweat in the area of innervation of the auriculotemporal nerve. An increase in salivation is also characteristic. Often there are changes in the size of the pupil on the side of the same name, the presence of stereotypical attacks with bright vegetative manifestations.

The differential diagnosis of pain syndrome from neuralgia of the mandibular nerve, pain dysfunction of the temporomandibular joint is based on the characteristic vegetative coloring of an attack in Frey syndrome.

Emergency aid during an attack is provided by the appointment of xolinolytics (atropine platyphyllin), analgesics (sedalgin, tempalgin), vegetotropic (ergotamine tartrate, belladonna alkaloids), tranquilizers (diazepam, relanium), antihistamines, neuroleptics, non-steroidal anti-inflammatory drugs. In some cases, effective introduction of aminazine with diphenhydramine.

Vitamins of group B, lidase (64 units subcutaneously 15-20 times each), aloe are used for treatment.

Among the physiotherapeutic procedures, diadynamic currents, electrophoresis of potassium iodide or lidase, ultrasound on the area of the parotid salivary gland, ultrasound on the area of the parotid gland are offered.

It is important to carry out a differential diagnosis of trigeminal neuralgia from neuropathy of the trigeminal nerve. Neuropathy of the trigeminal nerve is accompanied by morphological changes in the nerve itself (disruptions in the structure of myelin, as well as the axial cylinder, the presence of foci of hemorrhage). Characteristic changes in the sensitive area (loss symptoms).

Infections, intoxications, injuries, inflammatory, allergic and autoimmune processes lead to the development of neuropathy of the trigeminal nerve. The nature of the pain is often aching, dull, mostly constant, accompanied by changes in sensitivity in the area of innervation of the affected root, paresthesias, and in the case of neuropathy of the mandibular nerve - by a disorder of the masticatory muscles. In addition, in the case of traumatic and toxic neuropathies, symptoms related to tissue trophic disturbances, which are innervated by the affected root, are often detected. As a rule, there are no trigger zones in neuropathies. The pain is constant and long-lasting, its intensity changes periodically.

The facial nerve (n. facialis, VII pair) is a mixed nerve, as it contains motor, sensitive and vegetative fibers. The sensitive and vegetative portion of the facial nerve is sometimes separated as a separate one - the 13th cranial nerve - the intermediate nerve. Since the nerve contains three types of fibers in its composition, then, accordingly, it has three nuclei, which are located in the Varolien bridge. Both parts of the nerve — motor and sensory-parasympathetic — go to the base of the brain in the bridge-cerebral triangle. Then the nerve bundles enter the facial canal of the temporal bone through the internal auditory opening, where they are divided into facial and intermediate nerves. The intermediate nerve got its name due to the fact that it is located between the facial nerve proper and the sphincteric nerve (8 pair of cranial nerves) when entering the internal auditory opening. The intermediate nerve includes sensitive fibers that carry taste impulses from the front two thirds of the tongue, and parasympathetic fibers to the submandibular and sublingual salivary glands, to the lacrimal gland and glands of the mucous membrane of the nasal cavity and soft palate. The facial nerve exits the skull through the stylomastoid foramen, forms a plexus in the parotid salivary gland and divides into separate branches that innervate the muscles of the face (nasal, zygomatic, circular muscles of the eye and mouth, etc.), as well as some neck muscles. The structure of the facial nerve In clinical practice, determining the nature of facial muscle paresis is of great importance. That is, the doctor must always clearly establish whether

the patient has central or peripheral paresis (paralysis) of the facial nerve. Establishing a topical and, ultimately, a clinical diagnosis depends on solving this issue. Therefore, we will focus on the movement of the motor fibers of the facial nerve in more detail. The motor nucleus of the facial nerve is located in the ventrolateral part of the Varolius pons, on the border with the medulla oblongata. The axons of the cells that form the motor nucleus of the facial nerve form the root of the facial nerve, which innervates facial muscles on its side. At the same time, the upper facial muscles are innervated from the upper parts of the nucleus, and the lower facial muscles (below the corner of the mouth) are innervated from the lower parts of the nucleus. Cortical-nuclear fibers approach the upper part of the nucleus both from its own side and from the opposite side due to the presence of a nuclear junction above it. And to the lower part of the nucleus of the facial nerve cortical-nuclear fibers come exclusively from the opposite hemisphere. This feature of the course of cortical-nuclear fibers is of great importance in topical diagnostics. Thus, if the cortical-nuclear fibers above the nucleus of the facial nerve on one side are affected, the function of only the lower facial muscles on the opposite side will be disabled. Clinically, this will be manifested by the smoothing of the nasolabial fold and the asymmetry of the mouth when showing the teeth - the mouth will be drawn in the healthy direction. At the same time, when the nucleus of the facial nerve or its root is damaged, the function of both the upper and lower mimic muscles on its side will be disabled. That is, central paralysis of the facial nerve will be characterized by paralysis of the lower facial muscles on the opposite side to the lesion. And peripheral paralysis of the facial nerve will be characterized by a violation of the activity of facial muscles on the entire half of the face on the side of the lesion.

According to the frequency of damage among cranial nerves, neuropathy of the facial nerve accounts for up to 40% of all cases of neuropathies and neuralgias, and ranks second among diseases of the peripheral nervous system. It is observed in 25 people per 100,000 population, it is an actual problem of neurology, physiotherapy, as well as an important social problem.

The disease was first described in the literature in 1836 p. (C. Bell) and that is why it is sometimes called "Bell's palsy" in the literature.

According to the etiological principle, neuropathies of the facial nerve are divided into idiopathic, which develop after hypothermia and have a seasonal nature; infectious-allergic, developing against the background of infectious diseases (tonsillitis, parotitis, pneumonia); ischemic, which develop as a result of impaired blood circulation in the nerve trunk (in case of collagenosis, diabetic angiopathy, arterial hypertension, vasculitis); otogenic (mesotympanitis, otitis, mastoiditis); traumatic (in the case of fractures of the pyramid of the temporal bone, nerve injuries during surgical interventions on the face, during anesthesia); hereditary (autosomal dominant type of inheritance with low gene penetrance).

In the pathogenesis of the disease, under the influence of one of the etiological factors, there is a violation of blood circulation in the vessels of the nerve. As a result, there is hypoxia of the membranes and nerve trunk with the development of swelling and pinching of the nerve in the narrow facial canal. Normally, the nerve occupies only 75% of the volume of the canal, but some people have a congenital narrowness of the canal of the facial nerve. All these factors lead to nerve dysfunction with the development of prosoparesis. Due to the fact that the facial nerve passes through the canal together with the median nerve, the clinical picture shows signs of damage to the median nerve. Damage to the facial nerve is more often unilateral, bilateral damage is no more than 2% of cases. Young and middle-aged women get sick more often.

The clinical picture of neuropathy of the facial nerve depends significantly on the level of damage and is characterized by paralysis of facial muscles, sensory and autonomic disorders. The main clinical manifestation is weakness of facial muscles (prosoparasis, prosoplegia).

Central paresis of the facial nerve develops depending on anatomical features. The cortical-nuclear pathway of the facial nerve makes a partial crossing to the upper half (or upper separate nucleus), and to the lower half of the nucleus (or lower separate nucleus) - a complete crossing, so in case of a unilateral lesion of the cortico-nuclear pathway, there is a violation of the innervation of facial muscles only those that are innervated by the lower nucleus of the facial nerve on the side

opposite to the lesion (mimic muscles located below the corner of the eye). Only the lower mimic muscles of the face are affected.

With a nuclear lesion, patients have signs of alternating syndromes (paresis of the facial nerve on the side of the lesion and hemiparesis on the opposite side), which is associated with involvement in the pathological process of the pyramidal pathway located next to the nucleus. In the case of this level of damage, twitching of mimic muscles on the side of the damage is sometimes observed, which is associated with irritation of the bodies of still living neurons.

When the root of the facial nerve is damaged in the area where it exits the brainstem, the patients show signs of damage to the vestibulocochlear nerve (decreased hearing, dizziness) along with facial muscle paresis.

In case of damage to the facial nerve in the channel before the departure of n. retrosus major, together with prosoparesis, there are signs of damage to the intermediate nerve (xerophthalmia, hyperacusis, xerostomia, impaired taste perception).

After leaving n. retrosus major clinical picture has the same symptoms, except for xerophthalmia, instead of it due to irritation of n. lacrimalis, lacrimation occurs.

After the removal of the stapedius, the patients do not show signs of xerophthalmia and hyperacusis, but along with prosoparesis, there is xerostomia, lacrimation, and impaired taste perception.

When the nerve is damaged in the case of exit from the canal, only prosoparesis is observed in patients, more often together with lacrimation.

Most neuropathies of the facial nerve have a favorable prognosis (complete recovery of the functions of the facial nerve when using traditional methods of treatment and are observed in 40-60% of patients; in the remaining patients, the recovery is incomplete). In addition, sometimes there is an increase in the tone of facial muscles with the development of contractures and synkinesis. It should be remembered that recurrent neuropathy of the facial nerve can be a clinical sign of Rossolimo-Melkersson-Rosenthal syndrome and, in this connection, has specific features in treatment. An electromyographic study is of great help in predicting the recovery of nerve function.

When examining the function of the facial nerve, pay attention to the symmetry of the eyelids, the position of the eyebrows, the uniformity of the frontal and nasolabial folds, the placement of the corners of the mouth, the presence of fibrillar and fascicular twitching of the facial muscles, the secretion of tears and saliva. In order to study the function of facial muscles, the patient is offered to: wrinkle the forehead, furrow the eyebrows, close the eyes tightly, bar the teeth, blow shocks, pull the lips into a tube, whistle, blow out a "candle". They also examine the sense of taste for sweet and sour (on each side of the tongue separately).

Due to the fact that in the pathogenesis of neuropathy of the facial nerve, the main place belongs to the development of vascular disorders and swelling of the nerve trunk, the main thing in the acute stage of the disease is the improvement of microcirculation and pathogenetic treatment. 3 for this purpose, anti-inflammatory and antibacterial drugs are prescribed (nonsteroidal anti-inflammatory drugs in medium doses, urosulfan 5 ml intravenously daily, a course of 7 - 10 times), vasoactive drugs: nicotinic acid - 1 ml daily intramuscularly, trental - 5 ml intravenous drip , dextran (reopoliglyukin) - 200 ml intravenous drip), diuretics (furosemide - 40 mg intramuscularly for 3 days with a 4-day break, euphylin 2.4% solution 5 ml intravenously daily).

Glucocorticoid therapy can be used in case of significant impairment of nerve function. Prednisolone is prescribed according to a 5-day scheme, starting with 30 - 60 mg per day (the dose is reduced by 5 mg every 5 days). The entire dose should be taken in the morning, mandatory intake of potassium preparations (panangin, asparkam). All patients are prescribed vitamin therapy.

In the acute period of the disease (the first 12 - 14 days), you cannot use drugs that improve nerve conduction (anticholinesterase drugs - proserin, galantamine). This is due to the fact that, if swelling and compression of the nerve are not reduced, the use of drugs that improve the passage of nerve impulses can lead to the development of contractures and synkinesis.

Much attention should be paid to the protection of mucous membranes (instill the eye with sulphacilnatium in the case of lagophthalmos, rinse the oral cavity in the case of xerostomia).

After 2 weeks, you can prescribe physioprocedures (ozokerite, mud applications on the healthy and paretic side, acupuncture reflexology, myotonic procedures).

If necessary, you can prescribe agents that affect tissue metabolism (Nerobol 2 ml once a week). Anticholinesterase drugs are prescribed to improve nerve conduction (proserin - 1 ml intramuscularly daily, galantamine, dibazole in powder - 0.02 g 2 times a day). During the recovery period, patients should do myogymnastics aimed at performing facial movements, gymnastics is performed in front of a mirror, if necessary, with the help of hands.

Syndrome of damage to the knee joint (Gant syndrome)

This disease was first described by Gantt (1909), who identified 4 variants of the course of the disease:

- herpetic rash without neurological manifestations;
- herpetic rash together with prosoparesis;
- a combination of herpetic rash, prosoparesis and hearing impairment;
- yci the above signs together with vestibular disorders.

The disease is caused by Herpes zoster virus, which enters the sensitive nodes of the cranial nerves and is activated under various conditions accompanied by a decrease in the activity of the body's immune system.

The clinical picture is characterized by a pain syndrome of varying severity, which is localized in the area of the external auditory meatus, sometimes in the soft palate.

A characteristic sign of the disease is the presence of a rash located in the innervation area of the intermediate nerve (tympanic cavity, tympanic membrane, external auditory canal, soft palate, and sometimes tonsil). Due to the fact that fibers of the facial nerve pass near the knee joint, sometimes the process spreads to them, and signs of prosoparesis appear in the clinical picture.

During an objective examination, patients are found to have impaired sensitivity (hyperesthesia, and over time - hypoesthesia) in the zone of innervation of the node, as well as impaired taste perception on the front 2/3 of the corresponding half of the tongue.

In some cases, vestibular disorders (nystagmus, dizziness) or hearing disorders (hyperacusis, ringing in the ears) are detected. On the side of prosoparesis, vegetative manifestations are observed in the form of injection of scleral vessels, lacrimation, swelling of the mucous membrane of the cheek. In some patients, the listed clinical signs of the disease are absent, and only attacks of pain in the external auditory canal and herpetic rash are observed.

The disease lasts several weeks. The prognosis is favorable in most cases.

In the treatment, antiviral drugs are used: zovirax intravenously and for local use in the form of ointment, drugs that increase the formation of endogenous interferon: curantyl - 0.025 g 2 times a day, dibazol (powder) - 0.02 g 2 times a day.

Analgesics, cyanocobalamin 1000 μg intramuscularly daily, novocaine intradermally before the external auditory canal or electrophoresis are prescribed for pain relief.

Neuralgia of the vedic nerve (Fayle's syndrome)

The vedic nerve is the junction of the large superficial and deep petrosal nerves.

The first is a branch of the facial nerve, and the second is a branch of the sympathetic plexus of the internal carotid artery.

Inflammatory processes of the sacral sinuses and pyramids of the temporal bone, injuries of the temporal bone, and metabolic disorders lead to the development of the disease.

Patients complain of attacks of pain in the area of the eye socket and nose. The pain radiates to the teeth, face, ear, and neck. Due to the fact that the nerve is connected to the pterygoid node and the knee node, the pain can spread to the corresponding half of the face and the occipital region. The pain occurs at night and lasts from several minutes to several hours.

Painkillers (analgesics) are prescribed. Antibiotics and anti-inflammatory agents are prescribed depending on the etiological factor. Taking vitamins of group B (thiamine, cyanocobalamin) is indicated.

Alternating syndromes:

This is a unilateral lesion of the brainstem, which consists in a violation of the function of one or more CNS on the side of the cell according to the peripheral type and central paralysis or paresis of the limbs or conduction disorders of sensitivity on the side opposite to the cell.

Depending on the level of damage to the brain stem, peduncular, pontine and bulbar syndromes are distinguished. They are named after the scientists who first described them.

Pontine alternating syndromes:

(occur when the bridge is damaged).

o	*The name of the syndrome*	*Damage to the nerve on the side of the cell*	*Symptoms on the opposite side*
.	Miyar Glubler	7 ChN	Central hemiplegia
.	Brisault-Sécar	Spasm of the facial muscles (irritation of the cells of the nucleus of the 7th ChN).	Central hemiplegia
.	Foville	7 + 6 CH	Central hemiplegia (+ sometimes hemianaesthesia due to damage to the medial loop).
.	Raymond Sestan	6 CN + ataxia + choreatetoid movements on the side of the cell or opposite (depending on whether cortico-pontine or ponto-cerebral fibers are affected).	Hemiparesis Hemianaesthesia.

Certain alternating syndromes can be observed in tumors, aneurysms, skull injuries, inflammations and parasitic diseases of the brain, but most often - in the case of impaired cerebral blood circulation (ischemic softening centers in the trunk).

Materials for self-control of training quality

Tests

1) The following nuclei of the trigeminal nerve are located in the bridge:

Answer options:

a) motor nucleus;
b) double core;
c) nucleus of the spinal cord;
d) lower salivary nucleus
e) all of the above
Correct answer: a)
2) What nuclei of the trigeminal nerve and facial nerve are located in the bridge?
Answer options:
a) core of the lower olive;
b) nuclei of the reticular formation;
c) motor nucleus;
d) motor nucleus;
Correct answers: d)

Tasks for self-control :
1. Where is the focus of the lesion in central paralysis of the facial nerve and what symptoms are characteristic of it?
The correct answer: cortico-nuclear pathways, only the lower half of the facial muscles is affected.
2. When are alternating paralysis formed, and what are the main symptoms characteristic of them?
The correct answer: with focal processes in the brain stem. The main symptoms: on the side of the focus - damage to the CN (depending on the level of damage), on the opposite side - conduction disorders.
3. Patient V., 50 years old. He has been suffering from diabetes for 8 years. During the last 3 months, he began to feel mild pain, numbness in his legs, "crawling ants". He did not turn to the doctor. Gradually, similar sensations arose in the upper limbs. Objectively: cranial nerve pathology was not detected. The range of motion is not limited. Tendon and periosteal reflexes on the hands are reduced. Knee reflexes are too low, Achilles reflexes are not triggered. Superficial and deep sensitivity on the hands from the level of the elbow joints, on the legs - from the upper third of the lower leg to the bottom.

Question:
1) What is the patient's syndrome and where is the lesion?
Right answer:
1) Alternating Miyar-Gubler syndrome, focus in the bridge.

4. Patient T., 64 years old, developed a disorder of pain sensitivity in the perioral area on the left.

Question:
1) What nucleus is affected?
2) What parts of the nucleus are affected?
Right answer:
1) Core of the spinal cord;
2) Upper

Materials for classroom self-training:
List of educational practical tasks:
1. Master research methods:
a) sensitivity on the face;
b) reflexes from the cornea and conjunctiva;
c) functions of facial muscles;
d) functions of additional 3 branches of the facial nerve.

Instructional material for mastering professional skills
1. Investigate the neurological status of the patient and correctly interpret the obtained data.

2. Determine the level of nerve damage.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 9

Topic: Cranial nerves: IX, X, XI, XII and syndromes of their damage. Bulbar, pseudobulbar and alternating paralysis

Purpose: to acquaint applicants with the symptoms of lesions of the medulla oblongata in various neurological diseases, to emphasize the threat to life of these complications.

Basic concepts:

Damage to the caudal group of cranial nerves leads to gross violations of important functions, which requires timely topical diagnosis of the focus in the trunk in order to determine adequate treatment.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Glossopharyngeal nerve (n. glossopharyngeus) is a mixed, but mainly sensitive nerve. The nervous system includes central and peripheral motoneurons for the pharyngeal muscles, receptors and conducting pathways, subcortical and cortical structures, fibers and autonomic nodes involved in ensuring taste sensitivity and salivation.

Its motor branch is small, it innervates only one cervical-pharyngeal muscle that raises the pharynx. Fibers of general and taste sensitivity are dendrites of cells of the upper and lower jugular ganglia, branching in the back third of the tongue, soft palate, pharynx, pharynx, front surface of the epiglottis, auditory tube and tympanic cavity.

Fibers from the lower node go to the taste buds of the back third of the tongue, and the axons of these cells penetrate the medulla oblongata and end in the taste nucleus. Fibers from the upper node of the glossopharyngeal nerve carry conductors of general sensitivity, in the medulla oblongata they approach the sensitive nucleus.

The vagus nerve (n. vagus) is a multifunctional nerve, similar in structure and function to the glossopharyngeal nerve, innervates the striated muscles of the digestive and respiratory system, parasympathetic innervation of most internal organs. Nerve damage is rarely isolated. However, there are separate nosological forms and syndromes. Symptoms of damage to the vagus nerve have to be observed by different specialists. Disorders of the innervation of the soft palate, pharynx, and larynx are of particular importance. Motor fibers for the transverse - striated muscles begin from the cells of the double nucleus (nucl. ambiquus) - the common somatic nucleus of the X and IX pairs. The axons of these cells innervate the muscles of the soft palate, pharynx, larynx, epiglottis, upper part of the esophagus, vocal cords (reverse nerve). From nucl cells. dorsalis n. vagi (parasympathetic nucleus) initiate motor fibers that innervate the smooth muscles of internal organs - bronchi, esophagus, gastrointestinal tract, vessels. Secretory fibers are directed to the stomach, pancreas, heart (inhibitory), to vessels (vasomotor). As part of the vagus nerve, there are taste fibers from the epiglottis and the back wall of the pharynx. They end in the core of a single path.

Sensitive pathways begin from the cells of the upper and lower nerve nodes (gangl. superior, gangl. inferior). The dendrites of the ganglion cells are directed to the occipital parts of the dura mater, the external auditory canal, the back surface of the auricle, the soft palate, the pharynx, and the larynx. Some of them go to the respiratory tract, gastrointestinal tract and other internal organs.

Thus, IX and X nerves have a lot in common in structure and function, they are studied simultaneously.

The hypoglossal nerve, XII pair (n. hypoglossus), is motor. It starts from the cells of the motor nucleus, located in the area of the rhomboid fossa triangle of the same name, descends through the entire medulla oblongata, reaching the I-II cervical segments of the spinal cord. The axons of the nerve cells of the nucleus form thin nerve roots, penetrate between the pyramids and olives of the medulla oblongata, form a common trunk that exits the skull through the hypoglossal canal of the occipital bone, and innervates the muscles of the tongue.

Syndromes of damage to the glossopharyngeal and vagus nerves:

The systems of the glossopharyngeal and vagus nerves are closely connected, so isolated damage to these nerves is practically not observed. In most cases, it is impossible to determine which nerve is affected more. However, there are several nosological forms with predominant damage to the glossopharyngeal or vagus nerves.

Methods of research of the IX-XII cranial nerves:

Studies of the function of the IX pair – the glossopharyngeal nerve and the X pair – the vagus nerve are carried out simultaneously due to the common innervation zone.

The patient is asked to open his mouth, while the median position of the tongue and the even position of the palatal folds are noted. Then they offer the patient to say the sound "a" and note the evenness of the contraction of the palatal folds and tongue. With a spatula, they touch the palatal folds on the right and left, note their contraction - a reflex from the soft palate. Then they touch the back wall of the pharynx on the right and left with a spatula. There is a contraction of pharyngeal constrictors (like movements during vomiting) - pharyngeal reflexes.

They give an assessment of the sonority of the patient's speech, which depends on the uniform tension of the vocal cords during phonation. Violation of phonation is called dysphonia, lack of phonation (whispered speech) is called aphonia.

The condition of the vocal cords during a visual examination can be described by an otolaryngologist, to whom such a patient should be consulted.

Then the patient is asked about the function of swallowing or is offered to swallow water. When swallowing is impaired, patients choke on food, a cough appears at the time of swallowing, and sometimes liquid food is poured into the nose. Disturbance of swallowing - dysphagia, its absence - aphagia.

To study the function of the accessory nerve (XI pair), first visually determine the correctness of the head position. With a unilateral lesion, a torticollis (torticollis) may be observed. Then the patient is offered to raise his shoulders (as if surprised), turn his head to the right, to the left. Visually determine the presence of atrophy of the trapezius and sternoclavicular-mastoid muscles, drooping of the upper arms. Muscle strength in these muscles decreases.

The twelfth pair is the hypoglossal nerve, which innervates the muscles of the tongue, ensures its movement and speech. The patient is offered to stick out his tongue and if it deviates to the side, they talk about the weakness of his muscles. Then they check the mobility of the tongue, its trophism.

In the presence of atrophy, the surface of the tongue becomes bumpy, it shrivels, fibrillar twitching of the muscles may appear. Bilateral paresis of the tongue leads to impaired speech, inability to pronounce words correctly - dysarthria.

If there is no speech at all - anarthria.

It must be remembered that when the central neurons (cortical-bulbar pathways) are damaged, there is a central paresis of the muscles that are innervated by the bulbar nerves, but when the central neurons are unilaterally damaged, there is no gross functional impairment, because the muscles of the throat, pharynx , soft palate, tongue have bilateral supranuclear connections with the motor zone of the cerebral cortex.

Neuralgia of the glossopharyngeal nerve

This disease was first described by Weilenberg in 1910, and in more detail in 1920 by Sicard and Robineau, so it is sometimes referred to as "Sicard-Robineau syndrome" in the literature.

The disease occurs in the case of traumatization of the tonsil with an enlarged styloid process,

as well as in the presence of calcification of the stylohyoid ligament, in the presence of tumors of the bridge-cerebral angle and larynx, aneurysm of the internal carotid artery, etc. In the development of the disease, metabolic disorders, atherosclerotic processes, long-term intoxications, and chronic purulent processes in the tonsils are important.

The disease takes the form of painful paroxysms lasting up to several minutes. The pain begins at the root of the tongue and tonsils and spreads to the throat, ear, sometimes to the corner of the lower jaw, eye, neck. An attack is provoked by talking, eating hot or cold food, irritation of the root of the tongue and tonsils (trigger areas). The pain is always unilateral. During the attack, the patient complains of dryness in sweat and throat, and after the attack, hypersalivation appears on the side of the pain paroxysm. Sometimes at the height of the pain, patients lose consciousness, which is explained by irritation of the IX branch of the n pair. depressor, as a result of which suppression of the vascular and respiratory centers occurs. The stage of exacerbation gradually turns into the stage of remission, which can last up to 1 year. But with the development of the disease, the attacks become more frequent and the intensity of the pain syndrome increases. In the future, the pain may be constant. Some patients have symptoms of loss of functions of the glossopharyngeal nerve (hypesthesia, decreased taste perception near the root of the tongue, hyposalivation). In this case, we are talking about the neuropathic stage of the disease. In the case of neuropathy, the pain is constant and has a wave-like course.

When determining the diagnosis, pay attention to: the patient's voice - sonority, hoarseness, nasal tone; swallowing food - dysphagia; lesions of the soft palate at rest and during phonation; taste perception on the back 1/3 of the tongue; reflexes from the soft palate and the back wall of the pharynx. During the examination of the neurological status of such patients, practically no deviations from the norm are observed. Trigger points located on the mucous membrane of the root of the tongue, tonsils, and on the back wall of the pharynx are revealed. During the sensitivity test, hyperesthesia is detected in the area of innervation of the glossopharyngeal nerve (posterior 1/3 of the tongue, pharynx, soft palate). In the period between attacks, there is a decrease in the pharyngeal reflex, a decrease in the mobility of the soft palate. During the study of taste perception, an increased taste sensitivity to bitter is observed or even taste irritations are perceived as bitter. Neuralgia of the glossopharyngeal nerve must be differentiated from neuralgia of the trigeminal nerve. These two diseases are united by the paroxysmal nature of the pain and the presence of trigger areas, but in the case of neuralgia of the glossopharyngeal nerve, pain and trigger areas are localized not on the skin of the face, but in the area of the root of the tongue, tonsils.

A differential sign of damage to the glossopharyngeal nerve is a decrease in pain paroxysm when the root of the tongue and the back wall of the pharynx are lubricated with an anesthetic solution (2% novocaine solution, 5% cocaine solution). For pain relief, carbamazepine or its analogues are used according to the schemes described in the section devoted to the treatment of trigeminal neuralgia. In the case of pronounced pain syndromes, neuroleptics, antidepressants, tranquilizers are used, and a 2% novocaine solution is injected into the root of the tongue. Diadynamic currents according to Yerokhina (on the area of innervation of the glossopharyngeal nerve and the projection of the upper cervical sympathetic node), galvanic currents have an analgesic effect from physiotherapeutic procedures. Physiotherapy procedures are carried out together with taking vitamins, immunomodulatory therapy. When conservative methods of treatment are ineffective, surgical operations are performed on the root of the glossopharyngeal nerve.

Neuralgia of the tympanic nerve (Reichert's syndrome)

The tympanic nerve is a branch of the glossopharyngeal nerve, but the clinical picture of its damage is very similar to ganglionitis of the knee joint. The etiology and pathogenesis of the disease are still not fully understood, and there is a different opinion about the role of infection and the vascular factor.

The clinical picture is characterized by attacks of sharp, shooting pain in the area of the external auditory meatus. Attacks occur up to 5-6 times a day. The disease lasts for several months, after which there is a period of remission.

Non-narcotic analgesics, neuroleptics, tranquilizers, and antihistamines are used in the treatment for pain relief. Vitamins of group B are used.

Among the physiotherapeutic procedures, the appointment of novocaine electrophoresis to the area of the auditory canal is indicated.

Neuralgia of the ear nerve

Due to the fact that the ear nerve has anastomoses with the glossopharyngeal and facial nerves, its isolated lesion is observed very rarely. The disease occurs in conditions of angina, influenza, syphilis.

The clinical picture is characterized by attacks of pain localized in the area of the external auditory meatus and eardrum. Bel is accompanied by nausea, sometimes vomiting.

Antibiotics, anti-inflammatory drugs, group vitamins are used for therapeutic purposes.

Neuralgia of the superior laryngeal nerve

The superior laryngeal nerve is a branch of the vagus nerve, which has motor and sensory fibers and innervates the muscles and mucous membrane of the larynx. The disease is very rare.

The disease occurs against the background of chronic pharyngitis, as well as after tonsillectomy and strumectomy. In case of neuralgia of the upper laryngeal nerve, paroxysms of pain occur in the larynx area. The pain is one-sided, often occurs during eating or swallowing, sometimes radiates to the lower jaw or ear. On the lateral surface of the neck, above the thyroid cartilage, there is a painful area. Cough and general weakness occur during the attack. During laryngoscopy, immobility of the corresponding half of the larynx, narrowing of the glottis is revealed. During the neurological examination, the pharyngeal reflex is practically absent. In the treatment for pain relief, analgesics are used in combination with antihistamines, anesthetics (0.5% solution of novocaine intravenously), cyanocobalamin - 1000 μg intramuscularly. In addition, thiamine is used, and the appointment of diadynamic currents is indicated from physiotherapeutic procedures.

Damage to the hypoglossal nerve system

The pathology is manifested by paresis or paralysis of the tongue muscles, convulsions and hyperkinesis of the tongue. Damage to the peripheral neuron gives a picture of peripheral paresis or paralysis of the muscles of half of the tongue. Its surface becomes uneven, wrinkled, fibrillar twitches are noted. With a central lesion due to damage to the cortical-nuclear pathways, hemiglossoparesis (hemiglossoplegia) develops without atrophy and fibrillary twitches. Deviation of the tongue when extended to the side opposite to the lesion in the brain. Convulsions of the tongue muscles (glossospasm) are tonic, clonic or mixed. Both halves of the tongue are drawn into the spasm. The course of various forms of lesions depends on the nature of the process, etiological factors. With a unilateral lesion of the hypoglossal nerve, its function is slightly impaired. Bilateral damage more often occurs with symptoms of damage to the IX and X pairs of cranial nerves. Their peripheral damage gives a picture of bulbar paralysis, and central - pseudobulbar paralysis. Bulbar syndrome is characterized by dysphonia, dysarthria, and dysphagia. During the examination, the immobility of the palatal arches, fibrillar twitching of the tongue muscles, their atrophy, and the absence of the pharyngeal reflex are revealed. In pseudobulbar syndrome, in addition to dysphagia, dysarthria, dysphonia, the symptoms of oral automatism are pronounced - violent laughter and crying.

Neuropathy of the hypoglossal nerve

The causes of neuropathy of the hypoglossal nerve can be infectious diseases (angina, meningitis, encephalitis, inflammatory processes of the maxillofacial system), injuries (operative interventions in the oral cavity, calcifying sialoadenitis of the submandibular salivary gland), intoxication, tumors of the tissues of the oral cavity. An isolated lesion of the hypoglossal nerve is rare.

There are motor disorders of the tongue muscles (paralysis or paresis), which during an objective examination is manifested by a deviation of the tongue towards the affected muscle, atrophy of the tongue (the tongue has a folded appearance), fibrillary twitches (in case of nuclear nerve damage) on the side damage As a result of impaired coordination of tongue movements

during conversation, patients have difficulties in pronouncing those sound combinations that require rapid tongue movements. This speech disorder is called dysarthria. In addition, patients have difficulties during chewing, swallowing food and forming a food lump.

In the case of bilateral damage, paralysis of the tongue muscles (glossoplegia) occurs, which leads to sharp disturbances in the act of chewing and swallowing, dysarthria, and the inability to protrude the tongue from the oral cavity.

In this pathology, the main disease is treated.

Glossodynia

The nature of the disease is not established. Most often, the occurrence of orofacial pain syndrome is associated with the pathology of the alimentary canal. In addition, the causes of glossodynia are disturbances in the emotional state of patients due to various stressful situations, pathology of the endocrine system (menopausal disorders, diabetes), vascular diseases. Under the influence of these factors, a pathological afferent flow of impulses to the cerebral cortex (mainly to the postcentral gyrus) is formed. Given that the face innervation zone occupies a very large area in the postcentral gyrus and has close connections with other parts of the brain, a focus of pathological excitement is formed in the cortex, which leads to a decrease in the threshold of pain perception in the area of innervation of the trigeminal nerve, and since the maximum flow of impulses goes from the upper third of the nucleus, then it is perceived by patients as pain in the orofacial region.

Women of elderly or climacteric age, as well as persons with pronounced social problems, are more often affected.

The characteristic clinical features of glossodynia are: the appearance of a paresthetic syndrome on the tongue and mucous membrane of the oral cavity - mucous form, and sometimes on the skin of the face - dermatomucosal form. More than 40 types of paresthesias have been described, but mostly patients complain of constant burning, tingling, and an unpleasant taste in sweat. Patients are excited and describe their complaints very emotionally. A pathognomonic symptom of glossodynia is the cessation of paresthesias during eating, so patients constantly eat something or chew gum; no visible changes are observed on the mucous membrane of the oral cavity. Most patients have signs of impaired taste perception and salivation (both hypo- and hypersalivation can be observed). The aggravation of the disease coincides with the aggravation of visceral pathology, stressful conditions, and a decrease in the body's reactivity. During an objective examination, no visible changes are observed on the mucous membrane of the oral cavity. During the study of the neurostomatological status, mildly expressed signs of bulbar syndrome are revealed (decreased pharyngeal and soft palate reflexes, limited tongue protrusion, possible dysarthria).

During the study of the sensory function in the area of innervation of the branches of the trigeminal nerve, sensitivity disorders are not observed, but only hyperesthesia is detected in the internal area of Zelder, which corresponds to the innervation of the upper third of the nucleus of the spinal pathway of the trigeminal nerve.

To establish the diagnosis of "glossodynia", it is necessary to exclude symptomatic glossitis, which are characterized by changes in the mucous membrane of the oral cavity (plaque, edema, desquamation, ulcers).

The symptoms of glossodynia are very similar to the symptoms of neuritis or neuralgia of the lingual nerve, which is mostly manifested by pain in half of the tongue, which increases during chewing, without changes in taste perception and salivation. In addition, this disease is characterized by sensitivity disorders in the form of hypoesthesia on the tongue.

In the event of paresthesias in patients with metal prosthetic teeth, it is necessary to exclude the presence of galvanism associated with the occurrence of currents between different metals (it should be remembered that such processes occur more often in soldered structures or in the presence of prostheses made of different metals).

Sometimes the causes of paresthetic syndrome can be injuries to the mucous membrane of the tongue by the sharp edges of teeth and fillings, but in this case there are no characteristic signs

of glossodynia (ceasing of paresthesias during eating, as well as bulbar syndrome).

Sometimes an unpleasant taste in the mouth can be one of the first symptoms of diabetes, so all patients need to determine the level of glucose in the blood.

A comprehensive approach should be used in the treatment of glossodynia. Together with the treatment of a somatic disease (gastritis, duodenal ulcer, cholecystitis, pancreatitis, pathological menopause, etc.), tranquilizers are used (tazepam 30-90 mg per day, phenazepam 0.75-1.5 mg per day, Novopasit 1 tablespoon 3 times a day), antidepressants (amitriptyline 25 - 100 mg per day). When using antidepressants, it is necessary to remember that they can cause orthostatic hypertension and heart rhythm disturbances.

In the case of severe paresthetic syndrome, 1 tablet of phenibut per night can be prescribed. 3 in order to reduce the phenomena of hyposalivation, potassium iodide, bromhexine, mouthwash with ethonium, artificial and natural sialic substitutes are used. Vascular agents (trental, cavinton, cinnarizine) are used to improve microcirculation. The purpose of vitamins (thiamine, cyanocobalamin, ascorbic acid), vegetotropic drugs (beloid, belataminal, platyfillin), antihistamine drugs (pipolfen, fenkarol) is shown. Local anesthetic mixtures (anesthesin-metacin paste, novocaine solution, lidocaine) are used for the inhibitory effect on peripheral mechanisms.

Reflexotherapy methods (acupuncture, electropuncture, including intraoral, transcutaneous electronic neurostimulation) are successfully used in complex treatment. Physiotherapy methods include endonasal electrophoresis of novocaine, massage, galvanization of the upper cervical sympathetic nodes. In recent years, the positive analgesic effect of laser acupuncture has been proven.

All patients with glossodynia need treatment from a psychotherapist.

Bilateral damage to the nuclei of the IX, X, XII pairs leads to bulbar paralysis, and bilateral damage to the supranuclear pathways leads to pseudobulbar paralysis.

Bulbar paralysis is always more severe, patients often die from aspiration pneumonia.

Bulbar palsy develops according to the peripheral type, since the nuclei of motor cranial nerves are the second, peripheral neurons.

Pseudobulbar palsies are central, as the first, central neurons of the motor pathways are damaged.

Symptoms of bulbar paralysis:	Symptoms of pseudobulbar paralysis:
Dysphonia (aphonia)	Dysphonia
Dysphagia (Aphagia)	Dysphagia
Dysarthria (anarthria)	Dysarthria
Atrophy of the tongue muscles, fibrillar twitching of the muscles	Trophics of the tongue muscles are not disturbed
Foul tone of the tongue (rhinolalia)	Foul tone of the tongue (rhinolalia)
Reduction or loss of reflexes from the pharynx and soft palate.	Reflexes from the pharynx and soft palate are alive or even enhanced
	Violent laughter or crying

Alternating syndromes:

This is a unilateral lesion of the brainstem, which consists in a violation of the function of one or more CNS on the side of the cell according to the peripheral type and central paralysis or paresis of the limbs or conduction disorders of sensitivity on the side opposite to the cell.

Depending on the level of damage to the brain stem, peduncular, pontine and bulbar syndromes are distinguished. They are named after the scientists who first described them.

Bulbar alternating syndromes:

(with damage to the medulla oblongata)

o	The name of the syndrome	Damage to the nerve on the side of the cell	Symptoms on the opposite side
.	Jackson	12 ChN	Hemiplegia, hemiparesis (central)
.	Avellis	12, 9 and 10 ChN	Hemiplegia, hemiparesis
.	Schmidt	9, 10, 11, 12 ChN	Hemiplegia (sometimes hemianaesthesia)
.	Wallenberg-Zakharchenko	10 CN + Claude-Benard-Horner syndrome (damage of sympathetic fibers) + disorder of pain and temperature sensitivity according to the segmental type on the face (damage of nucl. Tractus spinalis 5 CN), + hemiataxia and nystagmus (damage of cerebellar pathways).	Hemianesthesia of surface sensitivity (alternating hemianesthesia). Hemiplegia.

Certain alternating syndromes can be observed in tumors, aneurysms, skull injuries, inflammations and parasitic diseases of the brain, but most often - in the case of impaired cerebral blood circulation (ischemic softening centers in the trunk).

Materials for self-control of training quality

Tests

1) The nuclei of the following cranial nerves are located in the medulla oblongata:

Answer options:

a) the nucleus of the hyoid nerve;

b) double core of IX, X, XI pairs ;

c) the nucleus of the single bundle of the IX, X pairs and the nucleus of the spinal cord of the V pair ;

d) the lower salivary nucleus of the IX pair and the dorsal nucleus of the X pair;

e) all of the above

Correct answer: e)

2) What nuclei are in the medulla oblongata?

Answer options:

a) core of the lower olive;

b) nuclei of the reticular formation;

c) Holl nuclei;

d) Burdach nuclei;

Correct answers: a, b, c, d)

3) What centers are located in the medulla oblongata?

Answer options:

a) breathing;

b) swallowing;
c) vascular and motor;
d) sweating;
Correct answer: a, b, c)

Tasks for self-control :

1. Where is the focus of the lesion in bulbar paralysis and what symptoms are characteristic of it?
   The correct answer: the medulla oblongata is affected, namely the nuclei of the IX, X, XII pairs of the CMN. Dysphonia, dysphagia, dysarthria, atrophy of tongue muscles.
2. When are alternating paralysis formed, and what are the main symptoms characteristic of them?
   The correct answer: with focal processes in the brain stem. The main symptoms: on the side of the focus - CNS damage (depending on the level of damage), on the opposite side - central hemiparesis.
3. The patient complains of impaired speech and poor swallowing. An objective examination revealed atrophy of the tongue muscles, fibrillar twitching of the latter, decreased reflexes: pharyngeal and soft palate, dysphonia, dysphagia, dysarthria. What kind of paralysis does the patient have and where is the focus of the lesion?
   Correct answer: bulbar palsy, the medulla oblongata is affected.
4. Patient T., 64 years old, after repeated disruption of cerebral blood circulation in both hemispheres of the brain, had disordered speech and swallowing, and had bouts of violent crying. Objectively: dysphonia, dysphagia, dysarthria. There is no atrophy and fibrillary twitching of the tongue. Pharyngeal reflexes are strengthened, there are reflexes of oral automatism - proboscis, sucking, naso-labial. Attacks of violent crying are periodically observed.
   Question:
   1) What is the patient's syndrome?
   2) Where is the lesion?

   Right answer:
   1) Pseudobulbar syndrome
   2) Cortical-bulbar pathways were affected in both hemispheres of the brain.

Materials for classroom self-training:
List of educational practical tasks:
1. Master research methods:
   a) mobility of the soft palate;
   b) reflexes from the pharynx and soft palate;
   c) speech articulation;
   d) functions of the sternocleidomastoid muscle;
   e) trophics of the tongue;
   f) functions of the trapezius muscle
2. Diagnose bulbar paralysis;
3. Diagnose pseudobulbar paralysis

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 10

Topic: Pathology of the autonomic nervous system

Purpose: to create in the students an idea of the importance of the normal functioning of the autonomic nervous system for a balanced adaptation of the body to the conditions of the external environment.

Basic concepts: the autonomic (autonomic) nervous system is often affected after

craniocerebral injuries, inflammatory diseases of the brain and meninges, with neuroses and other disorders of the nervous system. The ability to diagnose dysfunction of the autonomic nervous system helps the doctor to prescribe adequate treatment to the patient in a timely manner and to improve adaptation capabilities.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Modern physiology defines the autonomic nervous system as a part of the nervous system that regulates the activity of internal organs and metabolism in the body. Along with its direct regulatory activity of internal organs, glands, vessels, and smooth muscles, the autonomic nervous system performs an adaptation-trophic function, adapts organs and tissues to the best and most perfect performance of activities regulated by the somatic nervous system.

The autonomic nervous system functions in close contact with the endocrine system, with humoral factors, neurotransmitters, electrolytes and metabolites, forming with them a rather complex complex that ensures the integrity of the body and the stability of its internal environment (homeostasis).

There is a close relationship between the autonomic and somatic nervous systems. The cranial and spinal nerves contain fibers of the autonomic nervous system. The main morphological unit of the autonomic nervous system, like the somatic one, is the neuron, and the main functional unit is the reflex arc.

The autonomic nervous system has a central department (cells and fibers are located in the brain and spinal cord) and a peripheral department (all other structures).

There are also parasympathetic and sympathetic divisions. The main difference between them lies in the functional innervation and is determined by the relationship to the means that act on the autonomic nervous system. So, for example, the sympathetic nervous system is excited by adrenaline, the parasympathetic – by acetylcholine. Ergotamine has an inhibitory effect on the sympathetic nervous system, and atropine has an inhibitory effect on the parasympathetic nervous system.

Anatomically, the autonomic nervous system, like the somatic nervous system, has central and peripheral divisions. For better understanding, it is divided into sympathetic and parasympathetic departments. Phylogenetically, both divisions are ancient formations. However, the parasympathetic division is phylogenetically older than the sympathetic.

The sympathetic nervous system contributes to the rapid mobilization of energy and adaptation of the body to the constantly changing conditions of the external environment. Through adrenergic structures, it provides somatovegetative correlation in various manifestations of the body's activity. In particular, in behavioral acts, in the processes of physical and mental work. This is basically an ergotropic system associated with catabolic (dissimilatory) processes.

The parasympathetic nervous system, on the contrary, contributes to ensuring the stability of the internal environment, manages the recovery processes of the body's losses of energy and nutrients, and increases the activity of assimilative processes. It plays a particularly important role in the regulation of digestion and certain phases of sleep. This is a trophotropic system associated with anabolic (assimilative) functions

Anatomy of the autonomic nervous system

In the central nervous system, there is a segmental apparatus of the autonomic nervous system (cells located in the brain stem and spinal cord), which give rise to preganglionic efferent fibers, and a suprasegmental section, which includes the nuclei of the hypothalamus, as well as the limbicoreticular complex and some sections of the associative zone of the cerebral cortex, which make mainly suppressive effect on the hypothalamus.

The cortex of the central lobe of the medial surface of the cerebral hemispheres (limbic lobe) is the cortical lobe of the visceral analyzer. The term limbic system belongs to the American scientist McClean (1952), who proposed his scheme of the structure and activity of the limbic system or visceral brain. According to McClean, the limbic system includes a number of cortical and subcortical structures, in particular:

- olfactory bulbs, tracts, triangle,
- anterior perforated substance (substantia perforata anterior).
- on the medial surface of the hemispheres, it includes:
- septum rellucidum,
- girdle gyrus (g.cinguli),
- seahorse twist,
- orbital lobes of the frontal lobe,
- the front part of the islet,
- the pole of the temporal lobe.

Important structures of the limbic system are the hippocampus, the dentate gyrus, the island of the old cortex near the corpus callosum (Idusium griseum), the base and prebase of the hippocampus.

The subcortical structures of the limbic system are the caudate nucleus, shell, amygdala nucleus, anterior (nonspecific) nuclei of the thalamus, hypothalamus, and frenum nuclei. Among the subcortical structures of the limbic system, the central place belongs to the hypothalamus. Thus, the limbic system is a complex intersection of ascending and descending pathways that form multiple closed circles anatomically connected to the reticular formation of the brain stem.

Physiology of the limbic system.

1. The limbic system is a visceral brain that receives afferent impulses from internal organs.

2. It takes part in the implementation of emotional reactions.

3. It is the nervous substrate of memory, which stores not genetically inherited, but acquired experience. Bilateral removal of the medial surface of the temporal lobes of the brain causes severe memory disorders, retrograde amnesia occurs, the ability to remember new things is impaired, and short-term memory suffers sharply.

4. The limbic system provides motivation for thirst, hunger, sexual desire, regulates the rhythm of sleep and alertness, participates in the regulation of the respiratory, cardiovascular, digestive, genitourinary systems, vegetative reactions, ensures the integration of the cardiovascular and respiratory systems, endocrine and vegetative systems functions. It influences the activity of the new cortex, makes visceral sensations conscious, especially in pathological conditions of internal organs.

The second representative of the central department of the autonomic nervous system is the hypothalamic area, in which higher subcortical mechanisms of regulation of sympathetic and parasympathetic innervation are carried out.

There are 32 groups of cell nuclei in the hypothalamus: central gray matter, paraventricular, supraoptic (n. supraopticus), nucleus of the gray hump (n. tuberis cinerei), infunbulo-tuberal nuclei. Posterior group (n. mamilloinfundubularis), nuclei of mammillary bodies, nuclei of Lewis bodies. In the walls of the III ventricle, there are paramedian and connecting nuclei. In the mammillary bodies, the nuclei of the mammillary bodies (mammillary, premammillary, medial, supramammillary, lateral). The hypothalamus is connected to other parts of the nervous system: the cerebral cortex, the visual humps, the extrapyramidal system, the lower nuclei of the brainstem and spinal cord, the reticular formation, and the pituitary gland. The front part of the hypothalamus is with the parasympathetic nervous system, the back part is with the sympathetic nervous system.

In the hypothalamus, 7 tropic neurohormones were found, activating the release of the corresponding pituitary hormone - releasing factors and 3 inhibiting (inhibiting) the release of tropic hormones from the pituitary gland.

Releasing factors include:

1. ACTH - releasing factor - corticoliberin
2. Thyriotropin - releasing factor - thyroliberin
3. Releasing factor of luteinizing hormone - luliberin
4. Releasing factor of the follicle-stimulating hormone - foliliberin
5. Momatotropin-releasing factor - somatoliberin
6. Prolactin-releasing factor - prolactoliberin
7. Releasing factor of melanocyte-stimulating hormone - melanocytoliberin

Inhibitory (inhibiting) factors include:

1. Prolactin - inhibitory factor - prolactostatin
2. Melanocytoinhibitory factor - melanocytostatin
3. Somatotropin-inhibiting factor - somatostatin

A change in the concentration of hypothalamic neurohormones affects the biosynthesis and release of the corresponding tropic hormones by the anterior lobe of the pituitary gland, which leads to the development of endocrine diseases: acromegaly, Itsenko-Cushing's disease, goiter, hypothyroidism, pituitary cachexia, pluriglandular syndrome, and others.

The hypothalamus takes part in the regulation of the following body functions:

1. Activities of the cardiovascular system
2. Thermoregulation
3. Water, carbohydrate, fat, mineral, mucopolysaccharide metabolism
4. Permeability of blood vessels and tissue membranes
5. Functions of endocrine glands both through the pituitary gland and through the sympathetic and parasympathetic divisions of the autonomic nervous system
6. Morphology
7. Functions of organs of the gastrointestinal tract.
8. Ensures stability of the internal environment
9. In adaptive adaptation, thanks to the cortical-subcortical relationship
10. Plays an important role in emotional behavior.

The hypothalamus is connected with the reticular formation and the limbic system.

The parasympathetic department of the autonomic nervous system is represented by four lobes:

1. hypothalamic
2. mesencephalic
3. bulbar
4. sacral.

In the hypothalamus, the parasympathetic nervous system is represented in the anterior part by the supraoptic nuclei. Its cells, preganglionic neurons, control the cells of the posterior lobe of the pituitary gland, which are modified postganglionic neurons.

In the mesencephalic section, the parasympathetic nervous system is represented by autonomic nerve cells and fibers of the oculomotor nerve (Yakubovich's nuclei and Perlea's nucleus at the bottom of the aqueduct of the midbrain), the fibers of which innervate the pupillary sphincter -–m. sphincter pupille and ciliary muscle (function of accommodation.

They belong to the bulbar department

- the upper salivary nucleus, which innervates the lacrimal and submandibular and sublingual salivary glands;
- the lower salivary nucleus, which innervates the parotid salivary glands
- the posterior core of the vagus nerve, from which fibers go to the larynx, trachea, bronchi, heart and other organs of the chest and abdominal cavity, that is, to all internal organs, walls, vessels, except for the pelvic organs.

The sacral division of the parasympathetic nervous system includes cell groups in the gray matter of the spinal cord at the level of II, III and IV sacral segments. Their axons form internal pelvic nerves (nn splanchnici pelvini), which innervate the muscles and mucous membrane of the pelvic organs (bladder, rectum, external and internal genitals).

The sympathetic division of the autonomic nervous system, in addition to the central representation in the hypothalamic division (posterior lobe), is represented by cell groups (first neurons) located in the gray matter of the lateral horns of the spinal cord from the VIII cervical to the II and III lumbar segments. The axons of these cells in the composition of the anterior roots, and then the white connecting branches (rr communicantes albi) enter the nodes of the sympathetic trunk (gangl. truncis sympathici), which are placed symmetrically in the form of chains on the sides of the spinal trunk, 20-25 nodes on each side. In the coccyx, both chains are connected by means of an odd knot (gangl. impar). Some fibers penetrate the nodes of the symmetrical sympathetic trunk and end in them or in intramural nodes.

In these nodes, there are second neurons, the processes of which go directly to one or another organ. Thus, prenodal (preganglionic) and postnodal (postganglionic) vegetative fibers are distinguished. Fibers that go to the nodes of the abdominal cavity, merging into large nerve trunks - the large internal nerve (n. splanchnicus major) from the V-IX thoracic nodes and the small internal nerve (n. splanchnicus minor) from the X-XI thoracic nodes. The largest prevertebral nodes are the paired abdominal node (gangl. celiacus), upper and lower mesenteric nodes (gangl. mesentericum superius et inferius).

Parasympathetic fibers from the vagus nerve join prevertebral and intramural nodes and plexuses. Sympathetic fibers in the muscular membrane of the stomach form the musculo-intestinal plexus (Auerbach - plexus niyenferius), and from it fibers go to the submucosal base of the gastric mucosa, forming the submucosal plexus (Meissner) - plexus submucosus). These plexuses extend to the intestine, esophagus and pharynx.

Part of the fibers of the cells of the lateral horns, which do not go to the paravertebral, prevertebral and intramural nodes, approach the somatic peripheral nerves and in their composition go to the muscles, blood vessels, skin and its appendages (sweat glands and muscles that raise the hair).

From the nodes of the sympathetic trunk, sympathetic fibers go to organs and parts of the body. Therefore, a certain clinical picture corresponds to the lesion of each node.

For example, a lesion of the upper cervical node (gangl. cervicale superius) is characterized by narrowing of the pupil, narrowing of the eye slit, enophthalmos (Bernard-Horner syndrome);

with damage to the cervicothoracic or stellate node (gangl. cervicothoracicum seu stellatum) heart disorders, pain and sensitivity disorders in the upper limb and upper chest are characteristic.

The thoracic part of the sympathetic trunk consists of 10-12 nodes. After the ganglionic fibers from them go to the intercostal nerves, vessels and organs of the thoracic and abdominal cavity; from the I-V nodes - to the cardiac plexus, from the V-X nodes - the large and small internal nerves go to the abdominal (solar) plexus and mesenteric nodes.

The lumbar region consists of 4-5 nodes, from which the fibers go to the sacral root nerves, the abdominal plexus, and the abdominal part of the aorta.

Sympathetic innervation does not have such a clear distribution as somatic:

- sympathetic fibers coming from VIII cervical and I, II, III thoracic segments innervate the face and neck;
- from IV-VII – upper extremity,
- from VIII and IX - trunk,
- from X-XII thoracic, I and II lumbar segments - the lower limb.

Sympathetic postganglionic fibers (together with parasympathetic) form a plexus around vessels and internal organs of the chest and abdominal cavity. The largest plexuses are the thoracic aortic, abdominal aortic, superior mesenteric, and superior hypogastric plexuses.

Peculiarities of the activity of the autonomic nervous system.

The autonomic nervous system regulates the processes that take place in organs and tissues.

However, these processes can be disturbed in case of dysfunction of the autonomic nervous system; numerous disorders arise. Most pathological processes in it are caused not by loss of functions, but by irritation, that is, increased excitability of the central and peripheral apparatus. Repercussions are a feature of the autonomic nervous system: disturbances in some parts of this system can lead to changes in others.

Clinical manifestations of lesions of the autonomic nervous system

Pathological processes localized in the cerebral cortex can lead to the development of trophic disorders in the innervation zone.

When the lobulus paracentralis is damaged, urination is disturbed according to the central type, and when the limbic-reticular complex is damaged, the emotional sphere (of a permanent or affective nature) is disturbed. Anorexia or bulimia appears (rice), sexual disorders, memory is disturbed in the type of amentary Korsaksky syndrome, in which the patient loses the ability to remember current events, sleep disorders, psychosensory disorders, changes in consciousness can be manifested by akinetic mutism, attacks of truncal and temporal epilepsy, disorders of the cardiovascular and respiratory systems.

Irritation of limbic structures is accompanied by an elevated background of mood, euphoria, extreme emotional reactions, excited restlessness, hypersexuality.

Functional suppression of the limbic system is manifested by a lowered mood background, abulia, a decrease in sexual desire, hypochondriac fixation of unpleasant sensations from internal organs, a state of panasthenia.

When the hypothalamus is damaged, various clinical manifestations (syndromes) may occur:

- Vegetative-vascular-visceral
- Exchange and endocrine
- Nervous-trophic

4. Neuromuscular
5. Violation of thermoregulation
6. Sleep disturbance
7. Psychopathological
8. Diencephalic epilepsy.

Most often, damage to the hypothalamus manifests itself in vegetative-vascular, neuro-endocrine disorders, disorders of thermoregulation, water, mineral, fat and protein metabolism, sleep and alertness disorders. Women aged 20-40 are more often ill.

Hypothalamic syndrome with vegetative-vascular disorders (vegetative-vascular form) is most common. In the inter-crisis state, patients complain of general weakness, increased fatigue, physical and mental exhaustion, increased sensitivity to changes in meteorological factors, pain in the heart, palpitations, a feeling of lack of air, and irregular breathing. Symptoms from the gastrointestinal tract are often leading: pain in the epigastrium, unpleasant sensations in the intestines, nausea, belching of air and bile, urges to defecate, sometimes diarrhea. An objective examination reveals invigoration of tendon and periosteal reflexes, asymmetry of blood pressure, its fluctuations with a tendency to increase, tachycardia, lability of the pulse, increased sweating, persistent diffuse red dermographism, trembling of the eyelids and fingers of outstretched hands, a tendency to allergic reactions. Emotional disorders (anxiety, fear), sleep disturbances are observed. More often, vegetative landslides have a sympathetic direction.

Less often, in the vegetative-vascular form, parasympathetic manifestations dominate, but they can be combined (mixed form). Against the background of permanent vegetative disorders, vegetative-vascular paroxysms (up to 15-20 minutes) or crises (from 15-20 minutes to 2-3 hours or longer) occur. More often, they are provoked by emotional stress, changes in weather conditions, menstruation, pain factors, stressful situations, etc. Attacks occur more often in the afternoon or at night, without warning. Sometimes attacks are preceded by changes in mood, headache, unpleasant sensations in the heart area, a state of brokenness, weakness.

Depending on the dominance of autonomic disorders, crises can be:

- sympathetic-adrenal,

- vago-insular (parasympathetic)
- mixed

In sympathetic-adrenal crisis:
- a headache appears or worsens,
- there is a heartbeat,
- numbness and coldness of the extremities,
- blood pressure rises to 150/90-180/110 mm Hg. art.,
- the pulse increases to 110-140 per minute,
- unpleasant sensations in the area of the heart, numbness, "goosebumps" are noted.
- in some patients, the body temperature rises to 38-39 0 C,
- there is excitement, motor restlessness,
- fear of death.

Vagoinsular crises are manifested:
- a feeling of heat in the head and face,
- headache, heaviness in the head,
- sometimes there are unpleasant sensations in the epigastric area, nausea,
- heart failure,
- general weakness, dizziness, sweating.

During the examination, the following is noted:
- slowing of the pulse (up to 45-50 per minute),
- decrease in blood pressure to 80/50-90/60 mm Hg.
- urges to defecate, increased intestinal peristalsis may occur,
- breathing becomes difficult,
- possible allergic manifestations in the form of urticaria or Quincke's edema.

Mixed crises. They are characterized by a combination of symptoms typical for sympathetic-adrenal and vago-insular crises or their alternating manifestations. Autonomic paroxysms can occur not only when the hypothalamic area is affected, but also the temporal lobes, brain stem, autonomic ganglia, plexuses, and other structures of the autonomic nervous system.

By frequency, they distinguish:

frequent crises - 5 or more per month (including daily)

average frequency - 3-4 per month

rare - 1-2 per month

Neuroendocrine-metabolic form. Damage to the hypothalamus in most cases is accompanied by a violation of the function of the glands of internal secretion and most often the functions of the pituitary gland. There are disorders associated with hyper- and hypofunction of the pituitary gland and other glands of internal secretion. Common endocrine disorders occur against the background of vegetative disorders. Violations of fat, carbohydrate, protein, water-electrolyte metabolism, appetite in the form of bulimia or anorexia, thirst, sexual disorders, isolated forms of endocrine gland damage may be observed (for example, diabetes insipidus, thyrotoxicosis, etc.). However, more often there are violations of the functions of a number of endocrine glands. Suppression of the gonadotropic function of the pituitary gland is often noted, manifested by amenorrhea or dysmenorrhea in women, a decrease in potency in men. Neuroendocrine syndromes may develop: Itsenko-Cushing, Froehlich-Babinsky adeno-genital dystrophy, Lawrence-Moon-Bardet-Biddle, Morganhi-Stuard-Morel, Prader-Willi, Kleine-Levin, Alström-Halgren, Edwards, Barraker-Simons leukodystrophy syndrome , Dercum's disease, Madelung's disease, and mixed forms of cerebral obesity. Early menopause in young women, changes on the part of the thyroid gland of hypothalamic genesis, and acromegaloid phenomena are noted.

Neurodystrophic forms. Trophic disorders of the skin (itching, dryness, neurodermatitis, ulcers, bedsores), muscles, bones (osteomalacia, sclerosing), widespread ulcers in the mucous membrane of the stomach, the lower part of the esophagus, acute perforations of the esophagus, stomach, and duodenum.

Neuromuscular syndrome is manifested by periodic myasthenic or myotonic disorders, as

well as paroxysmal myoplegia. Combinations of different types of muscle disorders are often observed.

Violation of thermoregulation. It is characterized by a prolonged subfebrile temperature with its periodic increase in the form of hyperthermic crises (up to 38-40 ^{0} C). Manifestations of vegetative dystonia of sympathetic-adrenal or mixed type are also noted. Temperature disturbances do not affect the well-being of patients. Their feature is that the temperature mostly rises in the morning and falls in the evening. Inflammatory changes in the blood and urine are not detected. The use of aspirin in such patients does not reduce the temperature.

Sleep and alertness disorders. They are manifested by difficulty falling asleep, superficial, disturbed sleep at night and drowsiness during the day. Pathological drowsiness is less common. Sometimes hypersomnia resembles separate symptom complexes: narcolepsy, Kleine-Levin syndrome, Pickwick syndrome and other hypersomnic manifestations. In the mechanism of the development of hypersomnia, a certain role is assigned to the reduction of the activating effect on the cortex of the cerebral hemispheres of the reticular formation, which is localized at the hypothalamic-stem level.

Hypothalamic syndrome with neuropsychiatric disorders is manifested by asthenia, sleep disturbance, and decreased mental activity. Patients have synestopathy, restlessness, hyperpathic syndrome or hypochondriac disorders, characteristic affective disorders, mood changes from depression to elevated background. Often there are crises with the picture of a vaso-vegetative storm, so-called "panic attacks".

When the mesencephalic part of the autonomic nervous system is affected, anisocoria, mydriasis, accommodation disorders, and Argyle-Robertson syndrome occur.

In case of damage to the bulbar department (nn. salivatotorii), lacrimation and saliva secretion are disturbed. When the nuclei of the vagus and glossopharyngeal nerves are affected, bulbar paralysis, disorders of respiratory function and cardiac activity occur.

Spinal vegetative syndromes. When the lateral horns of the spinal cord are affected, there are vegetative crises with vascular and sweaty signs, cyanosis, edema of the extremities, changes in pulse and breathing rates, and dysfunction of the abdominal cavity and pelvic organs.

When the ganglia of the adjacent trunk are affected, a picture of truncitis with causalgias appears. When the upper cervical sympathetic node is affected, ptosis, miosis, and enophthalmos (Horner's syndrome) occur.

When it is irritated - the expansion of the eye socket and pupil (Pti syndrome), paleness of the face, ear auricle.

Thoracic sympathetic truncated cells and lesions of the stellate node are accompanied by sensitivity disorders of the type of hyperpathy, breathing disorders, palpitations, lability of the pulse, arrhythmia, pain in the neck and head, with radiation to the abdominal areas. Attacks occur according to the type of angina pectoris.

Lumbar sympathetic truncytes are difficult to distinguish from thoracic ones. Sensations of the vegetative type are localized in the abdominal cavity and the corresponding lower limb. Sweating disorders and vascular disorders are observed.

When the segments S3-S5 are affected, there are disorders of the functions of the pelvic organs according to the peripheral type: intohtinentio vera (true urinary incontinence) or ischuria paradoxa (paradoxical urinary incontinence - the release of urine in drops when the bladder is full).

Peripheral autonomic syndromes are observed when the peripheral nervous system is damaged due to damage to autonomic nerve cells, fibers or due to functional changes in their activity. Syndromes of peripheral nervous disorders consist of vegetative and somatic signs. They are most often observed in polyradiculoneuritis, plexitis, mononeuropathies.

- When the tone of the parasympathetic nervous system increases, the following are noted:
- narrowing of the pupils,
- cold, wet, bluish skin,
- bradycardia, decrease in blood pressure,
- asthmatic breathing,

- increased salivation,
- sweating,
- increased acidity of gastric juice, spastic constipation, which is replaced by diarrhea, relaxation of sphincters,
- frequent urges to urinate,
- bright red dermographism, tendency to obesity, edema.

There is apathy, asthenia, depression, tendency to unconsciousness, drowsiness

The state of vagotonia is characteristic of a sleeping person suffering from bronchial asthma.

The sympathicotonia is characterized by:
- shiny eyes, exophthalmos, wide pupils,
- pale dry skin,
- tachycardia, increased blood pressure,
- tendency to waste,
- weakened intestinal peristalsis, tendency to constipation,
- expansion of the bronchi,
- paresthesias, chills,
- unpleasant sensations in the area of the heart, retention of urine and stool.

Such persons do not tolerate sunny weather, bright light, noise, tremors, fear, wakefulness often appear,

In pathological conditions, both departments of the autonomic nervous system do not act antagonistically, but synergistically.

With frostbite (frostbite), the tone of both departments of the autonomic nervous system increases. In shock states - decreases.

Amphotony - balance. Hyperamphotonia is noted during puberty, and hypoamphotonia - during the involution period. Normotonia – balance of both departments of the autonomic nervous system (diphtonia – imbalance).

Vegetative dysfunction can be:
- generalized
- regional - manifested by changes in one visceral system (cardiovascular, digestive, genitourinary, thermoregulation, etc.)

Vegetative dysfunction can have:
- permanent
- paroxysmal character.

It should be noted that the majority of pathological processes in the autonomic nervous system are caused not by loss of its functions, but by irritation, that is, increased excitability of the central and peripheral apparatus. Repercussions are a feature of the autonomic nervous system: disturbances in some parts of this system can lead to changes in other parts.

Methods of research of the autonomic nervous system

Skin manifestations of vegetative disorders: discoloration, erythema, pigmentation, moisture, swelling, greasiness, hair growth (hypertrichosis, alopecia), nail trophic (thickening, delamination, fragility, Mesa strips), trophic ulcers, skin changes after herpes. Determination of iris color and pigmentation, size (mydriasis, miosis), uniformity (anisokoria). Claude-Bernard-Horner syndrome - ptosis, miosis, enophthalmos - occurs when cervical sympathetic nodes are affected.

Vascular reflexes in case of mechanical irritation of the skin. Distinguish between white (sympathicotonia); pink (Eytonia); red, elevator (vagotonia).

Reflex dermographism – stroke pressure with a sharp object. It disappears when the responsible segments of the spinal cord are affected.

Sweat reflexes are studied when the body is warmed up, the introduction of diaphoretic substances (pilocarpine, aspirin). Minor's method (iodostarch test). Research of electrocutaneous resistance (SHGR - galvanic skin response or evoked skin sympathetic potentials - VSHSP).

Study of the permeability of blood vessels (hydrophilicity) by McClure and Aldrich.

Pharmacological tests (histamine, adrenaline, etc.). The most convenient is the study of vegetative-cardiac reflexes.

- Study of autonomic and vascular reactions of the skin with the help of ultraviolet radiation - acceleration and deceleration of the reaction. Suppression of erythema in segmental lesions of the central nervous system.

- Thermal imaging study - objectifies some reflex-vascular reactions during scanning of infrared radiation against the background of viscero-cutaneous connections, during pain reactions, etc.

Currently, the study of the "vegetative portrait", which includes:

The study of vegetative tone (special tables) allows to assess the state of the nervous system at rest.

The study of autonomic reactivity - with the help of a large number of cardiovascular tests (for example, Dan'ini-Aschner, Thomas-Roux) - evaluates the speed of response to irritation and recovery after bringing the brain out of a state of rest, which is important for assessing the severity of damage to the central nervous system, prognosis and others

Vegetative maintenance of activity (physical, mental, emotional) - assesses the ability of the central nervous system to long-term support of vegetative reactivity at a certain level.

It is also necessary to palpate the projection of nodes of the sympathetic trunk, plexuses (periarterial, cervical, solar, etc.).

Research of vegetative algic points of Markelov-Birbrair, which are symmetrically located on the sides of the body and occur in the pathology of different departments of the central nervous system. Sliding palpation is performed. Pain reactions are assessed on a five-point scale. Construction of "algic curves" can determine regional, generalized, symmetrical, asymmetrical syndromes.

The study of sensitivity with the help of Zakharyin-Hed zones (viscero-cutaneous ligaments) indicate irritation of the segmental-peripheral visceral apparatus of the ANS.

Identify and differentiate paroxysmal autonomic disorders (dizziness, fainting, acrocyanosis, Quincke's edema, urticaria, vasomotor rhinitis, hypothalamic crises, trigeminal pain, migraine, sleep attacks, autonomic-visceral auras, etc.).

Cardiovascular tests:

The Kerdo index is used to assess tone in the cardiovascular system, the Kerdo index is calculated according to the following formula: $K = (1 – D/P)$, where K is the Kerdo index, D is diastolic pressure, P is pulse rate, eutonia, or vegetative equilibrium is observed when the value of the index is from -3 to +3, sympathicotonia – at values greater than +3, parasympathikotonia – at values less than -3.

The Aschner-Danini pericardiac reflex allows you to assess the autonomic reactivity of the parasympathetic nervous system. The reflex is induced by pressing for 20 seconds with the pads of the doctor's fingers on the front-side surfaces of the patient's eyeballs. Normally, the pulse rate decreases by an average of 8 beats in 1 minute. In the case of vagotonia, the pulse slows down by more than 10 beats per minute, in the case of sympathicotonia it does not change or becomes more frequent.

The Chermak reflex is very similar in mechanism to the Aschner reflex and occurs when the mm is pressed. sternocleidomastoidei.

Thomas-Roux solar reflex – pressing on the cells of the solar plexus projection, the reaction is similar to Aschner's reflex.

Prevel's orthostatic reflex consists in an increase in pulse rate and blood pressure as a result of a change in the position of the body in space - from horizontal to vertical and characterizes the vegetative support of activity. Normally, the pulse increases by 8-12 beats per minute, and blood pressure increases by 5-10 mm Hg.

Danielopolo's clinostatic reflex is characterized by a slowing of the pulse by 6-8 beats in 1 minute and a decrease in blood pressure by 5-10 mm Hg. in response to a change in body position from vertical to horizontal.

Lesions of the autonomic nervous system can manifest as psycho-autonomic disorders. Therefore, they conduct a study of the patient's emotional and personal characteristics, study the mental anamnesis, the possibility of mental injuries, perform a psychological examination using various methods and tests (Staberg, Eysenck, Kettel, Rorschach projective test, etc.).

Syndromes of dysfunction of the pelvic organs. Regulation of the function of urine excretion consists of two components: involuntary-reflex (at the level of the segmental apparatus of the spinal cord due to autonomic innervation of the non-striated muscles of the internal sphincter and detrusor of the bladder) and voluntary (involving the cerebral cortical zone, somatic fibers and striated muscles ligaments of the external sphincter, urinary canal, anterior abdominal wall and diaphragm of the pelvic floor).

Schematic innervation of the bladder can be depicted as follows:

1. The spinal center of parasympathetic innervation of the bladder is located in the lateral horns of the gray matter of the sacral spinal cord (segments S2 — S4).

Afferent impulses to this center come from the proprioceptors of the bladder wall through intervertebral ganglia S2 —S4, then the internal pelvic nerves (nn. splanchnici pelvici), pelvic nerve and posterior roots. The efferent part of the arc passes through the anterior roots, then the pelvic nerve (n. pelvicus) and terminates in the intramural parasympathetic ganglia of the bladder wall. Postganglionic fibers innervate the bladder detrusor and partly the internal sphincter.

2. The center of sympathetic innervation is located in the lateral horns at the level of L1 — L2 segments of the spinal cord.

Preganglionic fibers leave the spinal cord together with the anterior roots, pass through the sympathetic chain and terminate in the lower mesenteric node, where they pass to the second neuron. Postganglionic fibers in the composition of n. hypogastricus fit the non-striated muscles of the bladder. Efferent sympathetic fibers mainly regulate the lumen of the bladder vessels.

Automatic bladder emptying is provided by two segmental reflex arcs — parasympathetic and somatic. When the walls of the bladder are irritated due to its stretching, impulses are transmitted to the spinal parasympathetic center. From there, they go along the efferent fibers and cause the contraction of the bladder detrusor and the relaxation of the internal sphincter. The entry of urine into the urethra due to the opening of the internal sphincter leads to the start of a reflex arc for the striated sphincter. Relaxation of the external sphincter is accompanied by the release of urine. Such an involuntary call appears if the intravesical pressure increases by 5 mm Hg.

Voluntary regulation of the act of urination is carried out with the participation of the cortical sensory and motor zones of the bladder. Impulses reach the sensory area (gyrus fornicatus) from the sensory neurons of the intervertebral nodes S2-S4 through the posterior roots, posterior cords, and through the medulla oblongata. Via associative fibers, impulses from the sensory zone are transmitted to the motor neurons of the paracentral lobe, and then, as part of the pyramidal path, they reach the neurons of the anterior horns S2-S4. Further, the impulses go along the front roots through the genital plexus and in the composition of n. pudendus reach the external sphincter. Voluntary regulation of urine output includes, in addition to controlling the external sphincter of the urethra, also controlling the muscles of the abdomen, diaphragm, and pelvis.

In the case of a lateral violation of the cortical-spinal connections (transverse damage to the spinal cord at the cervical or thoracic level), a pathology of voluntary regulation of urine output occurs. If the disorder occurs acutely, retention of urine (retentio urinae) first develops. In the future, in connection with the increase in reflex excitability of the segmental apparatus of the spinal cord, urinary retention is replaced by periodic incontinence (incontinentio interimttens).

In milder cases, imperative urges to urinate are observed.

In the case of acute processes, in which the parasympathetic innervation of the bladder is disturbed, there is a delay in the release of urine. Denervation of the internal and external sphincters is accompanied by true urinary incontinence (incontinentio vera). Such incontinence develops in case of damage to the sacral segments of the spinal cord, the roots of the horse's tail, n. hypogastricus and n. pudendus.

Paradoxical urinary incontinence (ishuria paradoxa) occurs in the presence of elements of

urinary retention (the bladder does not empty arbitrarily) and urinary incontinence due to mechanical overstretching of the sphincter.

The mechanism of violation of the act of defecation is the same as that of urinary incontinence. Delayed bowel movements indicate bilateral damage to the pyramidal tract. Faecal incontinence can occur simultaneously with urinary incontinence.

Bilateral damage to the pyramidal tracts or peripheral spinal centers at the level of the sacral segments of the spinal cord causes a violation of sexual intercourse.

Materials for self-control of training quality

Tests

1) How is the autonomic nervous system divided?

Answer options:
a) cortex;
b) striary;
c) pretty;
d) parasympathetic
Correct answer: c, d)

2) Where are the higher vegetative centers located?

Answer options:
a) in the cortex;
b) in the thalamus;
c) in the hypothalamus;
d) in the medulla oblongata
Correct answer: c)

3) What vegetative samples do you know?

Answer options:
a) dermographism;
b) Romberg test;
c) Stewart-Holmes test;
d) ortho-clinostatic test.
Correct answer: a, d)

4) To diagnose a violation of the animal nervous system research?

Answer options:
a) muscle tone;
b) coordination of movements;
c) reflexes;
d) pilomotor reaction of the skin;
e) clinoorthostatic test.
Correct answer: a, b, c)

5) To diagnose a violation of the autonomic nervous system research?

Answer options:
a) muscle tone;
b) coordination of movements;
c) reflexes;
d) pilomotor reaction of the skin;
e) clinoorthostatic test.
Correct answer: d, e)

6) What is observed when the parasympathetic nervous system is irritated?

Answer options:
a) muscle weakness;

b) tachycardia;
c) bradycardia;
d) violation of movement coordination;
Correct answer: c)

7) What is observed when the sympathetic nervous system is irritated?

Answer options:
a) muscle weakness;
b) tachycardia;
c) bradycardia;
d) violation of movement coordination;
Correct answer: b)

8) What are the most typical symptoms of irritation of the parasympathetic nervous system?

Answer options:
a) tachycardia;
b) bradycardia;
c) bradycardia;
d) increase in blood pressure;
f) decrease in blood pressure;
f) skin hyperemia;
g) paleness of the skin
Correct answer: b, d, e)

9) What symptoms are most typical for irritation of the sympathetic nervous system?

Answer options:
a) tachycardia;
b) bradycardia;
c) bradycardia;
d) increase in blood pressure;
f) decrease in blood pressure;
f) skin hyperemia;
g) paleness of the skin
Correct answer: a, c, g)

Tasks for self-control :

1. The patient received a closed craniocerebral injury. After some time, he began to complain of headache, sweating, palpitations. An objective examination revealed tachycardia - a pulse of 110 per second, fluctuations in blood pressure from 110/70 to 90/50 mmHg, hyperhidrosis of the distal parts of the limbs, red diffuse dermographism, asthenization. Make a diagnosis and determine the topic of the lesion.

Correct answer: post-traumatic vegetative-vascular dystonia; focal lesion - hypothalamic zone.

Materials for classroom self-training:

List of educational practical tasks:

1. Master research methods:
   a) dermographism;
   b) pilomotor reaction;
   c) hydrophilicity of the skin;
   d) Danini-Ashner reflex;
   e) clino-orthostatic test.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 11

Topic: Localization of functions in the cerebral cortex. Injury syndromes

Purpose: to ensure the acquisition of knowledge and skills related to: anatomy and physiology of the cerebral cortex, clinical symptoms and methods of determining various forms of aphasia, agnosia, apraxia; determination of the syndromes of damage to each part of the hemispheres of the cerebrum; symptoms of damage to the left and right hemispheres of the brain

Basic concepts: with various lesions of the cerebral hemispheres (acute disorders of cerebral blood circulation, craniocerebral injuries, including childbirth, encephalitis, tumors, etc.), disorders of the higher cortical functions of a person occur. For their study (language, gnosis, praxis, etc.), the problem of localization of functions in the cerebral cortex becomes important. The correct methodological approach to the main regularities of brain activity makes it possible to understand the localization of functions in the cortex as a dynamic process determined by the conditions of the internal and external environment.

Language is a means of communication between people, serves for the exchange of information and is an important function of the nervous system. Violation of language functions leads to a disorder of the IU signal system, leads to deep disability.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

The cerebrum consists of two hemispheres - left and right. They are divided among themselves by a longitudinal gap of the cerebrum, in the depth of which there is a large adhesion that connects both hemispheres. This adhesion consists of nerve fibers and is called the corpus callosum. The surface of each hemisphere is covered with a cortex consisting of cells and divided by a large number of furrows. Areas of the cortex located between the furrows are called gyri. The deepest furrows divide each hemisphere into lobes: frontal, parietal, occipital, and temporal. Each hemisphere has three surfaces: the upper lateral (superior), medial (inner) and lower surfaces. The largest furrow on the upper lateral surface of the cerebrum hemisphere is the lateral (side) furrow, which is also called the Sylvian furrow after the author's name. The bottom of this furrow is the insular portion (islet). The lateral furrow separates the temporal lobe from the parietal and frontal lobes. The second large furrow of the upper lateral surface of the hemisphere is the central one, which is also called Roland's furrow after the author's name. It separates the frontal lobe from the parietal lobe. At the back, the parietal lobe adjoins the occipital lobe. The border between them is the parietal-occipital groove, located mainly on the medial surface of the cerebral hemisphere. The furrow located in front of the central furrow is called the precentral furrow. Together they make up the precentral gyrus. The precentral gyrus belongs to the frontal lobe. On the lateral surface of the frontal lobe, in addition, three horizontally located gyri are distinguished - the upper, middle and lower frontal gyri. Behind the central sulcus is the postcentral sulcus, between which is the postcentral gyrus. The postcentral gyrus already belongs to the parietal lobe. The intraparietal groove divides the parietal lobe into the upper and lower parietal lobes. In the latter, supramarginal and angular gyri are distinguished. The upper, middle and lower temporal gyri are located longitudinally on the upper lateral surface of the temporal lobe. The number and direction of convolutions and grooves of the upper lateral surface of the occipital lobe of the hemispheres is very variable. On the medial surface of the hemispheres, above the corpus callosum, there is an arcuate cingulate gyrus that passes into the parahippocampal gyrus (seahorse gyrus). Convolutions of the upper lateral surface of the frontal, parietal, and occipital lobes also pass to the medial

surface of the hemispheres. At the border between the frontal and parietal lobes lies the paracentral lobe, which is formed as a result of the transition of the precentral gyrus into the postcentral gyrus. On the inner surface of the occipital lobe there is a furrow, above which lies the gyrus, which is called the wedge, and below it is the lingual gyrus. The furrows and convolutions of the lower surface of the hemispheres belong to different lobes. The white matter of the cerebral hemispheres is represented by myelin fibers (nerve cell processes), which have different functional significance. Associative fibers connect the gray matter of the cortex within one hemisphere; commissural fibers connect mostly symmetrical areas of the cortex located in different hemispheres; projection fibers are conductive paths that connect the cortex with the lower parts of the brain and with the spinal cord. Conductive pathways form a radiant crown, which consists of fan-like scattered fibers, and an internal capsule. The latter lies between the basal nuclei. It distinguishes the front and back legs and the knee placed between them. The front leg separates the caudate nucleus from the lenticular nucleus, the posterior one separates the lenticular nucleus from the thalamus.

The study of the structural features of the structure of the crust is called architectonics.

Cells of the cortex of the large hemispheres are less specialized. than neurons of other parts of the brain; however, certain groups of them are anatomically and physiologically closely related to certain specialized parts of the brain. The microscopic structure of the cerebral cortex is different in its various departments. These morphological differences of the cortex made it possible to distinguish separate cortical cytoarchitectonic fields. There are several variants of crustal field classifications. Most researchers allocate 50 fields. The microscopic structure of the cortex is quite complex. The cortex consists of a number of layers of cells and their fibers. The main type of structure of the crust is six-layered, but it is not uniform everywhere. There are areas of the cortex where one of the layers is expressed quite significantly, and the other - weakly. In other areas of the crust, some layers are subdivided into sublayers, etc.

It was established that the regions of the cortex associated with a certain function have a similar structure. Areas of the cortex, which are close in their functional significance in animals and humans, have a certain similarity in structure. The morphological and functional heterogeneity of the cerebral cortex made it possible to distinguish the centers of vision, hearing, touch, etc., which have their own specific localization. However, it is incorrect to speak of the cortical center as a strictly limited group of neurons. It is necessary to remember that the specialization of areas of the cortex is formed in the process of vital activity. In early childhood, the functional zones of the cortex overlap each other, so their boundaries are vague and unclear. Only in the process of learning, accumulating one's own experience in practical activities, there is a gradual concentration of functional zones into separate centers. The white matter of the large hemispheres consists of nerve conductors. According to the anatomical and functional features, white matter fibers are divided into associative, commissural and projection). Associative fibers unite different areas of the cortex within one hemisphere. These fibers are short and long. Short fibers usually have an arcuate shape and connect adjacent convolutions. Long fibers connect distant areas of the cortex. It is customary to call those fibers that connect topographically identical areas of the right and left hemispheres as commissural. Commissural fibers form three adhesions: anterior white adhesion, vault adhesion, corpus callosum. The anterior white commissure connects the olfactory regions of the right and left hemispheres. The vault junction connects the hippocampal gyrus of the right and left hemispheres. The main mass of commissural fibers passes through the corpus callosum, connecting symmetrical areas of both hemispheres of the brain. It is customary to call those fibers that connect the brain hemispheres from the lower level with the brain stem and spinal cord as projection fibers. As part of the projection fibers, there are conductive paths that carry afferent (sensory) and efferent (motor) information.

In the question of the localization of functions in the cerebral cortex, domestic neurology proceeds from the teachings of I.P. Pavlov on the dynamic localization of functions. On the basis of experimental studies, I.P. Pavlov proved that the cortex of the cerebrum is represented by a set of analyzers, where each of them has a central zone, the core of the analyzer, and a peripheral zone, where the cortical representation is scattered. As a result of this structure of the analyzer, its cortical

zones seem to overlap each other and form a closely related morphological association. Dynamic localization of functions in the cortex implies the possibility of using the same brain structures to provide different functions. This means that different parts of the cerebral cortex are involved in the performance of one or another function. For example, such higher mental processes as speech, writing, reading, calculation, etc., are never carried out by any isolated center, but rely on a complex system of cooperatively working areas of the brain. Dynamic localization of functions does not deny the presence of centers in the cerebral cortex, but their function is determined by connections with other areas of the cortex.

Studies of the functions of the cortex of the cerebrum For the diagnosis of diseases of the nervous system, studies of language and related reading, writing, numbers, as well as gnosis and praxis are of great importance. The study of language implies the study of several of its characteristics. First of all, they study the patient's understanding of language. To do this, he is offered to perform several simple tasks, without accompanying their explanation with facial expressions ("close your eyes", "show your nose", "take a spoon", etc.), and then gradually make the tasks more difficult. Impaired understanding of words is evidenced by the patient's speech, which does not make any sense while preserving the pronunciation of individual words. No less important is the definition of memory for words. The patient is shown various familiar objects, offering to name them. In the event of a speech disorder, the patient in this case feels difficulty and replaces the name of the object with a description of its properties. It is often enough to suggest the first syllable, and the patient remembers the forgotten name. When finding out the patient's understanding of speech, it is necessary to check whether he understands complex grammatical structures, since in some cases the speech disorder comes down to just such a defect. To this end, they are asked to explain what is the difference between, for example, a house in the dark and darkness in a house, etc. Together with the understanding of words, the character of the patient's own language is clarified. In severe cases, it may be completely absent, and the patient tries to communicate with gestures or facial expressions. Only individual sounds, exclamations are successful. With less pronounced speech disorders, the ability to pronounce individual words is preserved. Patients often pronounce them incorrectly, replacing one syllable or the whole word with another. The grammatical structure of the language may be disturbed. Dysarthria, which occurs in bulbar and pseudobulbar syndromes, chanted speech in cerebellar lesions, fading, monotonous speech in lesions of the extrapyramidal system, and hysterical mutism should be distinguished from speech disorders in cortical disorders. All these types of speech disorders are not accompanied by impaired understanding of words and are characterized by the ability to build words and phrases. Along with speaking, reading, writing, and numeracy are tested. When examining gnosis, the patient is shown familiar objects and asked to name them. Such gnosis, based on visual information, is called visual. When examining auditory gnosis, the patient, without seeing the source of the sounds, must determine their identity, for example, a fight or the ticking of a clock, a car horn, etc. Tactile gnosis (stereognosis) consists in recognizing objects by touch. To determine praxis, the patient is offered to perform some actions and move his arms or legs in a certain way, stand up, sit down, walk in a certain direction, etc. Then they offer to perform actions with imaginary objects, for example, show how to light a match, smoke a cigarette, write, use a fork, etc. Stages of diagnostic search for VKF violations.

1 stage. It is necessary to identify the presence or absence of VKF pathology in the patient, using the following information for this purpose: a) complaints of the patient about lack of speech, difficulty in speaking, reading, writing, arithmetic, gnosis, impossibility of performing acquired practical skills (dressing, combing hair, lighting matches, etc.); b) research of impressive and expressive language, writing, reading, arithmetic, praxis, gnostic functions; c) draw a conclusion about the presence or absence of cortical disorders.

2 stage. To clarify the nature of cortical disorders based on the examination of the patient and examination data, based on the following criteria:

APHASIA: a) motor – inability to repeat speech, lack of automated speech and narrative speech; b) sensory - misunderstanding of the meaning of words, the meaning of simple and

complex sentences, impaired phonemic hearing; c) amnestic - the inability to immediately name the displayed familiar object with knowledge of the purpose of the object, recalling the first letters or syllables helps to remember the name of the object.

APRAXIA: a) motor – impossibility of performing simple movements, everyday actions, actions with real and imaginary objects, impossibility of imitation (imitation) of actions: b) ideational – impossibility of performing a given action and its sequence, loss of sequence of actions (action algorithm). c) constructive – violation of constructive actions (construction of geometric shapes, assembling a whole from parts).

ALEXIA: failure to recognize read text, letters, impaired reading aloud and reading "about oneself".

AGRAPHIA: loss of the ability to write, to connect letters into words and words into sentences, as well as a violation of the sequence of writing syllables and a violation of writing under dictation.

AGNOSIA: a) visual – impaired recognition of objects, failure to recognize familiar faces, impaired visual perception of size and shape; b) auditory - impaired recognition of sounds, noises; c) gustatory - impaired recognition of food and other substances by taste; d) olfactory - impaired recognition of substances and food by smell; e) astereognosis - impaired recognition of familiar objects by touch.

AUTOTOPAGNOSIA: disorientation in one's own body - failure to recognize parts of one's own body, false perception of one's limbs, incorrect assessment of the size and shape of body parts (micropsia, macropsia).

ANOSOGNOSIA: unawareness of one's defect (paralysis of half of the body), illness.

APRACTOGNOSIA: not recognizing one's fingers and not being able to perform actions with them.

Methodology for determining aphasias.

The presence of fast spontaneous speech, understanding of speech and repetition of phrases are evaluated, the type of aphasia depends on the test results:

Differential diagnosis of aphasias

Type of aphasia and localization of the pathological focus	Presence of rapid spontaneous speech	Language comprehension	Repetition of phrases
Total	There is none	There is none	There is none
Broca (motor), posterior part of the inferior frontal gyrus on the left in right-handed people	There is none	IS	There is none
Wernicke (sensory), posterior part of the upper temporal gyrus on the left in right-handed people	IS	There is none	There is none
Transcortical motor (premotor area on the left in right-handed people)	There is none	IS	IS
Transcortical sensory (temporal-occipital area on the left in right-handed people)	IS	There is none	IS

Conductive or conductive (subcortical area of the blood supply of the left middle cerebral artery in right-handed people)	IS	IS	There is none
amnestic (anomia)	IS	IS	IS

Methods of detecting apraxias (kinetic, ideational, spatial, constructive).

In order to detect apraxia, the patient is offered to perform complex actions with real objects (doing hair, etc.), with imaginary objects (showing how to pour water, etc.), then it is suggested to perform actions (threaten with a finger, draw a plan of a room, make a figure out of matches). If the patient cannot perform a certain action, the doctor demonstrates it and offers to repeat it.

Kinetic apraxia occurs when the premotor zone of the cortex of the frontal lobe is damaged. It is characterized by a violation of both complex movements and actions according to the task, following imitation. Motor perseverations are characteristic, that is, involuntary repetition of the same movement.

Ideational apraxia occurs as a result of damage to the supramarginal gyrus of the left parietal lobe in right-handed people and is bilateral. The patient loses the plan or idea of a complex action, its phases are rearranged, cannot perform certain verbal tasks (threats with a finger), but can repeat the doctor's actions.

Spatial apraxia occurs as a result of damage to the inferior parietal and parietal-occipital regions of the left hemisphere. Leads to a violation of spatial relations. For example, a patient cannot draw a plan of a room according to the task.

Constructive apraxia is caused by damage to the left angular gyrus. At the same time, the patient cannot make a whole out of parts, for example, make a geometric figure out of matches.

Methods of detecting agnosias (visual, auditory, astereognosis, anosognosia).

Visual (optical) agnosia is tested by showing objects that the patient must name.

Auditory agnosia is tested by recognizing objects by their characteristic sounds (a clock ticks, a dog barks).

Stereognosis is examined by feeling things that the patient must name with closed eyes. There are the following requirements for checking stereognosis:

A) A preliminary check of tactile and muscle-joint sensitivity should not reveal their disorders, since pseudostereognosis occurs when these types of sensitivity are affected.

B) The subjects to be checked must be familiar to the patient.

C) The objects being tested should not produce familiar sounds.

Anosognosia is checked by detecting the patient's awareness of his defect (for example, paresis).

The frontal lobe of the cerebral hemispheres is located in front of Roland's sulcus and includes the precentral gyrus, premotor, and pole-prefrontal areas. 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 cortex 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. In the 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 monotype, that is, an arm or a leg is damaged depending on the location of the cortex lesion. Irritation of the precentral gyrus is accompanied by attacks of Jacksonian 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. In the case of damage to the posterior parts 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). In the presence of damage to the cortex of the premotor zone, patients develop 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 bradykinesia and oligokinesia, bradypsychia, slowing down of thinking processes, initiative is found. In the clinic, in the case of frontal injuries, there are other extrapyramidal disorders: grasping phenomena, involuntary automatic grasping of objects placed in the palm (Yanishevsky-Bekhterev reflex). Lesions of the frontal lobes may be accompanied by the appearance of reflexes of oral automatism (mouth reflex of Bekhterev, nasolabial reflex of Astvatsaturov and distant-oral reflex of Karchikyan), as well as subcortical reflexes (palm-chin reflex of Marinescu-Radovichi). 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 mouth. It is known that from the pole part of the frontal lobe, or the so-called prefrontal cortex, the frontal-pontine cerebellar pathways begin, which are included in the system of coordination of voluntary movements. In the case of their damage, cortical (frontal) ataxia occurs, which is manifested mainly by trunk ataxia, gait and standing disorders (astasia-abasia). In the case of light injuries, there is a wobble during walking with a deviation towards 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 in the presence of lesions of the cerebral cortex of various localization. But they are especially often observed in the case of frontal pathology. Changes in behavior and mental-intellectual disorders are observed. They come down to loss of initiative, personality, interest in the environment. Patients lack criticism of their actions: they are prone to banal jokes (moriya), euphoria. The sloppiness of the patient is typical. Such a peculiar change in the field of behavior and psyche is interpreted as frontal psyche. Of the symptoms that occur in the presence of damage to only the left frontal lobe (or the right in left-handed people), aphatic disorders are of topical and diagnostic importance. Motor aphasia is observed in the case of damage to Broca's zone, that is, the posterior part of the third frontal gyrus. 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.

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 (gyrus parietalis superior) and the lower parietal (gyrus parietalis inferior). In the latter, two gyruses are distinguished: the supramarginal gyrus (gyrus supramar ginalis), which covers the end of the sylvian sulcus, and the angular gyrus (gyrus angularis), which is directly adjacent to the upper temporal lobe. 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 of the body on the opposite side, i.e. according to the monotype, depending on the place of damage to the cortex. These disorders are more clearly marked 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 irritated areas 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 is accompanied by the development of astereognosis — the spread of the ability to recognize objects by feeling them with closed eyes. Patients describe individual qualities of an object, but cannot synthesize its image. A pathognomonic syndrome in the case of damage to the lower parietal lobe is the appearance of body diagram disorders. Damage to the parietal lobe of the left hemisphere of the brain (in right-handed people) leads to the occurrence of apraxia - a disorder of complex purposeful actions, provided elementary movements are preserved. Foci in the area of the supramarginal gyrus cause kinesthetic or ideational apraxia,

and damage to the angular gyrus is associated with the occurrence of spatial or constructive apraxia.

The temporal lobe is separated from the frontal and parietal lobes by the lateral furrow, in the depth of which is the Rayleigh island. On the outer surface of the parietal 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 occipitotemporal gyrus is located laterally, and the hippocampal 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), taste (cortex around the insula), and olfactory (parahippocampal 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), there is a center for understanding speech (Wernicke's zone). Efferent pathways diverge from the temporal lobe to all areas of the cortex (frontal, parietal, occipital), as well as to the subcortex and trunk. Therefore, if the temporal lobe is affected, there are violations of the functions of the corresponding analyzers, disorders of higher nervous activity. In case 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 perceptive devices on the periphery is bilateral. In the case of bilateral damage to the temporal lobes, auditory agnosia occurs. Attacks of vestibular-cortical vertigo, which are systemic in nature, are quite typical for damage to the temporal lobe. Ataxic disorders arise in the presence of processes in those areas where the temporal-bridge 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-quadrant 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. 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 operational 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 (fissura parietooccipitalis); on the outer surface, it does not have a clear border that separated it from the parietal and temporal lobes. The inner surface of the occipital lobe is divided by the fissura calcarina into a wedge (cuneus) and a gyrus lingual (gyrus lingualis). The occipital lobe is directly related to the function of vision. On its inner surface, in the area of the spur furrow, the visual pathways end, that is, there are primary projection cortical fields of the visual analyzer (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 foci of damage are insignificant, there are defects in the form of islands in the opposite fields of vision, so-called scotomas. Destruction of the cortex in the fissura calcarina, cuneus, gyrus lingualis 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. The most characteristic disorders caused by irritation of the cortex of the

inner surface of the occipital lobe are photopsia, flashes of light, lightning, and 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 cortical areas of olfactory (hippocampus, transparent septum, cingulate gyrus) and gustatory (cortex around the insular lobe) 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 of 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 the cortical olfactory and gustatory areas produces corresponding hallucinations. Frequent symptoms of damage to the limbic cortex are memory disorders of the Korsak syndrome type with amnesia, pseudoreminiscences (false memories), emotional disorders, phobias.

The corpus callosum connects the cerebral hemispheres. Symptoms of damage to the corpus callosum depend on the localization of the pathological process. In particular, in the presence of foci in the front parts (genu corporis callosi), mental disorders (frontal psyche) and frontal-callosis syndrome come to the fore. The latter is accompanied by akinesia, amimia, spontaneity, astasia-abasia, memory impairment, and decreased self-criticism. Patients have apraxic disorders, reflexes of oral automatism, 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; amnestic disorders, pseudoreminiscences, as well as psychoillusory disorders (already seen syndrome) are characteristic of damage to the fibers connecting the temporal lobes of the brain; foci in the posterior parts of the corpus callosum cause the development of optical agnosia. If the corpus callosum is affected, pseudobulbar disorders are often found. The subcortical parts of the brain include the white matter of the cerebral hemispheres and the internal capsule, the basal nuclei, and the diencephalon. Pathological processes (hemorrhage, ischemia, tumors, etc.) often develop simultaneously in several of the listed formations, but it is possible to involve only one of them (full or partial).

The white matter of the hemispheres and the internal capsule. On a horizontal section of the brain, you can see the white subcortical substance (centrum semiovale) with a radiant crown and an internal capsule. The internal capsule is a layer of white matter between the lenticular nucleus, on the one hand, and the head of the caudate nucleus and the thalamus, on the other. The front and back legs (thighs) and the knee are distinguished in the internal capsule. Pathological foci, which are located in the internal capsule, interrupt the pathways originating here and cause movement and sensory disorders on the opposite side. The front two-thirds of the posterior leg of the internal capsule is formed by the pyramidal tract, the cortico-nuclear pathway passes through the knee. The nature of the syndromes of their damage depends on the degree of spread of the pathological focus. Isolated foci in the knee are rare. Of course, they also extend to the front part of the hind leg. With such a lesion, there is a central hemiplegia contralateral to the focus with a Wernicke-Mann posture, a circumflex gait, typical for capsular lesions. At the same time, the central paresis of the tongue and the lower (color-mouth) part of the facial muscles is noted. The posterior third of the posterior leg of the internal capsule consists of axons of thalamus cells that conduct impulses of all types of sensitivity to the cerebral cortex and subcortical formations. An isolated lesion of the posterior third of the hind leg causes hemianesthesia and sensitive hemiataxia on the opposite side. Since the focus is located above the optic tubercle, sensitivity to gross pain and temperature irritations persists and often takes the form of hyperpathy. These syndromes can sometimes be joined by hemianopsia due to the destruction of the visual radiance associated with the very back

parts of the internal capsule. This hemianopsia differs from tractus in the preservation of the hemiopic reaction of the pupils to light. In this case, the central fields of vision may fall out, which is not observed when the cortex of the occipital fields (projection zone of the visual analyzer) is affected. Affecting the entire hind leg causes "three-hemi syndrome", but of a slightly different nature. Its structure includes hemiplegia, hemianesthesia and hemianopsia on the opposite side of the focus. Due to hemiparesis, hemiataxia does not develop in these cases.

Localization of functions in the cerebral cortex

Localization	Cortical syndromes
Frontal fate	- Paralysis, paresis - Anosmia - Mental disorders - Frontal ataxia - Motor aphasia -Agraphia - Visual paralysis - Tangible phenomena -motor Jacksonian epilepsy (when irritated)
Occipital fate	-Visual and color agnosia - Agnostic alexia - Homonymous hemianopsia -Visual hallucinations, photopsia (when irritated)
Crown fate	- Asteriognosia -Alexia - Autotopagnosia -Anesthesia - Sensitive Jacksonian epilepsy (when irritated)
Temporal fate	-Sensory and amnestic aphasia -Ataxia - Hemianopsia -Vestibular dizziness -Alexia - Auditory, olfactory, taste agnosia - Olfactory and auditory hallucinations (when irritated)

Left hemisphere	-Aphasia -Agraphia -Apraxia -Alexia - Acalculia
Right hemisphere	- Autotopagnosia - Anosognosia - Mental disorders

Chronic vegetative state syndrome is a symptom complex that sometimes occurs after the patient emerges from a coma and is characterized by recovery of alertness with complete loss of cognitive functions, the cyclical change of sleep and alertness can be preserved. The patient's eyes open involuntarily. Breathing, systemic blood circulation, and cardiac activity are functioning normally, but at the same time, movements, speech, and response to verbal stimuli are absent. The vegetative state, which lasts for 2-4 weeks, as a rule, gives rise to an absolutely bad prognosis - almost all patients die from intercurrent infections or bedsores. The morphological basis of the vegetative state is a massive lesion of the forebrain, often complete death of the cerebral cortex, hippocampus and basal nuclei, while the brainstem remains intact. The last circumstance distinguishes the vegetative state from brain death.

The syndrome of a "locked" person is characterized by the lack of an adequate response to external, including verbal, stimuli due to tetraplegia and paralysis of the bilbar, facial and masticatory muscles. The most common cause is a heart attack of the base of the bridge, which leads to the destruction of the cortico-spinal and cortico-nuclear pathways. Due to the preservation of supranuclear fibers to the oculomotor muscles, preservation of eye movements and blinking is noted. The reticular formation, which determines the absence of defects of consciousness, is also not damaged.

Brain death is a condition in which the brain is irreversibly damaged and does not function, while cardiac and respiratory functions are artificially supported. The diagnosis of brain death is based on the absence of brain activity, stem functions and the irreversibility of the condition. Brain death criteria: pupils fixed, dilated and unresponsive to light; coreal reflexes are absent; oculocephalic and oculovestibular reflexes are absent; there is no pharyngeal reflex; there are no purposeful movements or facial grimaces to painful stimuli applied to the limbs, trunk or face; the patient's respiratory center does not respond to an increase in the level of CO2 in the blood if the patient is disconnected from the ventilator; electrical silence of the brain (isoelectric EEG). Such tests should be repeated several times at intervals to ensure that the symptoms remain absent

Materials for self-control of training quality

Tests

1) What speech disorder occurs when the posterior part of the inferior frontal gyrus is damaged in the left hemisphere (in right-handed people)?

Answer options:
a) motor aphasia;
b) sensory aphasia;
c) sensory aphasia;
d) dysarthria;
f) amnestic aphasia.
Correct answer: a)

2) Left hemisphere damage syndromes?

Answer options:
a) aphasia;
b) apraxia;
c) agraphia;
d) autotopagnosia;
f) anosognosia
Correct answers: a, b, c)

3) Syndromes of damage to the right hemisphere?

Answer options:
a) aphasia;
b) apraxia;
c) agraphia;
d) autotopagnosia;
f) anosognosia
Correct answers: d, e)

4) Syndromes of damage to the occipital lobe?

Answer options:
a) visual agnosia;
b) agnostic alexia;
c) homonymous hemianopsia;
d) alexia, agraphia, acalculia;
f) apraxia.
Correct answers: a, b, c)

5) Syndromes of damage to the parietal lobe?

Answer options:
a) visual agnosia;
b) agnostic alexia;
c) homonymous hemianopsia;
d) alexia, agraphia, acalculia;
f) apraxia.
Correct answers: d, e)

Tasks for self-control :

1. The patient developed right-sided central hemiparesis after an acute disturbance of cerebral circulation. Expressive language is completely absent. The patient utters only 2 words - "good" and "no". Determine the localization of the focus?

Correct answer: the left middle cerebral artery and Broca's center were affected.

Materials for classroom self-training:

List of educational practical tasks:
1. Master research methods:
   a) motor aphasia;
   b) sensory aphasia;
   c) amnestic aphasia;
2. Diagnose angular syndrome.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 12

Topic: Cerebrospinal fluid, its changes. Meningeal syndrome

Purpose: to ensure the acquisition of knowledge and skills related to: anatomy and physiology of the membranes of the brain, clinical symptoms and methods of determining their irritation, to know the circulation of the cerebrospinal fluid and symptoms of changes in its pressure.

Basic concepts: irritation and inflammation of the meninges, as well as changes in the pressure of the cerebrospinal fluid can occur in the practice of any doctor, these conditions require a quick response to prevent death and disability of the patient.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

The brain and spinal cord are covered by three membranes - the outermost is the dura mater, then the arachnoid and pia mater. The membranes of the brain and spinal cord primarily perform a protective function. The hard shell consists of two leaves. The outer leaf forms the periosteum of the bones of the skull and the spinal canal; the inner leaf within the cranium is densely attached to the outer one, and only in some places the leaves of the hard shell diverge, forming the so-called sinuses in which venous blood circulates. Within the spinal canal, the leaves of the dura mater do not grow together, and between them there is a subdural space, which is filled with fatty tissue. The arachnoid membrane of the brain is located under the dura mater and has no blood vessels. It is separated from the hard shell of the brain by the subdural space, and from the soft one by the subarachnoid, filled with cerebrospinal fluid. The subarachnoid (subarachnoid space) within the cranium expands in places to form brain cisterns. Brain cisterns are mainly located on its lower (basal) surface. In this way, the brain, as it were, rests on a hydraulic cushion and thanks to this, it is not injured against the bones of the base of the skull during walking, running and other human movements. Within the spinal cord, the expansion of the subarachnoid space is expressed below the end of the spinal cord in the region of the horse's tail - the so-called terminal cistern. The soft shell of the brain covers the substance of the brain, which is very rich in blood vessels and nerves. It is closely connected with the brain substance, goes deep into it along the blood vessels (perivascular spaces). Penetrating into the ventricles of the brain (III, IV and lateral), it participates in the formation of vascular plexuses that produce cerebrospinal fluid. Cerebrospinal fluid, which fills the subarachnoid space of the brain and spinal cord, is produced by vascular plexuses located in the ventricles of the brain. From the lateral ventricles, the cerebrospinal fluid penetrates through the interventricular foramina (foramina of Monro) into the III ventricle, and then through the aqueduct of the midbrain into the IV ventricle and from it — through the foramina of Lyushka and Maghandi into the subarachnoid space of the brain and spinal cord. The outflow of cerebrospinal fluid occurs mainly through the venous system of the brain. CSF circulation scheme Cerebrospinal fluid ensures the normal functioning of the central nervous system. It protects the brain substance from mechanical damage when the body position changes, participates in the metabolism of the brain and spinal cord, delivering nutrients to them and removing metabolic products from them, and also maintains the stability of the brain's internal environment. In addition to the ventricles and subarachnoid spaces of the brain and spinal cord, cerebrospinal fluid is contained in the perivascular and pericellular spaces of the brain substance. The composition of cerebrospinal fluid includes water, cells (lymphocytes), protein substances, glucose, chlorides, electrolytes, trace elements, vitamins, hormones. The total amount of cerebrospinal fluid in an adult is normally 120-150 ml.

Meningeal symptom complex.

The syndrome of irritation of the meninges is one of the main syndromes in neurology. The clinic of this syndrome should be known by all medical workers because this syndrome occurs, as a rule, with two life-threatening diseases - with inflammation of the meninges (meningitis) and with subarachnoid hemorrhage. The most common symptom of the meningeal symptom complex is headache. The headache is very intense, usually diffuse, throbbing in nature. A headache, and this is a very important sign, is accompanied by nausea or even vomiting. This is the so-called cerebral vomiting, that is, vomiting that can occur even without previous nausea and that does not bring relief, unlike vomiting in the case of a disease of the gastrointestinal tract. In addition, any movement, sharp sound, bright light increase the headache. This is the so-called general hyperesthesia - that is, increased sensitivity to all stimuli. It is the second characteristic symptom of the meningeal symptom complex. The position of the patient in bed with irritation of the meninges is extremely characteristic. The so-called meningeal position occurs: the patient lies on his side with the head thrown back and the legs bent to the stomach, bent at the knee and hip joints. With this position, the tension of the spinal roots, which are enveloped by the meninges, decreases, and accordingly, the patient's pain in this position decreases. Meningeal pose is the extreme degree of severity of meningeal symptoms. There are cases when the meningeal position is not pronounced in a patient with irritation of the meninges. Then you should check the following meningeal symptoms, which in their essence are a provocation of a not sharply expressed meningeal posture.

Methodology for the study of meningeal signs (rigidity of the occipital muscles, symptoms of Kernig, Brudzinsky, Mendel, Bekhterev), the method of performing a lumbar puncture and assessing cerebrospinal fluid indicators in normal conditions and with meningitis of various etiologies.

Stiffness of the occipital muscles - an attempt to bend the patient's head causes a sharp increase in the tone of the occipital muscles.

Kernig's symptom - the patient's leg, which is in a horizontal position facing up, is bent at a right angle in the hip and knee joints, the extension of the lower limb in the knee joint is impossible due to the increased tone of the muscles of the back surface of the thigh.

Brudzinsky's upper symptom - during the examination of the stiffness of the occipital muscles, the patient's lower limbs involuntarily bend at the hip and knee joints.

Brudzynski's average symptom - when pressing on the area of the pubic symphysis, the patient's lower limbs involuntarily bend at the hip and knee joints.

Brudzynski's lower symptom - during Kernig's symptom check, the second lower limb of the patient involuntarily bends in the hip and knee joints.

Mendel's symptom - when pressing on the front wall of the external auditory canal, there is a sharp pain and a grimace of pain.

Bekhterev's symptom - percussion of the zygomatic arch causes a sharp headache and painful grimace.

Lumbar puncture (lumbar puncture, puncture of the subarachnoid space of the spinal cord, spinal puncture, lumbar puncture) — the introduction of a needle into the subarachnoid space of the spinal cord for diagnostic or therapeutic purposes.

For the timely detection of drug allergies and prevention of complications when using anesthetics in accordance with the instructions on the procedure for diagnosing drug allergies (order of the Ministry of Health and the AMS of Ukraine No. 127/18 dated 02.04.2002), a skin test must be performed (according to the methodology given in the appendix 2 to Order No. 127/18).

Purposes and indications for lumbar puncture

Lumbar puncture is performed for diagnostic or therapeutic purposes.

For diagnostic purposes, a puncture is performed to examine the cerebrospinal fluid. When analyzing the cerebrospinal fluid, the color, transparency, and cellular composition must be determined. It is possible to study the biochemical composition of the liquor, conduct microbiological tests, including its cultivation on special media. During a lumbar puncture, CSF

pressure is measured, the patency of the subarachnoid space of the spinal cord is examined using compression tests.

For therapeutic purposes, lumbar puncture is performed to remove cerebrospinal fluid and normalize cerebrospinal fluid circulation, to control conditions associated with combined hydrocephalus, as well as to rehabilitate cerebrospinal fluid in meningitis of various etiologies and to administer drugs (antibiotics, antiseptics, cytostatics).

There are absolute and relative indications for performing a lumbar puncture.

Absolute indications: suspicion of infection of the central nervous system (meningitis, encephalitis, ventriculitis), oncological lesions of the membranes of the brain and spinal cord, normotensive hydrocephalus; diagnosis of cerebrospinal fluid and detection of cerebrospinal fluid fistulas using dyes injected into the subarachnoid space; diagnosis of subarachnoid hemorrhage when CT scan is not possible.

Relative indications: fever of unclear origin in children under 2 years of age, septic embolism of vessels, demyelinating processes, inflammatory polyneuropathies, paraneoplastic syndromes, systemic lupus erythematosus, etc.

Contraindications to lumbar puncture:

In the presence of a voluminous brain tumor, occlusive hydrocephalus, signs of severe cerebral edema and intracranial hypertension, there is a risk of axial impingement during a lumbar puncture, its probability increases with the use of thick needles and removal of a large amount of cerebrospinal fluid. In these conditions, a lumbar puncture is performed only in cases of extreme necessity, and the amount of extracted cerebrospinal fluid should be minimal. If symptoms of wedging appear during puncture (currently this is a very rare situation), urgent endolumbar injection of the required amount of fluid is recommended. Other contraindications to performing a lumbar puncture are not considered so absolute. These include infectious processes in the lumbosacral region, blood coagulation disorders, taking anticoagulants and antiplatelet agents (risk of epidural or subdural hemorrhage with secondary compression of the spinal cord). Caution when performing a lumbar puncture (withdrawal of a minimal amount of cerebrospinal fluid) is necessary in case of suspicion of hemorrhage from an aneurysm of the cerebral vessels (risk of re-rupture) and blockade of the subarachnoid space of the spinal cord (risk of appearance or worsening of neurological deficit).

Lumbar puncture technique:

The patient lies on his side, the hips are brought to the body, the head is tilted forward, the assistant holds the patient firmly. The skin of the lumbar area is disinfected with a 5% alcohol solution of iodine, then thoroughly wiped with alcohol, the skin and subcutaneous fat in the puncture area are infiltrated with a 1% solution of novocaine. The needle is inserted between the spinous processes of the L3-L4 vertebrae. A needle with a mandrel is inserted sagittally in a slightly upward position, at a depth of about 5 cm the resistance of the yellow ligament is felt, after passing through which the needle pierces the dura mater, which is accompanied by a characteristic crunching sound, deepening by 2-3 mm, the needle ends up in the subarachnoid space. In order to carry out chemical and bacteriological research, 5-10 ml of cerebrospinal fluid are collected in two sterile test tubes. After that, the needle is removed and the puncture site is covered with a sterile napkin.

Measurement of CSF pressure and compression tests

Immediately after the appearance of cerebrospinal fluid in the pavilion of the needle, it is possible to measure the pressure in the subarachnoid space by connecting a plastic tube to the needle or a special system. During the pressure measurement, the patient should be as relaxed as possible. Normal fluid pressure in a sitting position is 300 mm H2O, while lying down it is 100-200 mm H2O. Indirectly, the pressure level can be estimated by the rate of outflow of the cerebrospinal fluid (60 drops per minute conditionally corresponds to normal pressure). The pressure increases with inflammatory processes of the meninges and vascular plexuses, impaired fluid outflow due to increased pressure in the venous system (venous stasis). Fluid dynamics tests are used to determine the patency of the subarachnoid spaces.

Queckenstedt test. After determining the initial pressure of the cerebrospinal fluid, compress the jugular veins for no longer than 10 seconds. At the same time, normally the pressure increases by an average of 10-20 cm H2O. and normalizes after 10 s after the compression is stopped.

During Stukei's test, a fist is pressed on the abdomen in the navel area for 10 seconds, creating stagnation in the inferior vena cava system, where blood flows from the thoracic and lumbosacral divisions of the spinal cord, epidural veins. Normally, the pressure also increases, but more slowly and not as significantly as with the Queckenstedt test.

Impurities of blood in the cerebrospinal fluid

Blood impurities in the cerebrospinal fluid are most typical for subarachnoid hemorrhage. In some cases, during a lumbar puncture, a blood vessel may be damaged, and an admixture of "traveler's blood" appears in the cerebrospinal fluid. In the case of intense bleeding and if it is impossible to obtain cerebrospinal fluid, it is necessary to change the direction or point of cotton to another level. When obtaining cerebrospinal fluid with blood, a differential diagnosis between subarachnoid hemorrhage and admixture of "traveler's blood" should be carried out. For this purpose, liquor is collected in three test tubes. With subarachnoid hemorrhage, the cerebrospinal fluid in all three test tubes is colored almost the same. In the case of a traumatic puncture, the cerebrospinal fluid from the first to the third tube will be gradually cleared. Another way is to assess the color of the supernatant fluid: yellow liquor (xanthochromic) is a reliable sign of hemorrhage. Xanthochromia appears 2-4 hours after a subarachnoid hemorrhage (result of hemoglobin degradation with erythrocyte disintegration). A small subarachnoid hemorrhage can be difficult to distinguish visually from inflammatory changes, in this case one should wait for the results of a laboratory study. Rarely, xanthochromia can be a consequence of hyperbilirubinemia.

Study of the composition of liquor

For a standard study, cerebrospinal fluid is taken in three test tubes: for general, biochemical and microbiological analyses.

Standard clinical analysis of cerebrospinal fluid includes assessment of density, pH, color and transparency of cerebrospinal fluid before and after centrifugation, assessment of total cytosis (normally no more than 5 lymphocytes per 1 μl), determination of protein content. The relative density of cerebrospinal fluid is normally 1.005-1.008, it is increased during inflammatory processes, decreased during excessive fluid formation. The normal pH is 7.35-7.8, it decreases with meningitis, encephalitis, and increases with syphilis of the brain, epilepsy, chronic alcoholism.

The yellow color of the cerebrospinal fluid is possible with a high protein content, in the case of a previous subarachnoid hemorrhage, and with hyperbilirubinemia. With metastases of melanoma and jaundice, the cerebrospinal fluid can be dark. Significant neutrophilic cytosis is characteristic of bacterial infection, lymphocytic cytosis is characteristic of viral and chronic diseases. Eosinophils are characteristic of parasitic diseases. If there are 200-300 leukocytes in 1 μl, the cerebrospinal fluid becomes cloudy. To differentiate leukocytosis caused by subarachnoid hemorrhage, a leukocyte count is necessary, taking into account the fact that there is approximately 1 leukocyte per 700 erythrocytes in the blood. The protein content normally does not exceed 0.45 g/l and increases with meningitis, encephalitis, tumors of the spinal cord and brain, various forms of hydrocephalus, block of the subarachnoid space of the spinal cord, carcinomatosis, neurosyphilis, inflammatory diseases. A significant role is also played by colloidal reactions — the Lange reaction ("golden reaction"), the colloidal mastic reaction, the Takata-Ara reaction, etc.

During the biochemical analysis of the cerebrospinal fluid, the content of glucose (normally within the range of 2.2-3.9 mmol/l) and lactate (normally within the range of 1.1-2.4 mmol/l) is assessed. The assessment should be carried out taking into account the fact that the glucose content of the cerebrospinal fluid depends on the blood glucose concentration (50-75% of this value). A decrease in glucose content is a frequent sign of meningitis of various etiologies (most often of bacterial origin, including tuberculous), an increase in the concentration of glucose in the cerebrospinal fluid is possible in ischemic and hemorrhagic stroke.

A reduced content of chlorides in the cerebrospinal fluid is characteristic for meningitis, especially tuberculous, for neurosyphilis, brucellosis, an increase - for brain tumors, brain abscess, echinococcosis.

In the microbiological laboratory, it is possible to stain a smear or sediment of the cerebrospinal fluid depending on the presumed etiology of the pathogen: according to Gram - if a bacterial infection is suspected, for acid-resistant microorganisms - if tuberculosis is suspected, ink - if a fungal lesion is suspected. Cultivation of cerebrospinal fluid is carried out on special media, including media absorbing antibiotics (in the case of massive antibiotic therapy).

There are a large number of tests for detecting specific diseases, for example, the Wasserman reaction, RIF and RIBT to rule out neurosyphilis, tests for various antigens for typing tumor antigens, determining antibodies to various viruses, etc. During a bacteriological examination, meningococci, pneumococci, hemophilic bacilli, streptococci, staphylococci, listeria, tuberculosis mycobacteria. Bacteriological studies of cerebrospinal fluid are aimed at identifying the causative agents of various infections: coccal group (meningococci, pneumococci, staphylococci, and streptococci) in meningitis and brain abscesses, treponema pallidus in neurosyphilis, mycobacterium tuberculosis in tuberculous meningitis, toxoplasma in toxoplasmosis, cysticercus bubbles — with cysticercosis. Virological studies of the cerebrospinal fluid are aimed at establishing the viral etiology of the disease (some forms of encephalitis).

Mode after lumbar puncture

After a lumbar puncture, it is recommended to stay in bed for 2-3 hours in order to avoid the post-puncture syndrome caused by continued leakage of cerebrospinal fluid due to a defect in the dura mater.

Lumbar puncture complications

The total risk of complications is estimated at 0.1-0.5%. Possible complications include the following.

Axial wedging:

acute wedging during puncture in conditions of intracranial hypertension;

chronic wedging as a result of repeated lumbar punctures;

Meningism.

Infectious complications.

Headaches, as a rule, pass in a lying position.

Hemorrhagic complications, usually associated with blood coagulation disorders.

Epidermoid cysts as a result of using low-quality needles or without mandrel needles.

Root damage (persistent pain syndrome may develop).

Damage to the intervertebral disc with the formation of a disc herniation.

Administration of contrast agents, anesthetics, chemopreparations, antibacterial drugs into the subarachnoid space can cause a meningeal reaction. It is characterized by an increase in cytosis up to 1000 cells in the first day, an increase in protein content with normal glucose content and sterile culture. This reaction usually regresses quickly, but in rare cases can lead to arachnoiditis, sciatica, or myelitis

Indicators of cerebrospinal fluid in normal conditions and with meningitis of various etiologies

stand up	Liquor pressure, mm Hg.	Cytosis in 1 mm^3	Protein content, g/l	Glucose content
Norm	100-200	Less than 5 lymphocytes	0.2-0.45	From 50% to 75% of the glucose content in the blood

Bacterial meningitis	Increased	100-60000, mostly neutrophils	0.5-5	Less than 40% of blood glucose
Tuberculous meningitis	Increased	10-500, lymphocytes	0.5-5	Much less than 40% of blood glucose
Fungal meningitis	Increased	25-500, mainly lymphocytes	0.5-5	Less than 70% of blood glucose
Viral meningitis	Normal or elevated	An increase in the number of lymphocytes	0.5-2	Norm

The following complications of lumbar puncture are noted:

1. Getting the needle into the bone of the vertebra; in this case, the needle should, without removing it from the tissues, be pulled towards you a few times, change the direction and try to insert it again.

2. The end of clean blood from the holes of the needle indicates that it has entered the blood vessel. In this case, it is necessary to remove the needle and make a puncture in another intervertebral space. If there is an admixture of blood in the cerebrospinal fluid (due to damage to the blood vessels during the puncture), then as it passes from the needle, the cerebrospinal fluid gradually becomes clearer.

3. The penetration of the needle into the root causes a sudden, sharp pain that radiates to the leg and disappears when the direction of the needle is changed.

4. A broken needle due to improper handling can lead to serious consequences.

5. After a lumbar puncture, as a result of irritation of the meninges, a post-puncture syndrome with the phenomena of meningism may develop.

Materials for self-control of training quality

Tests

1) What speech disorder occurs with meningitis?

Answer options:
a) motor aphasia;
b) sensory aphasia;
c) none;
d) dysarthria;
e) amnestic aphasia
Correct answer: c)

2) Symptoms of hypertensive-liquid syndrome?

Answer options:
a) aphasia;
b) decreased vision;
c) agraphia;
d) headache;
f) anosognosia.
Correct answer: b, d)

3) Symptoms of hypotensive-liquid syndrome?

Answer options:
a) aphasia;
b) decreased vision;

c) agraphia;
d) headache;
f) anosognosia.
Correct answer: d)

4) Symptoms of meningitis?

Answer options:
a) visual agnosia;
b) Kernig symptom;
c) pleocytosis in the cerebrospinal fluid;
d) stiffness of the occipital muscles.
Correct answer: b, c, d)

5) Symptoms of meningism?

Answer options:
a) visual agnosia;
b) Kernig symptom;
c) pleocytosis in the cerebrospinal fluid;
d) stiffness of the occipital muscles.
Correct answer: b, d)

Tasks for self-control :

1. The patient developed a headache after basal meningitis, and his vision deteriorated. Determine the previous diagnosis?

Correct answer: Obstructive hydrocephalus.

Materials for classroom self-training:

List of educational practical tasks:
1. Master research methods:
   d) Kernig's symptom;
   e) Brudzinsky's symptom;
   f) Bekhterev's symptom.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 13

Topic: Functional diagnosis of diseases of the nervous system

Purpose: to acquaint applicants with basic electrophysiological and X-ray methods of researching the nervous system and indications for their use.

Basic concepts: electrophysiological methods of examining the nervous system (both peripheral and central) are extremely important in the diagnosis of various neurological injuries. Therefore, the ability to correctly apply and interpret one or another examination significantly expands the doctor's capabilities and creates favorable conditions for the appointment of a specific and adequate treatment of patients. It is impossible to diagnose tumors, brain injuries, inflammatory and vascular diseases, demyelinating diseases and a number of others without X-ray and liquefaction studies. Therefore, the wide implementation of these methods in medical practice ensures correct diagnosis, and hence, timely and adequate treatment.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Radiological examination of the skull and spine

Conventional radiography of the skull and spine is an integral part of a detailed examination in cases of pathology of a traumatic, spondylogenic and neoplastic nature, but has a rather limited value in other diseases. The technique is relatively simple, and the obtained data can be interpreted by most radiologists. More important in neurology and neurosurgery are six special radiological methods, which currently make it possible to visualize most parts of the brain and spinal cord, as well as their vessels.

Computed tomography (CT). CT allows differentiation of epidural, subdural, and intracerebral hemorrhages, changes in the ventricular system with volume processes, and also detects tumors, abscesses, granulomas [when CT is performed after intravenous administration of contrast agents], areas of cerebral edema, foci of infarction, hydrocephalus, and atrophy brain The simplicity of this non-invasive procedure, the minimal risk for patients with large lesions, and the low dose of X-ray radiation revolutionized diagnostic neurology and neurosurgery.

NMR tomography. Modern use of nuclear magnetic resonance (NMR) allows visualization of brain lesions that are not detected by CT. The NMR-tomography method is non-invasive and does not involve the influence of ionizing radiation. This study provides the possibility of tissue delineation without contrast enhancement, and since bone tissue does not interfere, the method is particularly convenient for visualizing the basal structures of the brain, primarily in the posterior cranial fossa. The NMR method has already significantly expanded the diagnostic possibilities of neuroradiology, and in the future, NMR spectroscopy should become a method of determining metabolites in the brain. The high resolution of NMR-tomography in distinguishing between white and gray matter determines its wide application in order to establish the localization of lesions in the white matter, for example due to demyelination. The method is also used to visualize the spinal cord, both on sagittal and transverse "slices".

Angiography. This method has been refined over the past 30 years until it has become relatively safe and extremely valuable for the diagnosis of arterial occlusions, aneurysms and vascular malformations, tumors, abscesses, and intracranial hemorrhages. But, after the discovery of CT and NMR tomography, it began to be used much less often. After local anesthesia, a needle or cannula is inserted percutaneously into the lumen of the brachial or femoral artery; it is possible to introduce a catheter, passing it through the aorta with cannulation of the main arteries in the neck. The introduction of a contrast agent allows visualization of the aortic arch, the initial sections of the carotid and vertebral blood supply systems, the passage of blood vessels through the neck region into the skull cavity and, with appropriate indications, the arteries of the spinal cord. Under optimal conditions, it is possible to clearly show cerebral arteries with a lumen diameter of up to 0.1 mm, as well as small veins of a similar diameter, vascular anomalies (angiomas, aneurysms), occluded arteries, slowing of blood flow due to increased intracranial pressure at volume processes and blockages of the sinuses of the dura mater and veins, the displacement of vessels by a volumetric process or the impossibility of filling intracranial vessels during brain death. Digital subtraction venous angiography, in which a contrast agent is injected into the brachial vein under pressure, is considered as an alternative or additional method, especially if the initial examination of the diameter of the lumen of large extracranial arteries is necessary.

Pneumoencephalography and ventriculography. The introduction of air into the subarachnoid space during a lumbar puncture, performed in the patient's sitting position, enables detailed visualization of the size and location of the ventricles, the subarachnoid space (superior spinal and cerebral) and, indirectly, the structures located between the ventricles and the meninges. However, this method is rarely used at present. Ventriculography, which was carried out by injecting air or a contrast agent directly into the lateral ventricles, also has mainly historical significance. CT and NMR tomography have practically replaced both pneumoencephalography and ventriculography.

Radioactive isotopes. Radioactive isotopes, such as technetium (brain scan), are sometimes used to diagnose tumors, voluminous processes of inflammatory genesis, viral encephalitis, and some vascular lesions, such as heart attacks in areas adjacent to the blood supply of the brain, the detection of which is difficult by other methods. Since this method is simple and non-invasive, the only limitation to its use is the high cost. The more significantly the blood-brain barrier is affected by the pathological process, the more clearly the affected area is determined when using these methods. In order to detect the displacement of the middle structures of the brain by a volumetric process, ultrasound can also be used.

Positron emission tomography

Positron emission tomography (PET) is an experimental research method currently available at the disposal of only a few centers. The procedure includes systemic administration of positron-emitting radionuclides of oxygen or 18F-deoxyglucose (18FDH) followed by computer tomography. Administration of labeled O2, CO2, and 18FDH makes it possible to quantitatively assess regional oxygen consumption, blood flow, and glucose utilization. An examination of patients with cerebrovascular lesions, convulsive disorders, degenerations was carried out. With a stroke, it is advisable to perform PET in the acute stage in order to distinguish between viable and non-viable tissue. In patients with epileptic manifestations, the study with 18FDH in the inter-seizure period allows to detect areas of reduced glucose metabolism in and around the epileptic focus, while during the seizure, an increase in glucose metabolism is registered. During the study of metabolic processes using 18FDG, a decrease in glucose uptake in the striatum was also demonstrated in patients with Huntington's chorea, in whom no pathological changes were detected on CT.

Electromyography (EMG)

This technique is carried out during the clinical examination of patients with neurological diseases in which the neuromuscular apparatus suffers, that is, with primary and secondary lesions of the skeletal muscles. Advanced EMG technique ("central EMG") provides a quantitative analysis of motor system function.

Electroencephalography (EEG)

EEG is part of the clinical examination of a patient with suspected brain damage; it is also used to assess the involvement of the central nervous system (CNS) in many diseases.

In addition to recording at rest, so-called tests with stimulation are usually conducted.

1. The patient is offered to make deep breathing movements with a frequency of 20 times per 1 minute for 3 minutes. Developing alkalosis and cerebral vasoconstriction can provoke characteristic signs of convulsive readiness and other changes.

2. A powerful light source (strobe) is placed in front of the patient's face, which flashes with a frequency of 1 to 20 in 1 s, while the patient's eyes are open, then closed. As a result, patients with photosensitivity may develop pathological discharges.

3. EEG is recorded after the patient is allowed to fall asleep naturally, or after oral or intravenous administration of sedative drugs. Procedures 1 and 2 are used more often, but recording during sleep is exceptionally effective in detecting changes, especially in cases of temporal lobe epilepsy and some other convulsive conditions. A frequent reason for nocturnal EEG registration is previous research on sleep disorders.

In order for the EEG to be most effective, it is necessary to observe certain conditions. The patient should not be under the influence of sedative drugs, remain for a long time without eating, since both sedatives and relative hypoglycemia lead to changes in the normal structure of the EEG. The same applies to states of mental concentration, hyperexcitability, and drowsiness, each of which contributes to suppression of the normal alpha rhythm and amplification of muscle artifacts. When examining patients with a possible diagnosis of epilepsy who are already receiving treatment for this condition, most clinicians prefer to conduct the first EEG recording against the background of continued medication.

Types of normal records. In a normal EEG in adults, somewhat asymmetric sinusoidal alpha waves (frequency 8-12 Hz, amplitude 50 μv) are registered in the occipital and parietal regions.

These waves rise and fall spontaneously and usually disappear quickly when the subject opens his eyes or focuses his attention on something. Faster waves with a frequency higher than 13 Hz and less high amplitude (10-20 μV), called beta waves, are determined symmetrically in the frontal regions. Very slow waves (delta waves), sharp waves and other unusual fluctuations are absent in a normal recording. When healthy subjects fall asleep, the oscillation rhythm is symmetrically expressed and characteristic waves appear (sharp waves and sleep spindles in the parietal regions); if the sleep is caused by barbiturates and benzodiazepines, then an increase in high-frequency activity is determined, which is considered normal (see ch. 20). In case of excessive high-frequency activity, it should be clarified whether the patient is taking any drugs of these groups.

During stroboscopic stimulation, a normal EEG may show a reaction of the occipital cortex to light flashes, called an evoked reaction or, at a higher rate of their repetition, a reaction of learning the rhythm of photostimulation. The clinical significance of the evoked response of the occipital cortex lies in the fact that it expands the diagnostic capabilities of EEG in several ways: 1) it gives the researcher logical confidence that the subject at least perceives light, 2) if the evoked response is absent in one hemisphere, but is determined in the other, then this serves as a physiological confirmation of the presence of a lesion that prevents the normal conduction of impulses from the optic tubercle to the occipital lobe, which is localized on the same side, 3) if light flashes cause pathological waves, then this indicates increased excitability. If the EEG activation procedure is continued, true convulsive discharges may be provoked ("photoparoxysmal" reaction); in the case of even higher sensitivity, pronounced myoclonic twitching of the muscles of the face and upper limbs, generalized convulsions may appear. These phenomena should be distinguished from a purely muscular reaction, also of a myoclonic nature, which is normally caused by the contracting muscles of the scalp and is often visible during routine EEG (photomyoclonus).

Types of pathological records. The most pronounced disturbance is the disappearance of the EEG curve and its replacement by "bioelectric silence of the brain", which means that the electrical activity of the cerebral cortex recorded from the scalp is less than 2 μV or absent. A similar isoelectric EEG can be induced by anesthetic doses of drugs such as barbiturates and by profound hypothermia (less than 70°F). However, in the absence of CNS depressant drugs or deep hypothermia, the "flat" type of recording (except for artifacts) in all leads is almost always the result of cerebral hypoxia, ischemia, or widespread cortical destruction. It is believed that if the patient does not have EEG activity, reflexes, spontaneous breathing and any muscle activity for 6 hours or more, one should talk about "irreversible coma". Such patients have extensive brain necrosis. It is impossible to restore the function of the central nervous system, in connection with which the patient can be considered dead, despite the preservation of vegetative (cardiovascular) functions, supported by mechanical devices, for example, respirators. None of the more than 900 patients examined over the past 18 years in a multidisciplinary clinic in Massachusetts was an exception.

Sometimes local areas of absence of EEG activity are observed in cases of large heart attacks, massive superficial brain tumors or in the presence of clots located between the cortex of the large hemisphere and the electrodes. These changes make it possible to precisely establish the localization of the pathological focus, but, of course, it is impossible to judge the nature of the process by them. In most cases, the sizes of the pathological foci are too small to be directly registered with the help of EEG, but the obtained curve can record pathological waves originating mainly from functionally rather than organically changed areas of the brain around the affected area. These pathological waves are slower and higher in amplitude (50-350 μV) than normal ones. Waves with a frequency of less than 4 Hz are called delta waves, from 4 to 7 Hz - theta waves; higher voltage and fast waves are designated as spikes or sharp waves. Sometimes fast and slow waves are observed simultaneously; when series of such waves suddenly, in the form of paroxysms, interrupt a relatively normal EEG recording, there is every reason to suspect epilepsy. Patients with petit mal (absence) seizures are characterized by the appearance of "peak-wave" complexes with a frequency of 3 Hz in all EEG leads at the same time, followed by their sudden

disappearance at the end of the attack.

Neurological conditions with pathological EEG. In the following forms of neurological diseases, EEG is essential for making the correct diagnosis.

Epilepsy. All types of generalized epileptic seizures (major and minor) are accompanied by certain periodically recorded electroencephalographic changes. Pathological changes in the EEG are often detected also in more limited types of epileptic seizures (complex partial, myoclonic, focal and Jacksonian). The only exception is some deep foci in the temporal lobe, the amplitude of discharges from which when approaching the scalp is insufficient to manifest against the background of physiological EEG activity, especially in the presence of a pronounced alpha rhythm. It is possible to determine the localization of an epileptic focus in the medial part of the temporal lobe with the help of the zygomatic and sphenoidal leads; the changes revealed at the same time are sometimes the only confirmation of epileptic activity during EEG. Among other exceptions, in which it is sometimes not possible to register EEG changes during a seizure, focal seizures (sensory, Jacksonian, myoclonic) are included. Probably, this fact means that the neuronal discharge is very deep in localization, discrete, short and asynchronous to be transmitted by volumetric conduction through the skull and recording by EEG electrodes located at a distance of about 2 cm from the cortex of the large hemispheres. Electroencephalographic manifestations of small, some myoclonic and large seizures are closely correlated with the clinical forms of paroxysms and can be detected in the period between attacks. Artifacts due to motor activity during a “seizure” are usually indistinguishable from electrical brain activity. Differentiating psychogenic seizures from true episeizures requires careful EEG analysis at the beginning of the seizure, when characteristic high-frequency activity can be detected, or immediately after the cessation of convulsive movements, when post-seizure slowing and suppression of waves must be determined in the case of a disordered state of consciousness. A normal alpha rhythm in the post-seizure period in an "unresponsive" patient suggests a psychogenic pseudoepileptic attack.

It is important to note that 20% of patients with minor and 40% of patients with generalized epilepsy have a normal EEG between attacks. When treated with anticonvulsants, there is also a tendency to decrease the severity of EEG changes. In the other 30-40% of patients with epilepsy in the periods between attacks, EEG abnormalities are detected, but they are of a non-specific nature, therefore, the diagnosis of epilepsy should be made only on the basis of accurate interpretation of clinical data in combination with EEG changes.

Tumor, brain abscess and subdural hematoma. Approximately 90% of patients with clinically manifest intracranial volume formations also have EEG changes. These changes depend on the localization and form of the pathological process. In addition to diffuse disorders, focal and localized slow waves (usually of the delta range) are considered classical in such cases, sometimes epileptic activity with a decrease in amplitude and synchronization of a normal rhythm is determined. The highest frequency of changes in EEG is observed in more rapidly increasing in size, bulky formations (abscess, some metastases, glioblastoma), especially in the case of their supratentorial localization (in 90-95% of cases in the last two, 100% - in abscesses). Slower-growing tumors (astrocytomas) and primarily neoplasms located outside the large hemispheres of the brain (meningiomas, pituitary tumors) often do not show EEG abnormalities, despite the presence of clear clinical manifestations. Electroencephalographic changes have a clear lateralization in 75-90% of patients with supratentorial tumors and abscesses, focal EEG abnormalities in brain metastasis can be detected when the metastasis is not yet visible on CT. Normal EEG results, as well as CT scan, practically rule out supratentorial tumors or brain abscess. Meanwhile, the EEG is normal in 20-25% of patients with subtentorial tumors.

Vascular diseases of the brain. Both diffuse and local changes on the EEG caused by vascular lesions of the brain (heart attacks and intracranial hemorrhages) mainly depend on their localization and size, and not on the nature of the pathological process. Shown. that EEG should be performed for the differential diagnosis of vascular hemiplegia. When the lesion is localized in the basin of the internal carotid artery, the area of reduced normal activity or excessive slow-wave impulses is almost always clearly defined in the corresponding area. If hemiplegia is caused by a

lesion of a small-caliber vessel, that is, a lacunar infarction localized in the deep parts of the hemispheres or the brain stem, then the EEG pattern is usually normal. Extensive hemispheric foci, in which a sharp depression of consciousness is noted, also cause the appearance of widespread, diffuse slow-wave activity of a nonspecific nature, the same as that determined in stupor or coma of any etiology. Changes begin after a few days, when brain swelling decreases, and focal activity (slow-wave activity or inhibition of physiological rhythms) may be noted. Infarcts of smaller sizes are accompanied by pronounced focal changes, which clearly indicate the lateralization of the focus, but do not allow its exact localization. Unlike tumors, the improvement continues, and after 3-6 months, in almost 50% of patients with acute cerebrovascular pathology, the EEG normalizes, despite the presence of a persistent neurological defect. In a similar situation, the prognosis is unfavorable. The subsequent detection of moderate or high-amplitude waves on the EEG, and especially spikes or sharp waves, indicates the presence of pathologically functioning tissue that may have epileptogenic properties. With an acute subarachnoid hemorrhage, the EEG can provide useful information about the side of its localization, while the severity of the changes depends on the degree of impact on the underlying brain tissue.

Brain injury. In the event of a stroke or rupture of the brain, changes similar to those in cerebrovascular pathology appear on the EEG. Diffuse changes often give way to focal changes, especially when lesions are localized on the lateral or upper side of the cerebral hemispheres. If there is no epileptic activity, the focal changes usually disappear after several weeks or months. Sharp waves or spikes sometimes appear after the disappearance of focal slow-wave changes. These disturbances or lack of EEG normalization usually precede the development of post-traumatic epilepsy. Therefore, performing an EEG in dynamics after a traumatic brain injury is valuable for determining the prognosis of epilepsy.

Diseases that cause coma and disorders of consciousness. In almost all conditions accompanied by some disturbances of consciousness, the EEG has a pathological character. In hypothyroidism, the rhythms are of a normal configuration, but are usually slow. In general, the deeper the disorder of consciousness, the more the EEG picture is changed. With pronounced disturbances of consciousness, bilateral, high-amplitude slow (delta) waves are detected, which are more noticeable in the recording from the frontal regions. This applies to such different conditions as acute meningitis and encephalitis, sudden changes in gas composition, blood glucose content, water-electrolyte balance, uremia, diabetic and hepatic coma, and loss of consciousness in massive brain lesions. With hepatic coma, the severity of changes on the EEG corresponds to the stages of confusion, stupor or coma. In addition, paroxysms of bilateral synchronous large sharp "triphasic waves" are characteristic, although they can also be present in other metabolic encephalopathies against the background of renal and pulmonary insufficiency. Diffuse degenerative lesions of the cerebral cortex (for example, in Alzheimer's disease) are accompanied by relatively weak diffuse slow waves of the theta range (4-7 Hz). In more rapidly progressing diseases, such as subacute sclerosing panencephalitis (PSPE), Creutzfeldt-Jakob disease and, to a lesser extent, cerebral lipidoses, very characteristic, almost pathognomonic EEG changes are also observed, consisting of repeated complex bursts of activity in the form of acute and slow waves. A normal EEG in a patient with apathy, lethargy, depression or memory loss is one of the evidences in favor of the diagnosis of affective disorder or schizophrenia.

EEG can also help the doctor manage a comatose patient. According to the results of the EEG, one can suspect such causes of coma as hepatic encephalopathy (bilateral synchronous triphasic waves), intoxication with barbiturates or benzodiazepines (excessive rapid activity), clinically prolonged epileptic discharges occupying a large volume of the lesion, diffuse anoxia-ischemia (a sign of " flash-suppression "with repeated generalized complexes separated by periods with a very low amplitude of the EEG curve).

Other brain lesions. Many disorders of the functions of the nervous system cause only weak changes on the EEG or are not accompanied by changes at all. Similar examples are multiple sclerosis and other demyelinating diseases, although in their advanced stages the EEG is pathological in 50% of patients. Alcoholic delirium, disease. Wernicke-Korsakov and withdrawal

syndrome seizures are accompanied by minor changes in the EEG, despite the dramatic nature of the clinical picture, and sometimes the EEG may not be changed at all. Some slow-wave activity usually accompanies a twilight state, which is referred to as hypokinetic delirium. It should be noted that neuroses and psychoses, such as manic-depressive disorders and schizophrenia, pathological conditions caused by hallucinogenic drugs such as LSD, and most cases of mental retardation are accompanied by minor or nonspecific changes.

Special application of EEG. Since the EEG provides information about the state and functioning of the brain, it is appropriate to use it for monitoring in the operating room when monitoring the viability of the brain during modern cardiac surgical interventions, which are becoming more and more extensive. For a long time, electroencephalographic equipment was used to determine the level of anesthesia. The use of simple equipment by anesthesiologists for monitoring the functions of the heart and brain in patients during surgical operations has not lost its importance even today.

Continuous EEG monitoring during carotid endarterectomy is a common practice these days - a manipulation performed on carefully selected patients suffering from stenotic or ulcerative lesions of the carotid artery. Characteristic changes in the EEG (especially a significant decrease in voltage) indicate the need for temporary anastomotic shunting to maintain sufficient cerebral blood flow to eliminate ischemic damage to the brain during surgery.

In neurosurgical operating rooms, EEGs can be recorded from the bare brain (electrocorticograms), while the focus of epileptic activity is more accurately localized than when recording from the scalp, which makes resection of functionally altered tissue possible.

Routine EEG is of diagnostic value in hysterical blindness. Similarly, a response to noise during daytime sleep can help confirm the presence of auditory perception in a patient simulating total deafness. Examining these responses may also be useful for evaluating hearing and vision in newborns.

Evoked potentials

The method of evoked potentials (EP) is a method of recording the electrical activity of groups of neurons in the spinal cord, brainstem, optic tubercle, and large hemispheres after stimulation of one or another afferent system by visual, auditory, or tactile influences. The amplitude of these potentials, recorded from the scalp using conventional electroencephalographic electrodes, varies from 0.5 and less to 20 μv. Due to their extremely small size, they can rarely be registered on an electroencephalography with ink sand against the background of the main electrical activity of the brain, which usually reaches an amplitude of 50 μV or more. Therefore, special equipment, including simple computers, is needed to extract from the main EEG curve the wave-like response that is of interest to the researcher. This technique is called "averaging" because the procedure involves repeating 100-1000 precisely timed stimuli and recording the electrical activity in a certain short interval after each stimulus. Further spontaneous activity on the EEG at each given moment of time after the stimulus can be negative, and at other moments - positive in its polarity, tends to be extinguished with a sufficient number of repetitions. On the other hand, the evoked response has stable temporal characteristics in relation to the stimulus and at a specific moment after the stimulus always appears as an electrical signal of the same shape. In this connection, the VP increases as repetitions occur, while the amplitude of the background curve decreases. It is important to use special amplifiers, to place the electrodes on the surface of the scalp with great accuracy, to give stimuli precisely on time and to minimize accompanying electrical artifacts. VP expand the possibilities of clinical neurological research of the corresponding afferent system, make it more sensitive and objective, but are not a more specific method of etiological diagnosis.

Visual evoked potentials. Visual evoked potentials obtained with the help of pattern alternation (VEP) have the longest history of clinical use. In the course of the study, patients are shown a reversible black-and-white checkerboard pattern projected on the screen. When the patient examines the alternating pattern, a characteristic wave-like impulse is formed in him, which can be registered from the scalp of the back of the head. Normally, this three-phase wave has a

distinctive positive peak with a latency period of 95-115 ms (usually denoted as P100; Fig. 341-5) from the moment of pattern reversal. The duration of this latent period, the duration of the response and the peak amplitude are measured . The latent period is the most important clinical parameter. Research is carried out separately for each eye. Purely monocular changes indicate the presence of a conduction defect below the chiasm.

Many forms of lesions of the optic nerve fibers with changes in the ChPZVP. Glaucoma, compression of the optic nerve, chiasm or tract by various volumetric formations, degenerative lesions of the optic tract often cause a decrease in the amplitude and/or an increase in the latency of the response. With a significant damage to the visual system, it is not possible to register reactions to stimulation of one or both eyes. In clinical practice, optic neuritis, often associated with multiple sclerosis, is the most common cause of changes in the CSFV. With demyelination of optic nerve fibers, both in primary demyelinating disease and in the pathological processes listed above, a slowing of conduction along nerve fibers and an increase in the latency of the positive peak of ChPZVP (up to 115-200 ms) are detected. Indeed, almost all patients with optic neuritis, even after the restoration of visual acuity to normal, have characteristic changes in the LVEF, while no abnormalities are detected during a detailed ophthalmological examination. If in patients with multiple sclerosis, the LVEF is within the normal range, then no abnormalities are detected during neuro-ophthalmological examinations. When LVEF is pathological, a significant number of patients have no abnormalities in visual fields, visual acuity, pupillary reactions, and fundus.

Approximately 50% of patients with multiple sclerosis, in whom the visual function has never been impaired, also find abnormalities on the part of ChPZVP, which indicates the high efficiency of this method in this disease. If the patient consults a doctor about the first episode of a neurological disease, in which the focus of the lesion is localized in the brain stem or spinal cord, then in the case of the detection of the pathological nature of the CPZVP or other does not clinically manifest the focus of the lesion in another department of the CNS (optic nerves), the diagnosis becomes more likely multiple sclerosis, which frees the patient from the need for a number of neuroradiological examinations.

Changes in visual acuity are not reflected on the CHPZVP until vision deteriorates so much that the patient is unable to distinguish a checkerboard pattern; patients with visual acuity of 20/200 and above can undergo examination. The only additional requirement is the patient's sufficient ability to interact and remain calm in a sitting position for 20 minutes while he examines the pattern. Newborns and children can also undergo this examination, but at the same time special methods are used.

Stem auditory evoked potentials. Trunk auditory evoked potentials (AEPs) are more difficult to study than AEPs, as they are much lower in amplitude, within 0.5 μV. They are caused by sound clicks that stimulate one ear through an earpiece. The patient can be both asleep and in a comatose state. It should be noted only that excess movements and muscle artifacts make it difficult to get an answer.

Somatosensory evoked potentials. Somatosensory evoked potentials (SEPs) are evoked by small, painless electrical stimuli directed at large sensory fibers of mixed nerves of the upper and lower extremities. The afferent impulse is registered at many levels as it travels towards the center; series of waves can be recorded that reflect activity in the trunks of peripheral nerves that conduct pathways of the spinal cord, the nuclei of the thin and sphenoid fascicles, the structures of the pons and / or cerebellum, as well as the optic tubercle, thalamocortical pathways, and the primary sensory fields of the cortex of the large hemispheres (see Fig. 341-5). Damage to these conductors at any level leads to changes in subsequent waves, which allows to establish or confirm the localization of the pathological process, similar to the way it is done in the study of SSVP.

Angiography is a valuable method that provides an X-ray image of brain vessels after the introduction of an X-ray contrast agent into them. is carried out with the aim of clarifying the localization of the pathological focus, clarifying its nature and character.

Brief information about the methods, the principles on which they are based and the main indications for use are given below

Electromyography

Principles of the method: Assessment of the functional state of nerves and muscles. Determination of the electrical activity of muscles, lesions of the neuromotor apparatus (primary muscular, neural, anterior horn lesions, peripheral nerve lesions)

Indications for use: Hereditary myopathy, myotonia, radiculoneuritis, extrapyramidal hyperkinesis, mono-, polyneuritis, axonal, demyelinating neuropathies

Evoked potentials

Principles of the method: Stimulation of receptors of the sensory system (visual, auditory, somato-sensory) causes an electrical signal in the corresponding area of the cortex

Indications for use: Retrobulbar neuritis, multiple sclerosis, brainstem glioma, brainstem infarctions, spinal cord tumors, myelopathy

Electroencephalography

Principles of the method: The method of recording spontaneous electrical activity of the brain using electrodes attached to the surface of the head. Determination of the functional activity of the brain, localization of the pathological focus in the brain, monitoring of the pathological process

Indications for use: Epilepsy, tumors, vascular, inflammatory, degenerative diseases of the brain, comatose states

Echoencephalography

Principles of the method: The method of echolocation inside the cranial space, which is based on the property of ultrasound to be reflected at the boundary of the environment endowed with different physical properties (skull bones, blood, cerebrospinal fluid, brain matter)

Indications for use: Tumors, abscess, edema, brain hematoma

Ultrasound dopplerography

Principles of the method: Non-invasive study of extracranial carotid and spinal vessels, as well as determination of the linear speed of blood flow and its direction. Study of the difference in the frequency of reflected ultrasound waves, which depends on the speed of blood circulation - the Doppler effect

Indications for use: Atherosclerosis, nonspecific aortoarteritis, deformation and stenosis of an aneurysm of extracranial vessels, angiodysplasia, extravasal compression of arteries

Craniography

Principles of the method Determination of the state of the skull as a whole: base of the skull, Turkish saddle, cranial sutures, facial skeleton

Indications for use: Craniocerebral trauma, voluminous intracranial neoplasms, hyperostosis of skull bones, neurinoma VIII and optic nerve, pituitary neoplasm

Brain angiography

Principles of the method Invasive method of imaging vessels using a series of craniograms during intra-arterial injection of a radiopaque substance

Indications for use: Congenital anomalies of vessels, arterial aneurysms, tumors, hematomas, abscesses, parasitic cysts

Spondylography

Principles of the method Determination of the walls and lumen of the spinal canal, intervertebral openings, shape, structure, contours of the vertebral bodies, their arches, spinous and transverse processes, as well as the condition of the intervertebral discs and joints

Indications for use: Sacralization of the V lumbar or lumbago of the I coccygeal vertebrae, spondylopathy, compression fracture of the vertebral body, tumors, spondylolisthesis, ankylosing spondylitis

Myelography

Principles of the method Contrast X-ray examination of the spinal cord (introduction of iodine-containing solutions through a lumbar puncture into the spinal canal)

Indications for use: Compression of the spinal cord or roots (intramedullary tumors, disc herniation, abscess, cyst)

Computed tomography

Principles of the method The use of X-rays, with the help of which we obtain sections of the brain and spinal cord in the axillary (horizontal) plane at different levels with subsequent processing on a computer

Indications for use: Hematomas, meningiomas, cysticerci, foci of calcification, ischemia, glial and metastatic tumors, cysts, herniated discs, hydrocephalus, brain atrophy, pathology of skull bones, vertebrae

Magnetic resonance imaging

Principles of the method: Use of nuclear magnetic resonance for a more detailed image of the brain and spinal cord in axial, frontal, sagittal projection in T1 and T2 modes

Indications for use: Multiple sclerosis, demyelinating diseases, pituitary adenoma, congenital brain anomalies, Arnold-Chiari anomalies, hydrocephalus, arteriovenous malformation, brain ischemia (in the first 48 hours), cerebral hemorrhage in the subacute period, dysplasias

Positron emission tomography

Principles of the method: Imaging of the brain (radioisotope diagnostics) using radiopharmaceuticals labeled with positron-emitting isotopes. Study of brain vital processes, including glucose metabolism and oxygen utilization, assessment of blood circulation and perfusion

Indications for use: Differentiation of malignant and benign tumors, Parkinson's disease, Huntington's disease.

Materials for self-control of training quality

Tests

1) Is the blood supply to the brain ensured?

Answer options:
a) by the system of the internal carotid artery and the artery of Adamkevich;
b) vertebro-basilar and anterior spinal artery;
c) internal carotid artery system and vertebro-basilar system.
Correct answer: c)

2) What electrophysiological methods are used to diagnose hydrocephalus syndrome?

Answer options:
a) EEG;
b) Echo-EG;
c) REG;
d) EMG
Correct answer: b)

3) What rhythms on the EEG are considered basic?

Answer options:
a) alpha rhythm;
b) beta rhythm;
c) delta rhythm;
d) gamma rhythm.
Correct answer: a, b)

4) What rhythms on the EEG are considered basic?

Answer options:
a) alpha rhythm;
b) beta rhythm;
c) delta rhythm;
d) gamma rhythm.
Correct answer: c)

5) What are the indications for prescribing REG?

Answer options:
a) cerebral atherosclerosis;
b) hydrocephalus syndrome;
c) stroke;
d) fluid dynamics disorders.
Correct answer: a, c)

6) What are the indications for the appointment of Echo-EG?

Answer options:
a) cerebral atherosclerosis;
b) hydrocephalus syndrome;
c) stroke;
d) fluid dynamics disorders.
Correct answer: b, d)

7) Is there a volume process of the brain on Echo-EG?

Answer options:
a) "M-ekha" dislocation;
b) hypertensive-hydrocephalic syndrome;
c) hypertensive CSF syndrome;
d) normotensive-CSF syndrome;
e) there will be no "M-ekha" dislocation
Correct answer: a, b)

Tasks for self-control :

1. The patient received a closed TBI, after some time convulsive attacks occurred. On the EEG, there is a focus of pathological activity (high-amplitude waves in the alpha range, pathological waves) in the left parietal-temporal zone. What syndrome has developed in the patient?

Correct answer: post-traumatic epileptic syndrome.

Materials for classroom self-training:

List of educational practical tasks:
1. Be able to read and decipher:
   a) recording of normal EEG;
   b) record of normal REG;
   c) recording of normal EMG;
   d) recording of normal ultrasound dopplerography.
2. Decipher echoencephaloscopy of a healthy person.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 14

Topic: Blood supply of the brain and spinal cord

The goal: to acquaint the applicants with the blood supply of the brain and spinal cord, to draw their attention to the importance of the normal functioning of the entire human vascular system for its full-fledged life.

Basic concepts: are the extreme importance of normal blood supply to the brain and spinal cord, in case of violation of which complex disorders of the nervous system occur in a person (paralysis, loss of sensitivity, speech, many practical and everyday skills, etc.), which often lead to severe and long-term disability . Therefore, timely diagnosis and adequate treatment of such patients have not only social, but also economic significance.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Blood supply to the brain

The brain is supplied with arterial blood from two pools: carotid and vertebrobasilar.

The carotid basin system in its initial section is represented by the common carotid arteries. The right common carotid artery is a branch of the brachiocephalic trunk, the left - directly departs from the aorta. At the level of the upper edge of the thyroid cartilage, the common carotid artery branches into the external and internal carotid arteries. Then, through the foramen caroticum, the internal carotid artery enters the canalis caroticum of the pyramid of the temporal bone. After the artery leaves the canal, it passes along the front side of the body of the pterygoid bone, enters the sinus cavernosus of the dura mater and reaches the place under the anterior perforated substance, where it divides into terminal branches. An important collateral branch of the internal carotid artery is the orbital artery. Branches depart from it that irrigate the eyeball, lacrimal gland, eyelids, skin of the forehead and partially - the walls of the nasal cavities. Terminal branches a. ophthalmica - suprablock and supraorbital anastomose with the branches of the external carotid artery. Then the artery lies in the Sylvian groove. The final branches of the internal carotid artery are represented by 4 arteries: the posterior connecting artery, which anastomoses with the posterior cerebral artery, which is a branch of the basilar artery; the anterior villous artery, which forms the vascular plexuses of the lateral cerebral ventricles and which plays a role in the production of cerebrospinal fluid and the blood supply of some nodes of the base of the brain; anterior cerebral artery and middle cerebral artery. The internal carotid artery is connected to the posterior cerebral artery by means of the posterior connecting arteries. The anterior cerebral arteries are connected to each other by means of the anterior connecting artery. Thanks to these anastomoses, the Willisian arterial circle - circulus arteriosus cerebry is formed at the base of the brain. The circle connects the arterial systems of the carotid and vertebrobasilar basins.

The anterior cerebral artery already within the circle of Willis gives off several small branches - anterior perforated arteries - aa. perforantes arteriores. They pierce the anterior perforated plate and feed part of the head of the caudate nucleus. The largest of them is the retrograde artery of Heibner, which supplies the anteromedial parts of the head of the caudate nucleus, the shell, and the anterior two-thirds of the anterior foot of the internal capsule. The most anterior cerebral artery lies above the corpus callosum and supplies arterial blood to the medial surface of the hemispheres from the frontal pole to the fissura parieto-occipitalis and the front two-thirds of the corpus callosum. Also, its branches can enter the orbital area of the base of the brain and the lateral surface of the frontal pole, the upper frontal gyrus, and the paracentral lobe.

The middle cerebral artery is the largest. It lies in the Sylvian sulcus and supplies blood to the entire convexity surface of the hemispheres (except for the areas irrigated by the anterior and posterior cerebral arteries) - the lower and middle frontal gyri, the anterior and posterior central gyri, the supramarginal and angular gyri, the insula, the outer surface of the temporal lobe, the anterior parts of the occipital destiny Within the circle of Willis, the middle cerebral artery gives off several thin branches that pierce the lateral parts of the anterior perforated plate, the so-called aa. perforantes mediales et laterales. The largest of the perforating arteries are aa. lenticulo-striatae and lenticulo-opticae. They supply blood to the subcortical nodes of the hemispheres, the fence, the posterior third of the anterior leg, and the upper part of the posterior leg of the internal capsule.

The vertebrobasilar pool in its proximal section is represented by vertebral arteries, which depart from the subclavian arteries at the level of the transverse process of the VI cervical vertebra

(segment V1). Here it enters the opening of its transverse process and rises up the channel of the transverse processes to the level of the II cervical vertebra (segment V2). Then the vertebral artery returns to the back, goes to the for. transversarium of the atlas (segment V3), passes through it and lies in sulcus a. vertebralis. In the extracranial section, the artery gives off branches to the muscles, bone and ligament apparatus of the cervical spine, and takes part in feeding the meninges.

The intracranial section of the vertebral artery is segment V4. In this department, branches depart to the dura mater of the posterior cranial fossa, the posterior and anterior spinal arteries, the posterior lower cerebellar artery, and the paramedian artery.

Posterior spinal artery - paired. It is located in the posterior lateral groove of the spinal cord and participates in the blood supply of the nuclei and fibers of the thin and sphenoid bundles. Anterior spinal artery - an unpaired one formed as a result of the fusion of two trunks departing from the vertebral arteries. It supplies blood to the pyramids, the medial loop, the medial longitudinal bundle, the nuclei of the hypoglossal nerve and the solitary tract, as well as the dorsal nucleus of the vagus nerve. The posterior inferior cerebellar artery is the largest branch of the vertebral artery, it supplies blood to the medulla oblongata and the lower parts of the cerebellum. The paramedian branch provides blood supply to the ventral and lateral parts of the medulla oblongata and the roots of IX-XII pairs of cranial nerves.

At the posterior edge of the Varoliev bridge, both vertebral arteries merge, forming the main artery - a. basilaris. It lies in the furrow of the bridge and on the slope of the occipital and sphenoid bones. Paramedian branches, short circumflex, long circumflex (paired - lower anterior cerebellar and upper cerebellar arteries) and posterior cerebral arteries depart from it. The largest of them are the lower anterior cerebellar, upper cerebellar and posterior cerebral arteries.

The lower anterior cerebellar artery departs from the main one at the level of its middle third and supplies blood to the cerebellar lobe and its anteroinferior surface.

The superior cerebellum departs from the upper part of the main artery and provides blood supply to the upper half of the cerebellar hemispheres, the cerebellum, and partially the tetrahedron.

The posterior cerebral artery is formed as a result of the division of the main artery. It nourishes the roof of the midbrain, the brain stem, the thalamus, the lower internal parts of the temporal lobe, the occipital lobe and partially the upper parietal lobe, gives small branches to the vascular plexus of the third and lateral ventricles of the brain.

There are anastomoses between the arterial systems, which begin to function when one arterial trunk is occluded. There are three levels of collateral circulation: extracranial, extra-intracranial, intracranial.

The extracranial level of collateral circulation is provided by the following anastomoses.

When the subclavian artery is occluded, the blood flow is carried out:

from the contralateral subclavian artery through the vertebral arteries;

from the homolateral vertebral artery through the deep and ascending arteries of the neck;

from the contralateral subclavian artery through the internal thoracic arteries;

from the external carotid artery through the upper and lower thyroid arteries.

When the initial section of the vertebral artery is occluded, the flow is carried out from the external carotid artery through the occipital artery and the muscular branches of the vertebral artery.

Extra-intracranial collateral circulation is carried out between the external and internal carotid arteries through the supraorbital anastomosis. Here, the supralobular and supraocular arteries from the system of the internal carotid artery and the terminal branches of the facial and superficial temporal arteries from the system of the external carotid artery join.

At the intracranial level, collateral blood circulation is carried out through the vessels of the circle of Willis. In addition, there is a cortical anastomotic system. It consists of anastomoses on the convex surface of the hemispheres. The terminal branches of the anterior, middle, and posterior cerebral arteries are anastomosed (in the area of the upper frontal sulcus, at the border of the upper and middle thirds of the central gyri, along the interparietal sulcus, in the area of the upper

occipital, lower and middle temporal, in the area of the wedge, precuneus, and ridge of the corpus callosum) . Perpendicular branches depart from the anastomotic network under the soft meninges deep into the gray and white matter of the brain. They form anastomoses in the area of the basal nuclei.

The venous system of the brain takes an active part in blood circulation and liquid circulation. Brain veins are divided into superficial and deep. Superficial veins lie in the cells of the subarachnoid space, anastomose and form a looped network on the surface of each of the hemispheres. They have venous blood draining from the cortex and white matter. The outflow of blood from the veins goes to the nearest cerebral sinus. Blood from the external and medial parts of the frontal, central and parietal-occipital regions flows mainly into the upper sagittal sinus, to a lesser extent - into the transverse, straight, cavernous and parietal-basic sinuses. In the deep veins of the brain, blood flow comes from the veins of the vascular plexus of the lateral ventricles, subcortical nodes, optic tuberosity, midbrain, pons, medulla oblongata, and cerebellum. The main collector of this system is the great vein of Galen, which flows into the direct sinus under the tent of the cerebellum. Blood from the upper sagittal and straight sinuses enters the transverse and sigmoid sinuses and is drained into the internal jugular vein.

Blood supply of the spinal cord

The beginning of the study of the blood supply of the spinal cord dates back to 1664, when the English doctor and anatomist T. Willis pointed out the existence of the anterior spinal artery.

Three arterial basins of the spinal cord are distinguished behind the dlinnik - cervicothoracic, thoracic and lower (lumbar-thoracic):

The cervicothoracic basin provides blood supply to the brain at the level of C1-D3. At the same time, the vascularization of the uppermost parts of the spinal cord (at the level of C1-C3) is carried out by one anterior and two posterior spinal arteries departing from the vertebral artery in the skull cavity. The blood supply to the rest of the spinal cord comes from the system of segmental radiculomedullary arteries. At the middle, lower cervical, and upper thoracic levels, the radiculomedullary arteries are branches of the extracranial vertebral and cervical arteries.

In the thoracic basin there is the following pattern of formation of radiculomedullary arteries. Intercostal arteries depart from the aorta, giving off dorsal branches, which in turn are divided into musculocutaneous and spinal branches. The spinal branch enters the spinal canal through the intervertebral foramen, where it divides into the anterior and posterior radiculomedullary arteries. The anterior radiculomedullary arteries merge to form one anterior spinal artery. The two posterior spinal arteries form the posterior ones.

In the lumbothoracic region, dorsal branches depart from the lumbar arteries, lateral sacral arteries, and iliac-lumbar arteries.

Thus, the anterior and posterior lumbar arteries represent a set of terminal branches of the radiculomedullary arteries. At the same time, along the course of the blood flow, there are zones with the opposite blood flow (at the points of branching and junction).

Zones of critical blood circulation are identified, where spinal ischemic strokes are possible. These are the junction zones of vascular basins - C_{IV}, D_{IV}, D_{XI}-L_{I}.

In addition to the spinal cord, radiculomedullary arteries supply blood to the membranes of the spinal cord, spinal roots, and spinal ganglia.

The number of radiculomedullary arteries varies from 6 to 28. At the same time, there are fewer anterior radiculomedullary arteries than posterior ones. Most often, there are 3 arteries in the neck, 2-3 in the upper and middle thoracic, 1-3 in the lower thoracic and lumbar.

The following large radiculomedullary arteries are distinguished:

1. Artery of cervical thickening.

2. Large anterior radiculomedullary artery of Adamkevich. Enters the spinal canal at the D_{VIII}-$D_{XII\ level}$.

3. Lower radiculomedullary artery of Deprog-Gotteron (exists in 15% of people). Enters at the level of L_{V}-S_{I}.

4. Upper additional radiculomedullary artery at the level of D_{II}-D_{IV}. Occurs with the trunk

type of blood supply.

Three arterial basins of blood supply to the spinal cord are distinguished by their cross section:

1. The central zone includes the anterior horns, the periependymal gelatinous substance, the lateral horn, the base of the posterior horn, Clark's columns, the deep divisions of the anterior and lateral columns of the spinal cord, and the ventral part of the posterior cords. This zone is 4/5 of the entire diameter of the spinal cord. Here, the blood supply comes from the anterior spinal arteries at the expense of the furrow-submerged arteries. There are two of them on each side.

2. The posterior arterial zone includes the posterior columns, the tops of the posterior horns, the posterior sections of the lateral columns. Here the blood supply comes from the posterior spinal arteries.

3. Peripheral arterial zone. Blood supply here is carried out from the system of short and long encircling arteries of the perimedullary vascular network.

The venous system of the spinal cord has central and peripheral parts. The peripheral system collects venous blood from the peripheral parts of the gray and mainly peripheral white matter of the spinal cord. It drains into the venous system of the pial network, which forms the posterior spinal or posterior spinal veins. The central anterior zone collects blood from the anterior commissure, the medial and central part of the anterior horn, and the anterior cord. The posterior central venous system includes the posterior cords and posterior horns. Venous blood drains into the sulcus veins, and then into the anterior spinal vein, located in the anterior fissure of the spinal cord. Blood flows from the pial venous network through the anterior and posterior root veins. Root veins merge into a common trunk and drain into the intravertebral plexus or intervertebral vein. From these formations, venous blood flows into the system of superior and inferior vena cava.

Depending on the localization of brain ischemia, certain neurological disorders occur.

A stroke with occlusion of the internal carotid artery is usually manifested by contralateral paresis and hypesthesia of the hand, central paresis of the facial and hyoid nerves, and often homolateral monocular blindness of a transient nature (orbital artery ischemia).

A stroke in the basin of the middle cerebral artery causes contralateral hemiplegia, hemianesthesia, hemianopsia with gaze paresis, aphasia (with damage to the dominant hemisphere) or anosognosia (with damage to the subdominant hemisphere). With a stroke in the pool of individual branches of the middle cerebral artery, various syndromes may occur: contralateral hemiparesis with an emphasis in the hand or monoparesis of the hand in combination with central paresis of the facial and hyoid nerves, motor aphasia, sensory aphasia, etc.

A stroke in the basin of the anterior cerebral artery is manifested by contralateral hemiparesis with a predominance in the proximal parts of the arm and leg or monoparesis of the leg, sometimes in combination with urinary incontinence.

Strokes in the vertebrobasilar system are much less common than strokes in the carotid system. Posterior cerebral artery occlusion most often causes contralateral hemianopsia and/or hemianesthesia. Occlusion of the vertebral artery or inferior posterior cerebellar artery is usually accompanied by dizziness, nausea, vomiting, swallowing disorder, hoarseness of voice, nystagmus, facial numbness and cerebellar ataxia on the side of the lesion and numbness of the limbs on the opposite side (Wallenberg-Zakharchenko syndrome). Vertigo, nausea, vomiting, and ataxia often occur with cerebellar infarction.

Lacunar stroke is caused by damage to the penetrating arteries of the basal ganglia, the internal capsule or the pons of the brain and is most often manifested by the following syndromes: hemiparesis ("purely motor stroke"), hemianesthesia ("purely sensory stroke"), hemiparesis with predominance in the leg and ataxia ("atactic hemiparesis ") or slurred speech and mild ataxia in the hand ("dysarthria / clumsy hand" syndrome).

Violation of spinal blood circulation.

Damage to the upper vascular basin is accompanied by the development of Unterharnscheidt's syncopal vertebral syndrome: in patients with cervical osteochondrosis, during sharp turns of the head, an attack occurs with a sudden short-term (for 2-3 minutes) loss of

consciousness. There is also significant weakness in the limbs, muscle hypotonia. Symptoms regress after 3-5 minutes. In the interval between attacks, patients complain of a feeling of heaviness, dull pain and numbness in the neck and hands. The attack is a consequence of ischemia of the trunk part of the brain and cervical thickening of the spinal cord.

The second variant of transient disturbances in the upper spinal basin is known as the syndrome of a sudden fall - drop attacks. With a sharp turn of the head to the side or pronounced overextension in the cervical region (throwing the head back), sudden muscle weakness develops and the patient falls. The attack lasts from a few seconds to 1-2 hours. The patient does not lose consciousness. The syndrome occurs against the background of degenerative-dystrophic lesions of the cervical spine (osteochondrosis) and is caused by transient ischemia of segments of the cervical thickening of the spinal cord.

Transient disorders of spinal blood circulation in the lower arterial basin, in particular in the course of the Adamkevich artery, were called myelogenic intermittent claudication. It mostly occurs after physical exertion, long walking and is clinically manifested by weakness and numbness of the legs, sometimes imperative calls to urinate. After a short rest (5-10 minutes), these phenomena disappear.

Caudogenic intermittent claudication occurs in the case of transient ischemia in the basin of the Deproge-Hauteron artery. When walking, patients experience painful paresthesias in the form of tingling, numbness in the distal parts of the legs and the perineum. If the patient continues to walk and does not rest, leg weakness appears - horse tail syndrome. The cause of such disorders is osteochondrosis of the lumbar spine or congenital narrowing of the spinal canal. Transient disorders of spinal blood circulation are often repeated and are harbingers of the development of persistent ischemia of the spinal cord.

Ischemic spinal stroke (spinal cord infarction) develops acutely. The disease is often preceded by short-term weakness of the limbs, transient sensitivity disorders (numbness, heartburn, unpleasant sensations in the muscles), pain in the innervation zone of the cervical, lumbar or sacral roots, as well as dysfunction of the pelvic organs in the form of imperative urges to urinate or its delay. The clinical picture of a spinal stroke is polymorphic and depends on the localization and prevalence of the ischemia focus. Most often, a heart attack develops in areas of adjacent blood supply.

Preobrazhensky syndrome - damage to the anterior 2/3 of the diameter of the spinal cord - a combination of lower paraplegia and dissociated type paraanesthesia (preserved deep sensitivity) is observed with occlusion of the intramedullary branches of the anterior spinal artery.

The cervical level is characterized by tetraplegia, sensitivity disorders and dysfunction of the pelvic organs according to the central type.

Damage to the thoracic part of the spinal cord is manifested by central paralysis of the legs, conduction disorders of all types of sensitivity and pelvic disorders of the central type.

When the focus is localized at the level of the lumbar thickening, lethargic lower paraplegia occurs with impaired sensitivity in the legs, urinary disorders. However, regardless of the level, a spinal infarction is accompanied by gross trophic disorders, rapid development of bedsores in the area of the sacrum, buttocks, back and heels. Dysfunction of the pelvic organs requires bladder catheterization, which entails bladder infection and ascending pyelocystitis. Bedsores and pyelocystitis are the source of sepsis in such patients, which makes the prognosis for recovery and life doubtful.

Slowly progressive (chronic) ischemic disorders of spinal blood circulation are called dyscirculatory ischemic myelopathy, or vertebrogenic myelopathy. They are characterized by certain clinical syndromes:

- amyotrophic, which is accompanied by lethargic paresis of the hands and feet, myotrophies, and fibrillar twitching of the muscles. The disease resembles a chronic form of poliomyelitis or amyotrophic lateral sclerosis;
- spastic syndrome includes lower spastic paraparesis;
- the syndrome of syringomyelia and posterior columns occurs in case of a blood supply

disorder in the system of posterior spinal arteries.

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

Materials for self-control of training quality

Tests

1) Which of the vessels feeds the occipital part of the brain?

Answer options:
a) Adamkevich's artery;
b) anterior cerebral artery;
c) posterior cerebral artery;
d) basilar artery.
Correct answer: c)

2) Which of the vessels feeds the spinal cord?

Answer options:
a) anterior and posterior spinal arteries;
b) main artery;
c) middle artery;
d) anterior connecting artery.
Correct answer: a)

3) What feeds the midbrain?

Answer options:
a) anterior cerebral artery;
b) posterior cerebral artery;
c) middle cerebral artery;
d) Deprog-Hotteron artery.
Correct answer: b)

4) What nourishes the corpus callosum?

Answer options:
a) anterior cerebral artery;
b) posterior cerebral artery;
c) middle cerebral artery;
d) Deprog-Hotteron artery.
Correct answer: a)

5) What feeds the inner capsule?

Answer options:
a) anterior cerebral artery;
b) posterior cerebral artery;
c) middle cerebral artery;
d) main artery;
e) spinal arteries.
Correct answer: c)

6) Which of the vessels feeds the cerebellum?

Answer options:
a) anterior cerebral artery;
b) posterior cerebral artery;
c) middle cerebral artery;
d) main artery;
e) spinal arteries.
Correct answer: d)

Tasks for self-control :

Patient K., 27 years old, complains of weakness of the lower limbs, difficulty in walking, numbness of the legs, periodic urinary incontinence. He fell ill about a year ago, suddenly, after a fall. Objectively: cranial nerves without pathology. Reflexes on the hands are alive, the same. Abdominal reflexes - the upper ones are reduced, the middle and lower ones are absent. Knee and Achilles high, with widespread reflexogenic zones. Pathological reflexes of Babinski, Rossolimo, Bekhterev are positive. Muscle strength is significantly reduced. Muscle tone is increased in the lower limbs. The sensitivity is reduced from the Th_{8} - $Th_{9\ level}$. There is no pathology on the X-ray of the thoracic spine.

Question:

1) What level of the spinal cord is affected?
2) In the basin of which artery the damage occurred?

Right answer:

1) The lower thoracic part of the spinal cord.
2) Artery of Adamkevich.

Materials for classroom self-training:

List of educational practical tasks:

1. Master research methods:
   a) motor functions;
   b) sensitive functions;
   c) presence of aphasia;
   d) practice of the patient;
   e) presence of agraphia, alexia, acalculia, autotopognosia, astereognosia.
2. Diagnose the damaged vascular pool.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 15

Topic: Vascular diseases of the brain and spinal cord. Chronic disorders of cerebral circulation

Purpose: to acquaint applicants with the modern classification of cerebrovascular diseases, the frequency of these disorders, and the main provisions for their treatment.

Basic concepts: vascular diseases of the brain occupy a leading place among diseases of the nervous system and are a frequent cause of death, temporary or permanent disability, and are also acute conditions that require urgent help from a doctor of any specialty. Mortality from cerebrovascular diseases in the overall structure of mortality is about 14%.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

The following main clinical forms of cerebral blood circulation disorders are distinguished:
A. Initial manifestations of insufficient blood supply to the brain.
1. Initial manifestations of insufficient blood supply to the brain
2. Initial manifestations of insufficient blood supply to the spinal cord.

B. Acute disorders of cerebral circulation
1. Transient disorders of cerebral circulation:
a) transient ischemic attacks;
b) hypertensive cerebral crises;
2. Acute hypertensive encephalopathy.
3. Membranous hemorrhage:
a) subarachnoid (subarachnoid);
b) epi- and subdural
4. Hemorrhage in the brain:
a) parenchymatous;
b) parenchymal - subarachnoid;
c) ventricular
5. Brain infarction:
a) atherothrombotic;
b) cardioembolic;
c) hemodynamic;
d) hemorheological;
e) lacunar.
B. Slowly progressive disorders of cerebral circulation:
1. Dyscirculatory encephalopathy;
2. Dyscirculatory myelopathy.
D. Consequences of a previously suffered cerebral stroke.

Initial manifestations of insufficient blood supply to the brain.

The initial manifestations of insufficiency of blood supply to the brain (PPNKM) are separated into an independent form of cerebrovascular pathology within the framework of the classification of vascular diseases of the nervous system that exists in Ukraine. This is of fundamental importance, since a timely diagnosis can ensure the highest effectiveness of treatment and preventive measures.

Etiology. The initial manifestations of insufficient blood supply to the brain develop against the background of systemic vascular diseases: cerebral atherosclerosis, arterial hypertension, vegetative-vascular dystonia.

Pathogenesis. Neurological disorders in the form of subjective asthenic manifestations, vegetative-vascular paroxysms appear under the condition of already existing pathology of the cardiovascular system. There is increased reactivity of the vascular system with a tendency to regional and general dystonic reactions. In optimal conditions, the lack of blood supply to the brain is compensated, but this compensation is unreliable, and the mechanisms of self-regulation of cerebral blood circulation work in an unstable mode. Inadequate hemodynamic reactions occur, which is largely due to morphofunctional changes in vascular reflexogenic zones (sinocarotid, from the arch of the aorta), as well as dysfunction of the subcortical autonomic centers of the hypothalamic area and the medulla oblongata.

In the pathogenesis of PPNKM, changes in cardio-hemodynamics and disturbances of cardiac activity play a significant role. The presence of close cerebrocardial relationships in the early stages of the formation of vascular pathology of the brain has been proven. With the development of persistent arterial hypertension and atherosclerosis, functional cardiac and cerebral disorders transform into coronary heart disease (CHD) and brain.

A significant place in the pathogenesis of PPNKM belongs to dystonic changes in the vessels of the brain, angioparesis with slowing of blood flow and violation of venous outflow, which occurs under such a condition. The pathology of the main arterial head (hemodynamically significant stenosis of the internal carotid and vertebral arteries, as well as the vessels of the circle of Willis) is also important, which is confirmed by the data of ultrasound dopplerography. The development of clinical symptoms in such cases occurs against the background of permanent insufficiency of blood supply to the brain. One of the mechanisms of the initial manifestations of

insufficiency of blood supply to the brain can be an increase in the viscosity of whole blood, a violation of the rheological properties of blood and microcirculation. Violations of neuronal metabolism and the functional state of the brain are also important in the development of PPNKM.

Clinic. Depending on the semiotics, there are three variants of the clinical course of PPNKM:

- preclinical,
- clinical
- paroxysmal.

The preclinical (asymptomatic) course is characterized by blood pressure instability, other signs of vegetative-vascular dystonia: acrocyanosis, acrohyperhidrosis, persistent red dermographism, paleness or redness of the skin, tremors of the fingers, eyelids, and invigoration of tendon reflexes. Patients have no subjective complaints. Signs of insufficient blood supply to the brain are revealed only during functional tests. They are characterized by changes in the bioelectric activity of the brain, hypersynchronous and desynchronous types of EEG. An increase or decrease in the tone of the vascular wall, lability of pulse waves, interhemispheric asymmetry, and difficulty in venous outflow are observed on REG. Sinus arrhythmia, violation of the depolarization phase, displacement of the ST segment and T wave are often found on ECT. Changes in biochemical indicators are characterized by an increase in the content of cholesterol, triglycerides, and lipoproteins.

The clinical (permanent) variant of the course of PPNKM is manifested by subjective signs of insufficient blood supply to the brain. The most typical complaints of patients: headache, dizziness, noise in the head, memory disorders, reduced mental capacity, etc. The basis for making a diagnosis can only be a combination of the listed two or more subjective symptoms that have been observed for a relatively long time and are often repeated (at least once a week during the last 3 months). It is characteristic that these complaints increase during mental stress, especially in conditions of hypoxia or overfatigue, poor sleep. After rest, the patient's condition improves or completely normalizes. Focal neurological symptoms are not observed in such patients, but subcortical reflexes, abdominal asymmetry, and invigoration of tendon and periosteal reflexes may be detected.

Signs of general vascular disease are found in patients:

- coronary cardiosclerosis,
- hypertrophy of the left ventricle of the heart,
- changes in the vessels of the fundus (angiopathy),
- symptoms of atherosclerotic lesions in other areas.

The diagnosis is also confirmed by changes in brain biopotentials, rheoencephalographic, biochemical indicators.

The paroxysmal course of PPNKM occurs mainly in patients with vegetative-vascular dystonia, arterial hypertension, and much less often - in the case of atherosclerosis of vessels. Vegetative-vascular paroxysms of the cephalic, vestibular, syncopal, sympathetic-adrenal, vago-insular and mixed type are dominant in the clinical picture. Paroxysmal disorders in most cases are a manifestation of dysfunction of the suprasegmental level of the autonomic nervous system, in particular, formations of the limbic-reticular complex.

The diagnosis of PPNKM is made on the basis of complaints, the clinical picture of the disease, as well as based on the data of electrophysiological and laboratory research methods. It is important to identify symptoms of general vascular disease: atherosclerosis, arterial hypertension, vegetative-vascular dystonia.

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 staged treatment of patients has been developed: polyclinic — hospital — resort — polyclinic, which includes dispensary supervision. At each of the stages, modern schemes of examination and treatment of patients are used, taking into account the variant of the clinical course, as well as the general vascular disease.

In the preclinical stage, if signs of vegetative-vascular lability are detected, a set of

preventive measures aimed at eliminating risk factors, stabilizing blood pressure in the presence of hypertension, and compensating the initial manifestations of atherosclerosis are used. Measures aimed at improving the working and living conditions of patients are of great importance. It is important to observe the diet: food rich in animal fats, cholesterol, and salt should be avoided; it is necessary to consume dairy-vegetable food, fish, boiled meat. Patients need to rest twice a year, sanatorium-resort treatment is recommended for them.

In the stage of clinical manifestations of the disease, outpatient or inpatient treatment is carried out taking into account the vascular pathology against which PPNKM developed. In the presence of neurosis-like complaints in the case of autonomic dystonia syndrome, sedatives (bromine, valerian), intermittent courses of treatment with tranquilizers in small doses, as well as belataminal, diphenhydramine, and calcium preparations are recommended. In case of arterial hypertension, anaprilin, obzidan, etc. are prescribed. If blood pressure is low, caffeine, tinctures of ginseng, Chinese lemongrass, pantocrine, levzei are recommended. Various methods of reflexology and electrosleep are used. Lipotropic agents play an important role in the treatment of atherosclerosis. In case of hemorheological changes, aspirin, curantyl, trental, sermion, plavix, agapurine (orally) are prescribed.

Drug treatment is also aimed at improving neuronal metabolism. For this purpose, nootropics are recommended (from the Greek "noo" - mind and "tropeo" - desire). These drugs include piracetam, encephabol, cerebrolysin, solcoseril, actovegin.

In the treatment of PPNKM, a significant place is occupied by physiotherapeutic measures, physical therapy and sanatorium-resort treatment, improvement of working and living conditions.

Treatment courses must be repeated twice a year. In case of arterial hypertension, maintenance doses of hypotensive agents are used to normalize blood pressure.

In the case of a paroxysmal course of PPNKM, α-adrenoblockers (piroxan), β-adrenoblockers (anaprilin, obsidan), antidepressants (amitriptyline, melipramine, ezopram, cipralex, miaser), and antlepsin (orally) are prescribed. From physiotherapeutic methods, the triad according to Graschenkov is used: intranasal electrophoresis of diphenhydramine, electrophoresis of areas of cervical sympathetic nodes and epigastric plexus.

Prevention is primary and secondary. Primary involves 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 prevention involves early detection and registration of patients with PPNKM, their timely treatment in order to prevent the progression of cerebrovascular insufficiency. During the treatment of patients, special attention should be paid to using the possibilities of day hospitals, as well as non-pharmacological methods of therapy. A large role in the prevention of PPNKM is played by medical examination of patients, planned neurological examinations.

Dyscirculatory encephalopathy is a set of progressive organic changes in brain tissue as a result of various cerebrovascular disorders.

Clinical picture of dyscirculatory encephalopathy

There are atherosclerotic, hypertensive, mixed (atherosclerosis and arterial hypertension), as well as venous encephalopathy.

Dyscirculatory atherosclerotic encephalopathy occurs most often. There are three stages of encephalopathy.

Stage I - moderately pronounced (compensated). It is manifested by cerebrasthenic syndrome (reduced memory, emotional instability, irritability, headaches, dizziness), dystonic vegetative-vascular reactions, various variants of psychopathological syndromes: asthenodepressive, asthenohypochondriac, paranoid, affective. There are no persistent focal neurological symptoms, but mild, transient, paroxysmal disorders are possible. In the initial stage of dyscirculatory encephalopathy, the hyperdynamic form of cerebrasthenia prevails, which manifests itself in disinhibition, excitability, incontinence, mood lability, and disturbed sleep.

Stage II - expressed (subcompensated). Structural organic changes increase, symptoms become not only more severe, but also more persistent. The headache is almost constant, persistent

noise in the head appears, psychopathological syndromes deepen, cerebrasthenia takes a hypodynamic form (lethargy, rapid fatigue, tearfulness, depressed mood, weakening of attention, reduced memory, sleep disorder). Diffuse cerebrovascular insufficiency leads to small focal lesions of the brain structures, while the corresponding clinical neurological syndrome is formed. The most common are pseudobulbar and amiostatic syndromes, as well as pyramidal insufficiency (dysarthria, dysphonia, reflexes of oral automatism, innervation disorders of the lower part of the face and language - deviation, slowness of movements, hypomimicness, stiffness, rhythmic tremor of the hands and head, dyscoordination, discomfort of movements in the fingers , visual impairment in the form of photopsia, hearing loss, epileptiform Jackson attacks, anisoreflexia, pathological reflexes of the flexor type).

Stage III - sharply expressed (decompensated). It is characterized by severe diffuse morphological changes in brain tissue, perivascular lacunae are formed, and granular atrophy of the cortex of the cerebral hemispheres may develop. On CT and MRI, areas of reduced density of white matter around the lateral ventricles and in the subcortical regions of the cerebral hemispheres (leukoareosis) are determined.

There is a deepening and complication of already existing symptoms, signs of damage to any area of the brain begin to prevail: cerebellar disorders, paresis, speech disorders, memory suffers sharply, vascular parkinsonism develops. Epileptiform attacks occur more often than in stage II. Severe mental changes often lead to deep dementia. Along with severe changes in the nervous system, the general somatic condition also suffers significantly. At this stage, acute disorders of cerebral blood circulation may develop.

Hypertensive dyscirculatory encephalopathy, unlike atherosclerotic encephalopathy, begins at a younger age, proceeds faster, especially against the background of cerebral hypertensive crises, new symptoms often appear acutely, during crises. Disinhibition, agitation, emotional instability, and euphoria come to the fore among mental changes.

Mixed dyscirculatory encephalopathy. It is characterized by a combination of features of atherosclerotic and hypertensive encephalopathy.

Venous encephalopathy is a special type of dyscirculatory encephalopathy and occurs in conditions that lead to a chronic violation of venous outflow from the cranial cavity (primary disorders of the regulation of venous tone - cerebral venous dystonia, pulmonary heart failure, craniostenosis, compression of extracranial and intracranial veins by pathological processes). Venous stasis with chronic edema develops in the substance of the brain. The clinical picture is dominated by the chronic-remitting hypertension syndrome: a dull, squeezing headache, which is aggravated by coughing, sneezing, straining, often - dizziness, lethargy, apathy, insomnia, frequent symptoms of diffuse small focal lesions of the brain, in severe cases - nausea, vomiting , congestion on the fundus, meningeal signs. Differential diagnosis of dyscirculatory encephalopathy.

In the initial stage, dyscirculatory encephalopathy should be differentiated from neuroses (functional disorders) and vegetative-vascular dystonia, in the later stages - from voluminous formations of the brain (tumor, cyst), parkinsonism of non-vascular genesis (postencephalitic, toxic, tumor, Parkinson's disease), from presenile and senile dementia, initial stages of Alzheimer's and Pick's disease, late epilepsy. Some variants of vascular dementia (Binswanger's disease) should also be differentiated from Schilder's periaxial encephalitis.

Treatment of encephalopathy. The following groups of drugs are used:

- Hypolipidemic and hypocholesterolemic drugs (clofibrate, polysponin, tribusponin, cetamifen, essentiale, phytin, polyunsaturated fatty acids, lipamide and methionine, etc.) are indicated for atherosclerotic encephalopathy.

- Hypotensive drugs (enap, clofelin, prestarium, atenolol, capoten, arifon, vincopan) are indicated for high blood pressure.

- Phlebotonic drugs (escusan, esflazid, troxevazin, anavenol, eufilin, redergin, cocarboxylase IV, glyvenol, caffeine) are indicated for venous dyscirculation. Angioprotectors (parmidine, anginin, etamzilate - indicated for high blood pressure, askorutin, vasobral).

- Disaggregants (curantyl, aspirin in small doses, trental, ticlid) are used to improve

microcirculation, prevent microthrombosis.

- Vasoactive drugs, including calcium antagonists (cavinton, vincamine, complamin, cinnarizine or stugeron, nifedipine, flunarizine)
- Nootropics (piracetam, encephabol or pyriditol, aminalon, picamilon, pantogam, cerebrolysin, glutamic acid, glycine, acefen).
- Metabolic drugs and antioxidants (vitamins B1, B6, ascorbic acid, retinol, aevit, lipoic acid, tocopherol, emoxipin, phosphaden, ATP, actovegin, lipostabil, pantothenic acid).
- Sedatives and other psychotropic drugs (tranquilizers, antidepressants, antipsychotics - in severe psychopathological syndromes).
- Anticholinesterase drugs (galantamine, stefaglabrin, amiridine) are indicated for vascular dementia.
- Adaptogens (Eleutherococcus, Saparal, Dibazole in small doses, Apilak) in the early stages of encephalopathy to reduce psychovegetative disorders.
- Physiotherapy procedures are used (galvanic collar according to Shcherbak, electrophoresis on the collar zone of euphilin and magnesium sulfate, electrophoresis according to Bourguignon noshpi, electrosleep, hyperbaric oxygenation, massage of the collar zone, etc.)

Vascular dementia is an acute or chronic decrease in cognitive functions that occurs as a result of a diffuse decrease in blood supply to the brain or local infarctions, which in most cases are associated with cerebrovascular diseases.

Vascular dementia is the second most important cause of dementia in the elderly. In most cases, it occurs in men, usually after the age of 70. It is most common in people who have vascular risk factors (including hypertension, diabetes, hyperlipidemia, smoking) and those who have had multiple strokes. Many patients have a combination of vascular dementia and Alzheimer's disease.

Vascular dementia occurs in situations where cerebral infarcts (or sometimes hemorrhages) lead to the loss of so many neurons or axons that the brain's functioning is disrupted. Vascular dementia results from disease of small vessels (lacunar disease) or medium-sized vessels (multi-infarct dementia).

Binswanger's dementia (subcortical atherosclerotic encephalopathy) is a rare variant of dementia arising on the background of lesions of small vessels of the brain, associated with severe poorly controlled arterial hypertension. In the development of the disease, multiple lacunar infarctions occur in the white and gray matter of the deep parts of the cerebral hemispheres.

Symptoms and signs are similar to other types of dementia. However, since vascular dementia is based on cerebral infarcts, the disease tends to develop discretely; each episode is accompanied by further intellectual decline, sometimes followed by a mild recovery. In the case of disease progression, deficit neurological symptoms often develop, which are represented by an increase in deep tendon reflexes, extensor plantar phenomena, gait disturbances, limb muscle weakness, hemiplegia, pseudobulbar paralysis with a syndrome of violent laughter and crying, extrapyramidal disorders. However, in the case of ischemic brain damage against the background of damage to small vessels, this deterioration is gradual. Cognitive functions may suffer selectively. Patients with incomplete aphasia can be largely aware of their deficit, therefore, with this type of dementia, depression may develop more often than with others. The diagnosis is similar to the diagnosis of other types of dementia. If there are focal neurological symptoms or evidence of cerebrovascular disease, a thorough stroke examination is mandatory. CT and MRI may reveal bilateral multiple infarcts in the hemispheres and limbic system, multiple lacunar cysts, or periventricular white matter lesions extending deep into the hemispheres. In Binswanger's dementia, neuroimaging reveals leukoencephalopathy in the area of the semioval center adjacent to the cortex, often with the presence of lacunae affecting gray matter structures in the depth of the brain hemispheres (including the basal ganglia, thalamus). In the differential diagnosis of vascular dementia and Alzheimer's disease, the use of the Hachinsky ischemic scale may be useful .

Prognosis of treatment. The 5-year mortality rate is 61%, and it is higher than for most other types of dementia, which is probably related to the concomitant disorders caused by atherosclerosis. In general, treatment is the same as for other dementias. However, vascular

dementia is preventable and its progression can be slowed down by lowering blood pressure and controlling it, cholesterol-lowering therapy, blood glucose regulation, and smoking cessation.

The effectiveness of drugs that improve cognitive functions, including cholinesterase inhibitors, has not been established. Despite this, due to the fact that many patients also have Alzheimer's disease, the use of these drugs may bring some benefit. The use of additional drugs for the correction of depression, psychosis and sleep disorders is useful.

Materials for self-control of training quality

Tests

1) What arteries supply the brain with blood?

Answer options:
a) internal carotid artery;
b) main artery;
c) Adamkevich's artery;
d) Zulch's artery.
Correct answer: a, b)

2) What arteries provide blood supply to the cerebellum?

Answer options:
a) anterior cerebral artery;
b) Adamkevich's artery;
c) main artery.
Correct answer: c)

3) What artery provides blood supply to the frontal lobe?

Answer options:
a) anterior cerebral artery;
b) posterior cerebral artery;
c) middle cerebral artery.
Correct answer: a, c)

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 16

Topic: Acute cerebral blood circulation disorder of the ischemic type. Acute hemorrhagic type cerebrovascular disorder.

Purpose: to acquaint applicants with the modern classification of cerebrovascular diseases, the frequency of these disorders, and the main provisions for their treatment.

Basic concepts: vascular diseases of the brain occupy a leading place among diseases of the nervous system and are a frequent cause of death, temporary or permanent disability, and are also acute conditions that require urgent help from a doctor of any specialty. Mortality from cerebrovascular diseases in the overall structure of mortality is about 14%.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Transient disorders of cerebral circulation

Transient cerebrovascular disorders (CCE) are acute disorders of cerebral hemodynamics, accompanied by general cerebral and focal symptoms, which regress within 24 hours after its development.

Patients with MPMK in neurological hospitals make up 20% of all patients with vascular diseases of the brain.

Transient ischemic attacks (TIA) and hypertensive cerebral crises belong to the group of transient disorders of cerebral circulation.

Etiology. The main reasons for the development of MPMK are atherosclerosis, stenotic lesions of the main arteries of the head, heart disease (heart disease, myocardial infarction).

Pathogenesis. Pathogenetic mechanisms of MPMC are diverse. Arteriogenic microembolisms with atheromatous masses from the carotid and vertebral arteries during the disintegration of atheromatous plaques, as well as platelet conglomerates formed on the altered area of the wall of large vessels, play a significant role in their occurrence. Having reached vessels of small caliber, more often cortical branches of cerebral arteries, emboli get stuck in them. By irritating the endothelium, they cause spasm of the surrounding vessels and increased permeability of their walls, followed by the development of perivascular edema of the brain tissue, which is accompanied by the appearance of focal symptoms. Since platelet emboli are quite loose, they are easily disintegrated or lysed, the edema caused by them is eliminated, which causes the reversal of the neurological deficit. Microemboli are also of cardiogenic origin. This mechanism is typical for patients with heart defects, myocardial infarction, atrial fibrillation, mitral valve prolapse.

MPMC can be caused by microthrombosis in case of blood disease (polycythemia, macroglobulinemia, thrombocytosis). Violations of microcirculation due to changes in the physical and chemical properties of blood and hemorheological disorders (increased blood viscosity, aggregation of platelets, erythrocytes, reduction of their deformation) are also important.

The mechanism of cerebrovascular insufficiency plays a certain role in the occurrence of MPMK. In the most general form, it is defined as a state of inconsistency between the need and the full supply of brain tissues with blood. Clinical symptoms can develop against the background of permanent insufficiency of blood supply to the brain due to atherosclerotic stenosis in its vascular system and especially under the influence of additional, extracerebral factors. These can be heart weakness, bleeding from internal organs, a decrease in blood pressure of various genesis. As a result of insufficient blood supply in certain areas of the brain, ischemia, hypoxia deepens, which is manifested by the corresponding focal symptoms. Restoration of tissue cerebral blood flow due to normalization of cardiohemodynamics or other factors is mostly accompanied by regression of neurological deficit.

In the development of MPMK, the mechanism of disruption of the reaction of autoregulation of cerebral blood flow in the event of a sharp increase or decrease in blood pressure is important. This mechanism is often accompanied by vasodilatation, excessive perfusion of brain tissue, and venous hypervolemia.

Clinical manifestations of MPMK can be caused not only by damage to the corresponding artery, but also by inadequate hemodynamic shifts of a compensatory nature, that is, by the phenomenon of theft. The essence of the phenomenon is the appearance of focal symptoms of insufficient blood supply to the brain not in the area of the affected artery, but in the area of the intact vessel that supplies blood to the basin of the affected artery. This mechanism is especially often the cause of impaired cerebral blood circulation in case of blockage of the proximal branches of the aortic arch (subclavian, common carotid arteries). A classic example is subclavian steal syndrome (SSS).

Hemodynamic and cerebral metabolism disorders are less important in the pathogenesis of MPMK.

One of the mechanisms of development of MPMK can be dystonic changes in cerebral vessels, especially in conditions of arterial hypertension. Violations of venous blood circulation are also of some importance. In some cases, MPMK can be caused by small focal hemorrhages. Vertebrogenic effects — compression of vertebral arteries by osteophytes in the area of vertebral joints — are also important in the development of MPMK.

I. Classification. Depending on the localization of dyscirculation, TIAs are divided into:

- TIA in the carotid basin

- TIA in the vertebro-basilar basin.

According to the degree of severity, the following are distinguished:

- light (last up to 1 hour),
- of medium difficulty (last up to 5-6 hours)
- severe (lasting more than 5-6 hours, up to a day).

Frequency criteria (according to E.V. Schmidt):

- frequent – 3 or more per year,
- rare - no more than 2 times a year.

II. Clinic and diagnostics.

MPMK is much more often manifested by cerebral vascular crises (in 2/3 of all patients).

The following types of cerebral vascular crises are distinguished: general, regional, mixed.

In the clinical picture of general cerebral crises, brain and autonomic vascular disorders prevail.

Depending on the localization of dyscirculatory disorders, regional crises are divided into carotid and vertebral-basilar.

Mixed cerebral crises are accompanied by general cerebral (significant headache, nausea, vomiting) and diffuse neurological symptoms (subcortical reflexes, convergence insufficiency, asymmetry of abdominal reflexes).

Cerebral hypertensive crises, taking into account the parameters of central hemodynamics, are divided into three types.

The hyperkinetic type of crisis occurs mainly in the early stages of arterial hypertension and is characterized by pronounced autonomic and vascular disorders, an increase in systolic blood pressure, an increase in cardiac output against the background of almost normal peripheral vascular resistance.

The hypokinetic type of crisis develops mainly in the later stages of hypertension and is accompanied by a significant increase in diastolic pressure, a decrease in stroke and minute blood volume, and an increase in peripheral vascular resistance.

In the case of a eukinetic type of crisis, cardiac output does not change significantly, systolic and diastolic pressure, and peripheral vascular resistance increase moderately. In the clinical picture, general brain symptoms are leading.

A single factor is of exceptional importance in the pathogenesis of cerebral hypertensive crises — disruption of the autoregulation reaction of cerebral blood flow. Two types of its violations are possible. The first is that in the case of a sharp increase in blood pressure, the cerebral (pial) arteries immediately expand, that is, the autoregulatory reaction of the narrowing of the vascular wall in response to changes in perfusion pressure is not carried out. In the case of the second type, which mainly occurs in conditions of a moderate increase in blood pressure, the pial arteries react with an autoregulatory reaction of constriction. Cerebral blood flow in this phase remains unchanged. However, a further increase in blood pressure leads to the fact that in the narrowed arteries, individual segments expand, that is, in these areas of the arteries, a disruption of the reaction of autoregulation of cerebral blood flow begins to occur. There is an alternation of widened and narrow segments along the course of the artery (sausage phenomenon). A peculiar "spotting" of vasomotor disorders is observed. Only in this phase does tissue cerebral blood flow begin to increase. As a result of impaired permeability of the blood-brain barrier, transudation of fluid and blood proteins into the interstitial tissue, brain edema may develop in patients. The most frequent complaint of patients is a significant headache accompanied by nausea and vomiting. Sometimes patients complain of dizziness, which is more often non-systemic and occurs during sudden head movements or head turns. There are usually no focal symptoms of brain damage, but stiffness of the occipital muscles, Kernig's symptom, and an increase in cerebrospinal fluid pressure may occur. Sometimes there are epileptic seizures.

Hypotonic cerebral crises are characterized by less pronounced general cerebral symptoms and are observed against the background of low blood pressure.

Transient ischemic attacks (TIA) are characterized mainly by focal symptoms, but are often

combined with general cerebral symptoms. If they are caused by dyscirculatory disturbances in the system of the internal carotid artery, then the most frequent are the focal symptoms of damage in the sensitive area, manifested by a feeling of numbness in the limbs, sometimes they spread to the face and other limited areas of the skin. Much less often, sensitivity disorders spread to one half of the body. There are paretic phenomena in the form of monoparesis, somewhat less often - hemiparesis, which are sometimes combined with mono- or hemihypesthesia, hemiparesthesia. There is an increase in tendon and periosteal reflexes and a decrease in superficial and abdominal reflexes, sometimes pathological reflexes, more often Babinski's reflex. If the left hemisphere of the brain is affected, speech disorders occur - transient aphasia or cortical dysarthria, which are often combined with one or another sensitivity or mobility disorder on the right side of the body. If TIAs are due to blockage or stenosis of the internal carotid artery in the neck, there is a transient crossed oculo-pyramidal Lascaux-Radovich syndrome: decreased vision or blindness on the side of the focus and weakness of the limbs on the opposite side. Sometimes reduced vision in one eye (amaurosis fugax) is combined only with hyperreflexia of the contralateral limbs. In some patients with pathology of the main vessels of the head of the MPMK, attacks of Jacksonian (cortical) epilepsy may occur.

MPMK in the system of vertebral-basilar vessels occur almost 2 times more often than in the carotid basin. They are characterized by attacks of systemic dizziness, vegetative-vascular disorders. Patients often complain of noise, ringing in the ears and head. Sometimes there is a decrease in hearing in one or both ears. A frequent complaint is a headache, more often of a throbbing nature, mainly in the occipital region. Nausea, vomiting, hiccups, pale face, cold sweat appear. Characteristic visual disorders in the form of photopsia, metamorphopsia, blurred vision, visual field defects. Symptoms of damage to the oculomotor nerves (double vision, convergence disorder, eye muscle paresis and gaze paresis), trigeminal (sensory disturbances on the face in Zelder areas), as well as signs of bulbar syndrome: dysarthria, dysphagia, dysphonia often appear. Possible cerebellar-stem disorders in the form of nystagmus, disorders of statics and coordination of movements. Alternating syndromes are relatively rare. Changes in the emotional and volitional sphere are much more common: increased fatigue, reduced work capacity, irritability, often depressed background mood, apathy, sometimes paroxysmal pressure increase. Attacks of a sudden fall can develop - drop attacks (drop attacks) - without loss of consciousness, which are observed in the presence of osteochondrosis of the cervical spine, especially during turns, changes in the position of the head. They are directly related to the transient loss of postural tone caused by ischemia of the brain stem (pons, medulla oblongata, reticular formation). In case of vascular damage to the stem-hypothalamic part of the brain, other paroxysmal conditions arise: hypersomnia, cataplexy syndromes, as well as vegetative-vascular crises. However, such disorders as migraine attacks, Meniere's disease, are not usually classified as MPMK.

The duration and reversibility of neurological symptoms in patients with MPMK is different, from short-term episodes to 24 hours. Most of them last less than half an hour. This indicates their hemodynamic, metabolic, and structural heterogeneity. Modern notions of cerebral palsy, based on the duration of ischemic episodes, are inaccurate, since more than 1/4 of patients show signs of brain infarction on CT. Therefore, the term TIA is proposed to call only those episodes of focal ischemia, when the neurological symptoms are completely reversed, and there are no signs of focal brain damage on CT.

Data of additional examination methods. To establish the main disease that led to the occurrence of TIA, special research methods are used:

a) ultrasound dopplerography, including transcranial or duplex scanning: determines the presence of stenoses or occlusions of the main arteries of the head and neck, as well as collateral circulation; in the clinic, the USG method is widely used for the examination of patients with vascular pathology of the brain, with the aim of early diagnosis of stenoses and occlusions of the main arteries of the head, the appointment of adequate vascular therapy, and timely surgical treatment.

Duplex Doppler scanning of the bifurcation of the common carotid artery: imaging of the

vessel wall

b) CT scan, MRI to exclude the focus of ischemia in severe TIA;

c) ECG – determines the state of coronary vessels, heart rhythm disorders;

d) R-graph of the cervical spine - reveals osteochondrosis, anomalies of the spine development;

e) rheological indicators of blood - detection of increased aggregation of platelets, viscosity, hematocrit;

g) general blood test - hypercholesterolemia, hyperproteinemia, etc.

g) examination of the fundus

Differential diagnosis is carried out with

a) migraine crisis

b) partial epileptic attack

c) Meniere's disease

d) hypoglycemic state

Migraine. It causes short-term neurological disorders, such as migraine aura in the form of hemianesthesia, hemiparesis, aphasia, and unilateral vision loss. It must be remembered that these symptoms are typically accompanied by a headache attack, and sometimes occur in isolation, as the equivalent of a migraine attack. It is diagnostically important that focal symptoms develop more slowly in migraine aura than in TIA (within 20-30 minutes).

Partial epileptic seizures. They can be manifested by transient motor, sensory, visual, speech disorders, which resembles the TIA clinic. However, with epiattacks, it is characteristic that sensory and motor disorders have the ability to spread - the so-called "Jackson's march". Characteristic changes on the EEG are detected in partial epiconvulsions. For the final diagnosis, a CT scan or MRI is required.

Meniere's disease. As with TIA, sudden dizziness accompanied by nausea and vomiting occurs. But with this pathology, the symptoms of brain stem damage (vertical nystagmus, diplopia, facial sensitivity disorders, pyramidal signs, etc.) characteristic of TIA in the vertebral-basilar basin are not noted .

Hypoglycemic state. Gives a similar clinic with TIA - weakness, dizziness, loss of consciousness. Differentiating features are the fact that in hypoglycemic state, the above-mentioned symptoms appear after sleep, at the moment of awakening, or after physical exertion. For differentiation during such a condition, it is necessary to examine the level of glucose in the blood. After parenteral administration of glucose, a significant improvement of the condition is noted.

Complication of the disease. A severe TIA, with severe cerebral and focal symptoms, with inadequate or untimely therapy, can turn into an ischemic stroke.

Tactics of treatment.

With heart failure and a decrease in systolic blood pressure below 120 mm Hg. cardiotonic drugs are prescribed: corglycon in a dose of 1 ml of a 0.06% solution or 0.25-1.0 ml of a 0.5% solution of strophanthin K intravenously in an isotonic sodium chloride solution, 1 ml of cordiamine subcutaneously or intramuscularly, 2 ml of a sulfocamphocaine solution intramuscularly If blood pressure is significantly reduced, prescribe 1 ml of 10% caffeine solution subcutaneously or intramuscularly.

With a moderate increase in blood pressure (systolic no more than 180 mm Hg), antispasmodics are prescribed: dibazole (2-4 ml of a 1% solution), papaverine hydrochloride (1-2 ml of a 2% solution), no-shpa (2-4 ml 2% solution). To improve cerebral blood circulation, vasoactive drugs are prescribed: 10 ml of a 2.4% solution of Euphilin in 10 ml of isotonic sodium chloride solution, 10-20 mg of cavinton intravenously in 200 ml of 0.9% sodium chloride solution, instenon 2 ml intravenously in 200 ml 5% glucose solution or by jet in 20 ml of 40% glucose solution, cinnarizine at a dose of 0.025 g three times a day or nimotop 60 mg 4 times a day.

To improve microcirculation and rheological properties of blood, pentoxifylline (5-10 ml intravenous drip in 250-500 ml isotonic sodium chloride solution), reopoliglukin or reogluman

(400 ml intravenous drip over 30-60 minutes), sermion (4-8 mg intravenous drop by drop).

To combat cerebral edema, mannitol is used in a dose of 150-200 ml of a 10-20% solution intravenously by drip, furosemide (0.04-0.08 in the morning), Lasix (2 ml of a 1% solution intramuscularly or intravenously in 40 ml of isotonic sodium solution chloride or with 10 ml of a 2.4% solution of Euphilin), Triampur (1-2 tablets in the morning), Dexon (4-8 mg intramuscularly or intravenously), human serum albumin in a dose of 50-100 ml of a 5% solution intravenously by drip, antihistamines (suprastin, diphenhydramine in a dose of 2 ml of 1% solution intramuscularly).

With a significant increase in blood pressure, treatment is carried out, as in a hypertensive crisis.

For dizziness, use beloid (belaspon, belataminal) 1 dragee 3 times a day), for vomiting and hiccups - aminazine (1 ml of 1.5% solution intramuscularly), validol (1 tablet under the tongue), haloperidol (1, 5-2 mg) by mouth or droperidol (1-2 ml of 0.25% solution) intravenously or 1 ml in 20 ml of 40% glucose solution intravenously, sibazone (2-4 ml of 0.5% solution intravenously), cerukal (0 .01 orally or 2 ml intramuscularly of 0.5% solution).

With intense cephalgias - baralgin (spasgam) 5 ml intramuscularly, intravenously; analgin, sedalgin, pentalgin, mustards on the collar zone.

To improve the metabolism of neurons, prescribe piracetam (20% solution 5-10 ml intravenously) or 1 capsule three times a day, encephabol one dragee three times a day, instenon in a dose of 1 dragee three times a day or intravenous drip in a dose of 2 ml in 200 ml of 5% glucose solution, cerebrolysin in a dose of 10 ml intravenously drip in 200 ml isotonic sodium chloride solution, actovegin (2-5 ml each intramuscularly or intravenously), aminalon (0.25 g 3 times by mouth), group B vitamins intramuscularly , cocarboxylase.

In the case of repeated TIAs caused by the pathology of the main vessels of the head in the extracranial department, the question of the possibility of surgical treatment is resolved.

The examination of work capacity depends on the severity of the TIA, its duration, the underlying disease, the nature and working conditions.

Indications for hospitalization are:

a) severe TIA and TIA of medium severity;

b) repeated TIA.

With light TIA, patients are recognized as temporarily unable to work with the issuance of a sick leave for up to 2 weeks, with TIA of medium severity – up to 3-4 weeks, with severe TIA – up to 1.5-2 months. Repeated, frequent TIAs, which limit the working capacity of patients, are an indication for referral to MSEK.

Dispensary. Patients with TIA are subject to dispensary observation with examination 1-2 times a year, need systematic treatment taking into account the main disease, rational employment.

Rehabilitation. It is possible in polyclinic conditions, in sanatoria-prophylaxis. Patients are prescribed rational psychotherapy, diet therapy, physical therapy, pharmacotherapy (including treatment of the main vascular disease).

Hypertensive cerebral crises make up 13-15% of all acute disorders of cerebral circulation.

Etiology. Hypertensive crises occur with hypertensive disease, symptomatic arterial hypertension, with diseases of the kidneys, adrenal glands, etc.

Pathogenesis. An increase in blood pressure more than 180-200 mm Hg. Art., leads to a violation of the mechanism of autoregulation of cerebral blood circulation, hyperemia, cerebral edema, deficiency of cerebral blood circulation develop.

I. Classification. There are three types of crises depending on the type of systemic hemodynamics: hyperkinetic, hypokinetic, eukinetic.

The hyperkinetic type is accompanied by an increase in cardiac output (cardiac index higher than 4.5 l.min.m2) with normal or slightly reduced total peripheral resistance.

The hypokinetic type is accompanied by a decrease in cardiac output (cardiac index lower than 2.8 l.min.m2) and a sharp increase in total peripheral resistance.

Eukinetic type - in which the cardiac output is normal, and the total peripheral resistance is moderately increased.

According to the degree of severity, there are light (lasting up to 1-2 hours), medium severity (lasting 3-4 hours), accompanied by light focal (more often trunk) symptoms, and severe crises (lasting up to a day), accompanied by scattered organic symptoms.

Frequency criteria: with light crises: frequent - 4 or more times a month, medium frequency - 3-4 times a month, rare - 1-2 times a month; with moderate and severe crises: frequent - 5 times (or more) a year, medium frequency - 3-5 times a year, rare - 1-2 times a year.

Diagnostics.

1. Complaints of a significant increase in blood pressure, headache, nausea, vomiting, excitement, palpitations, difficulty breathing, pain in the area of the heart.

2. Anamnesis. The disease often occurs acutely in patients with hypertension and symptomatic arterial hypertension.

3. Objective examination data. Patients are excited, inadequate, consciousness can be dimmed, sometimes epinaives appear against the background of pronounced general brain symptoms. Severe vegetative symptoms (hyperemia of the skin, tachycardia, tachypnea, hyperhidrosis, cold extremities, polyuria).

The clinic of hyperkinetic hypertensive crisis often begins suddenly, without precursors, systolic pressure rises, sharply expressed general brain symptoms (headache, psychomotor agitation, vomiting). Vegetative manifestations are bright. The duration of the crisis is insignificant. Characteristic for the initial stage of hypertensive disease.

A hypokinetic hypertensive crisis begins gradually against the background of long-term arterial hypertension, in the late stages of hypertensive disease, diastolic pressure increases, changes on the ECG are manifested in the form of a slowing of intraventricular conduction, a decrease in the ST segment. Patients are sleepy, the face is swollen, pale, diuresis is increased, the probability of a stroke is quite high.

Eukinetic hypertensive crisis develops quickly against the background of increased systolic and diastolic blood pressure. Complicated by left ventricular failure, cerebral edema.

4. Data of additional examination methods.

ECG - characteristic changes for hypertensive disease

CT scan, MRI (in vascular mode) to rule out brain hemorrhage, detect signs of cerebral edema.

Fundus - changes characteristic of hypertensive disease.

Differential diagnosis is carried out with

a) acute hypertensive encephalopathy. As a result of cerebral edema with microhemorrhages, acute general brain symptoms occur: loss of consciousness or stupor, psychomotor agitation, disorientation, tonic-clonic convulsions, mild focal symptoms, congestive discs of the optic nerves. Duration up to several days, coma and death of the patient are possible.

Principles of treatment. The modern approach to the treatment of cerebral vascular crisis requires mandatory hospitalization in an intensive care unit or intensive care unit, where there is the necessary equipment for intensive care and continuous monitoring of vital functions. Patients with cerebral vascular crisis should be observed by a neurologist and an intensivist, and according to indications, by doctors of other specialties. The principles of medical intervention in this category of patients are based on the leading pathogenetic mechanisms of the development of this pathological condition and the features of its clinical manifestations. The main areas of primary therapy include: lowering blood pressure, measures aimed at combating cerebral edema, anticonvulsant treatment. Correction of accompanying metabolic disorders, i.e. maintenance of homeostasis, neuroprotection, correction of disturbed hemorheological and hemostasiological parameters, are auxiliary, but no less important.

It should be noted that the symptoms of acute hypertensive encephalopathy usually quickly regress against the background of a decrease in blood pressure; however, a decrease in systolic blood pressure to approximately 160-170 mm Hg, diastolic blood pressure to 100-110 mm Hg is considered safe. during the first 24 hours. As a rule, they recommend an initial decrease in blood pressure by 25% from the initial need - to blood pressure indicators 10-15% higher than usual

numbers.

The drugs of choice for stopping hypertensive crisis are ACE inhibitors (captopril - 25 mg under the tongue, if necessary, 25 mg repeatedly every 30 - 60 minutes; and enalaprilat - 1.25 - 6 mg in the form of an IV bolus administered every 6:00), the appointment of hypotensive drugs of central action (clonidine - 0.1 - 0.2 mg orally, then 0.1 mg every hour, total dose no more than 0.6 - 0.8 mg) and ganglioblockers (pentamine, arfonad) are also not excluded. . In acute hypertensive encephalopathy associated with pheochromocytoma, the first-line drug is phentolamine (IV 5-10 mg, after stabilization of blood pressure at a normal or slightly elevated level IV 2.5-5 mg every 2-4 hours; instead of phentolamine it is possible to use an alpha-adrenoblocker tropafen - intramuscularly 20-40 mg or intravenously 10-20 mg). Among other antihypertensive agents, in the treatment of acute hypertensive encephalopathy, the alpha- and beta-adrenoblocker labetolol is used (IV drip 50 - 200 mg / day in 200 ml of isotonic NaCl solution, daily dose no more than 200 mg). In some cases, it is possible to use furosemide (which, in addition to lowering systemic blood pressure, causes a decrease in intracranial pressure, which is necessarily increased in cerebral vascular crisis) - 20 - 40 mg IV. Before using each of the above-mentioned drugs, it is necessary to evaluate all available contraindications to their use (so, for example, with furosemide - hypovolemia, hyperosmolarity, hypernatremia, etc.; enalaprilat and captopril - bilateral renal artery stenosis, etc.).

The use of peripheral vasodilators (sodium nitroprusside, nitroglycerin, diazoxide) is limited, as an opinion is expressed about their possible negative effect in acute hypertensive encephalopathy, due to the possibility of worsening venous outflow from the skull and, as a result, an additional increase in CSF pressure (they necessarily develop and are present in acute hypertensive encephalopathy) against the background of their use.

Complication. A severe hypertensive crisis can be complicated by acute hypertensive encephalopathy, cerebral hemorrhage, or subarachnoid hemorrhage.

Examination of working capacity. It depends on the severity of the hypertensive crisis, its duration, nature and working conditions. Indications for hospitalization are a severe hypertensive crisis, with ineffective emergency therapy, a repeated increase in blood pressure after the crisis subsides, destabilization of cardiac activity, acute hypertensive encephalopathy. In the case of light hypertensive crises, the patient is issued a sick leave for 3-7 days, in the case of a crisis of moderate severity - up to 10-15 days, severe - up to 1 month.

Dispensary. It includes systematic treatment of patients with hypertension and symptomatic arterial hypertension, dispensary supervision 1-2 times a year, rational employment taking into account additional restrictions of work activity due to repeated crises, timely determination of disability.

Rehabilitation. It is carried out in polyclinic conditions, with sanatorium-resort treatment. Includes rational psychotherapy, diet therapy, exercise therapy, pharmacotherapy for the prevention of repeated crises (sedatives, hypotensive drugs, diuretics).

Cerebral stroke. Cerebral stroke (from Latin insultus - blow, push) is an acute disturbance of cerebral blood circulation, which is accompanied by structural changes in the brain tissue and persistent organic neurological symptoms that persist for more than a day. According to the nature of the pathological process, two types of stroke are distinguished: hemorrhagic and ischemic (cerebral infarction, softening of the brain). Over the past decades, the structure of cerebrovascular diseases has changed due to the clear predominance of ischemic strokes over hemorrhagic ones. Currently, the ratio of brain hemorrhages to brain infarctions is 1:4. This is largely due to the increase in the specific weight of the elderly in the population.

Ischemic stroke (brain infarction).

Etiology. Ischemic stroke most often occurs as a result of atherosclerotic lesions of the main vessels of the head and brain, often against the background of arterial hypertension and diabetes. Less often, the cause of the disease is rheumatism, vasculitis, blood diseases. Mental and physical overstrain and stress play a provocative role in the development of ischemic stroke.

Pathogenesis. The circulatory system, as is known, has three components: the heart, which

acts as a pump that ensures the rhythmic supply of blood to the vessels; blood vessels and vascular content, which is understood as the amount, composition and properties of blood. Violation of the functioning of individual links of this complex system can be the cause of ischemic disorders of cerebral circulation. The development of an ischemic stroke can be facilitated by a complex of pathophysiological disorders, among which the leading ones are atherosclerotic lesions of the vessels of the brain, complicated by thrombosis and stenosis; violation of the rheological properties of blood and related microcirculation disorders, changes in systemic hemodynamics caused by various forms of heart pathology. Among the many mechanisms that directly cause ischemic disruption of cerebral circulation, the leading place belongs to thromboembolic and hemodynamic factors. That is, an ischemic stroke can develop either as a result of a complete blockage of the lumen of a vessel by a thrombus or embolus and blocking blood flow through it, or by the mechanism of vascular cerebral insufficiency, which manifests itself in the basin of a stenotic vessel and is aggravated by a violation of systemic hemodynamics. The implementation of pathogenetic prerequisites in focal ischemia with the development of cerebral infarction occurs as a result of the disruption of regional and systemic mechanisms of cerebral blood circulation compensation. About 40% of all ischemic strokes develop by the mechanism of thromboembolism of cerebral vessels. Atherosclerotic lesions of extra- and intracranial arteries lead to vessel thrombosis. Moreover, atherosclerotic plaques are much more common in the main vessels of the head than in intracerebral arteries. The source of cerebral embolism is often the products of the breakdown of atherosclerotic plaques from the carotid and vertebral arteries, loose fibrin-free thrombi, platelet aggregates. In addition to arterio-arterial emboli, in approximately 15-20% of cases, ischemic disorders of cerebral circulation are caused by cardiogenic embolism, which often occurs with endocarditis, heart defects, myocardial infarction, atrial fibrillation. During operations on the heart and vessels of the head, air embolism can be observed. Fat embolism can occur in injuries with bone fractures. A gas embolism is possible as a result of the accumulation of nitrogen in the blood during rapid decompression and a decrease in atmospheric pressure (when performing caisson work). In addition to thrombosis and embolism, hemodynamic mechanisms play an important role in the development of ischemic stroke, in particular vascular cerebral insufficiency, disruption of the autoregulation of cerebral blood flow, angiodystonic disorders in the vessels of the brain (vasoparesis, stasis), as well as cerebral steal syndromes. Regarding the spasm of cerebral vessels as a possible mechanism of ischemic stroke development, it should be noted that its existence is beyond doubt and is an important chain of the autoregulation system of cerebral blood circulation in response to a significant increase in systemic arterial pressure and a decrease in the concentration of carbon dioxide in the blood. However, not everyone recognizes the role of neurogenic spasms of cerebral vessels in the development of cerebral infarction. Most researchers have not received direct evidence of its role in the occurrence of cerebral ischemia. Pathophysiology. The use of the latest methodological approaches made it possible to study focal ischemia and its further evolution at the molecular level, contributed to the accumulation of fundamentally new facts, and the development of modern concepts of the pathogenesis of brain ischemia. One of them is the concept of "threshold ischemic blood flow". The threshold is determined by a critically low level of cerebral blood flow and insufficient oxygen supply. There is an upper ischemic threshold (electrical injury) with a blood flow of 20-18 ml/100g/1 min, below which somatosensory evoked potentials and EEG activity disappear, synaptic transmission is disrupted, but the energy potential, the function of ion pumps is preserved; and the lower ischemic threshold (energy damage) with a blood flow of 12-10 ml/100g/1 min, below which ATP is not synthesized, the function of cell membranes is disturbed, neurons lose potassium, gain calcium, sodium and water by osmosis. An increase in the concentration of calcium in neurons activates membrane phospholipases, promotes the release of excessively toxic fatty acids, and thus is a chain of many processes that lead to the destruction and death of brain cells. A decrease in cerebral perfusion below the threshold of energy damage is compensated for by increased extraction of oxygen from arterial blood by brain tissue. For a short period of time, it helps to maintain the metabolic level of oxygen and prevents the development of brain infarction. However, after that,

its consumption decreases. Insufficient supply of oxygen leads to the transition to anaerobic glycolysis to maintain the possibility of ATP synthesis due to lactic acid and CO2 accumulation, which leads to the development of metabolic acidosis. The latter is the main cause of cytotoxic (exchange) swelling of the brain, which develops in the intracellular sector a few hours after the formation of ischemia. 2-7 days after the development of an ischemic stroke, vasogenic swelling of the brain occurs in the extracellular sector. As a result of a decrease in perfusion pressure, histamine is released from mast cells, the blood-brain barrier is disrupted, which causes the transudation of fluid and blood proteins into the interstitial tissue. Cerebral edema further disrupts oxygen diffusion, cellular exchange and microhemocirculation, a vicious circle develops with increasingly severe damage and expansion of the ischemic focus. At this stage of the evolution of cerebral ischemia, autoregulation of cerebral blood flow is disturbed, platelet aggregation, intravascular stasis, venous stasis, and venous hypervolemia increase, which in turn deepens the degree of ischemia, making it irreversible. After that, the oxygen tension in the brain tissue does not decrease. The concept of the so-called ischemic penumbra is closely related to the concept of "threshold ischemic blood flow". Ischemic "penumbra" or border zone is an area that forms around the ischemic center or infarct core. This part of the ischemic brain is called the "ischemic penumbra" similar to the picture observed during a total solar eclipse, during which there are penumbra zones around the completely dark center (I. Azighir, 1982). From a clinical point of view, the significance of this zone is that the disturbances in the function of neurons in it are reversible within a limited time, sometimes reaching several hours. The duration of this tolerance is related to the degree of blood flow reduction. Its increase in the ischemic "penumbra" zone allows the normal functioning of the neurons of this area to be restored, while its decrease leads to the death of all types of cells, including not only neurons, but also neuroglial cells, which perform supporting and other auxiliary functions. Recently, the main stages of damage to brain tissue in cerebral ischemia have been determined, the understanding of which is extremely important for the justification of therapeutic intervention and the possible termination of pathological changes. Brain ischemia leads to a number of metabolic disorders in the cell. In particular, one of the main reasons for the death of neurons in the ischemic "penumbra" zone can be the glutamate cascade. Glutamate is an excitatory mediator and is found in many neurons in the brain. Under normal conditions, cells release glutamate as a result of depolarization of the outer membranes. In healthy brain tissue, neurons and neuroglia cells absorb excess glutamate from the intercellular space, but the cells of the ischemic penumbra, which surround the focus, are deprived of energy for this. Excessive accumulation of glutamate and its related compounds can lead to the death of brain neurons due to a cascade of bathobiochemical changes that form three stages of damage to brain tissue. In the first stage, as a result of a lack of oxygen and nutrients when the vessel is blocked, neurons excessively release glutamate, which activates various glutamate receptors on other neurons, causing negative intracellular effects. Namely, as a result of the binding of glutamate to H-methyl-D-aspartate receptors, calcium channels are opened, through which a significant amount of calcium ions pass into the cells. Neurons also take in sodium and water by osmosis, which leads to cell swelling. At this stage, the mechanisms of synaptic transmission, which are reversible, are disrupted. In the next, second stage, the concentration of calcium ions in cells increases partly because intracellular mediators cause the release of calcium from tissue depots. The activity of enzymes that increase sensitivity to glutamate and other exciting stimuli increases. Toxic excitation spreads to other cells. As a result of the second stage, conditions are created for the third stage of brain tissue damage, during which irreversible changes in cells occur. Increasing the concentration of calcium in the cell increases the activity of enzymes that break down DNA, proteins and phospholipids. One of the products of phospholipid degradation is arachidonic acid, the metabolism of which stimulates the formation of eicosanoids, which, together with platelet activation factor, lead to the deepening of microcirculation disorders, the formation of blood clots and the spread of ischemia. This cascade of pathobiochemical disorders leads to the death of neurons. So, in a short period of time from the moment of cerebral ischemia to the formation of irreversible brain damage, complex pathobiochemical and pathological processes take place.

Pathomorphology. Complete blockage of a cerebral vessel, which supplies blood to a certain area of the brain, leads to irreversible changes in neurons, their death within 5-10 minutes, that is, focal necrosis of the brain occurs. These two interrelated factors - time threshold and morphological changes - confirm the classical concept that among somatic organs the brain is particularly highly sensitive to hypoxia, most vulnerable to ischemia. Heart attacks can occur in various parts of the brain. Most often (up to 75%) they develop in the subcortical-capsular area, that is, in the basin of the middle cerebral artery. In the second place in terms of frequency are heart attacks localized in the stem part of the brain, which is supplied with blood by the arteries of the vertebral-basilar basin. The localization of a brain infarction is largely determined by the pathogenetic mechanism, as well as the pace of its development. Heart attacks, which occur in connection with vascular cerebral insufficiency, usually develop on the surface part of the brain, in the cortical layer. More often, this happens in the areas of closure of the peripheral branches of the middle cerebral artery with the areas of irrigation of the anterior and posterior cerebral arteries (zones of adjacent blood circulation). In other words, such heart attacks develop according to the principle of the "last straw" (Zulkh, 1955). The sizes of the infarction are different: from small cells to large ones that cover the cortical-subcortical-capsular area of the brain. The latter are often observed when the large trunk of the middle cerebral artery or the intracranial section of the internal carotid artery is blocked. Brain infarcts are white, red (hemorrhagic), and mixed (A.N. Koltover). White or gray heart attacks make up the main part of ischemic strokes and occur in 85-90% of their total number, mixed - in 5-10%, red heart attacks are even rarer. White heart attacks occur in different areas of the brain. At the initial stage of their formation under a microscope, swelling of nerve cells, paleness of protoplasm when staining cells, and their cytolysis are noted. A significant number of ganglion cells, changed according to the ischemic type, are also revealed. Subsequently, there is a restriction of the cell, in the center of which necrosis is formed with complete damage to neurons, glial cells, and blood vessels. The brain substance in the area of a heart attack turns into a mushy mass of gray color. The process ends with the formation of a scar and a cavity filled with serous fluid. Hemorrhagic (red) infarcts develop only in the brain substance, primarily in the cortex, somewhat less often - in the subcortical nodes, cerebellum and do not form in the white matter of the brain at all. Red infarcts are cells of red color, with a vitreous consistency, which resemble hemorrhages of the hemorrhagic seepage type. But the mechanism of their development is different. Hemorrhagic heart attacks are always preceded by ischemia, then hemorrhage joins the ischemic tissue. Microscopic examination of the cell reveals a large number of cells that have changed according to the ischemic type. Hemorrhagic heart attacks always have a clear border. Haemorrhages of the hemorrhagic seepage type are small red cells that do not have a clear border. As a rule, there are no signs of ischemia in the foci of hemorrhages of the hemorrhagic seepage type. They never develop in the cortex and cerebellum, but usually in the visual hillocks and pons. Mixed heart attacks include such brain infarctions, which are ischemic in some areas, and hemorrhagic in others. Moreover, hemorrhagic areas develop only in the gray matter of the brain. Clinic. Ischemic stroke occurs mainly in middle-aged and elderly people, but sometimes it can also develop in young people. The disease occurs at any time of the day, most often during sleep or immediately after it. In some cases, ischemic stroke occurs after physical exertion, psycho-emotional overstrain, alcohol consumption. The development of cerebral infarction is often preceded by transient disturbances of cerebral circulation. The most characteristic of an ischemic stroke is the gradual increase of focal neurological symptoms over several hours, sometimes 2-3 days. Sometimes there is a flashing type of development of symptoms, when the degree of their severity changes. In approximately 1/3 of cases, the disease develops acutely, apoplectiformly. Pseudotumorous development of brain infarction, when focal symptoms increase over several weeks, is much less common. A characteristic feature of an ischemic stroke is the predominance of focal neurological symptoms over general brain symptoms, which are sometimes absent at all. The focal symptomatology of a brain infarction is determined by the localization of ischemia, the vascular pool in which a violation of cerebral blood circulation occurred. More serious disorders of consciousness with the development of sopor or comatose state are observed only in large

hemispheric infarctions, which are accompanied by significant swelling of the brain and secondary dislocation-stem syndrome. This mostly occurs when the internal carotid artery is blocked in the intracranial section, or the main trunk of the middle cerebral artery. Loss of consciousness also occurs with an ischemic stroke in the vessels of the vertebral-basilar basin. Vegetative disorders and meningeal signs do not occur in the initial period of ischemic stroke. They can appear with the development of cerebral edema. Heart rhythm disturbances are often registered in many patients with obvious signs of heart failure. Blood pressure is normal or low. Arterial hypertension is quite often observed. Among patients with ischemic stroke, a "small stroke" is distinguished, when the restoration of neurological functions is completed within 2 to 21 days. Since 1981, it has been included in the WHO classification of cerebrovascular diseases and in the classification of vascular diseases of the nervous system existing in Ukraine. Clinical manifestations of a small ischemic stroke often resemble the course of lacunar infarctions - a special form of vascular pathology of the brain. Lacunar infarcts are one of the clinical forms of ischemic disorders of cerebral circulation caused by damage to intracerebral arteries in arterial hypertension and are characterized by the development of small foci of necrosis in the deep parts of the brain. In the structure of cerebrovascular diseases, they make up approximately 19% of all cases of ischemic strokes. As clinical and morphological comparisons as a result of computed tomography studies show, lacunar infarcts are a morphological substrate of hypertensive encephalopathy and vascular dementia. Embolic cerebral infarction occurs more often in young and middle-aged people. The disease develops suddenly, without any precursors, often after physical effort or under the influence of an emotional factor. Loss of consciousness is possible. The coma that may develop is short-lived and shallow. Convulsive attacks are observed more often than in other forms of ischemic stroke. Most often, an embolic stroke occurs in the basin of the branches of the left middle cerebral artery with damage to the internal capsule and subcortical nodes, which causes the development of right-sided hemiplegia or hemiparesis, speech impairment. Sometimes retinal vessel embolism is possible, manifested by scotomas and even blindness. Recovery of lost functions, although incomplete, occurs relatively quickly. The diagnosis of an ischemic stroke is based on a careful study of the pre-stroke period, analysis of the rate of its occurrence and the dynamics of the disease. It is important to determine whether the patient has arterial hypertension, manifestations of coronary heart disease (heart rhythm and conduction disturbances, signs of blood circulation insufficiency), clarify the medical history (experienced myocardial infarction, stroke, transient ischemic attacks), identify possible risk factors (diabetes mellitus, nicotine intoxication , alcohol abuse, excess body weight, hereditary predisposition), carefully examine the pulsation of the peripheral and main vessels of the head and neck. The obtained data in combination with clinical neurological symptoms, the results of an examination of the fundus, rheological properties of blood, cerebrospinal fluid, echo and electroencephalography, ultrasound dopplerography, ECG, as well as radiological methods - angiography, computer tomography - make it possible to make a definitive diagnosis of ischemic stroke and differentiate it from other diseases that have a similar clinical course.

Given the different tactics of treatment of brain hemorrhages and cerebral infarction, timely differential diagnosis of these clinical forms of acute cerebrovascular disorder is important. It should be noted that individual symptoms have a relative diagnostic value for determining the nature of a stroke. However, a certain combination of symptoms, taking into account the data of laboratory and instrumental studies, allow to correctly recognize the nature of a stroke in the vast majority of cases. Of course, computer tomography of the head is of decisive importance in the differential diagnosis of hemorrhagic and ischemic stroke, with the help of which a cerebral hemorrhage can be diagnosed immediately after the development of a stroke by the presence of a focus of increased density in the brain, and a brain infarction in the form of cells of reduced density is detected in the middle or at the end of the first day after the onset of the disease.

On angiograms with hemorrhaging in the hemisphere, there is an avascular zone and displacement of arterial branches. Disruption of the contrast agent in the main and intracerebral arteries is revealed. Cerebral stroke must be differentiated from diseases that more or less resemble

an acute violation of cerebral circulation in terms of development and course. These include: 1) traumatic brain injury in the acute period; 2) myocardial infarction, which is accompanied by loss of consciousness; 3) brain tumors with apoplectiform development due to hemorrhage into the tumor; 4) hyper- or hypoglycemic coma; 5) uremia; 6) epilepsy.

Clinical manifestations of a cerebral stroke resemble a closed craniocerebral injury, especially in cases where the patient has a disorder of consciousness. In the presence of signs of injury, X-ray of the skull, echoencephalography, examination of cerebrospinal fluid is necessary. With epidural and subdural hematomas of traumatic genesis, there is a violation of the integrity of the bones of the skull, a shift in the median M-echo signal, blood impurities in the cerebrospinal fluid. The data of cerebral angiography and computer tomography of the brain help to determine the nature of the pathological process.

Myocardial infarction is very similar to cerebral stroke; it also develops suddenly, with loss of consciousness due to a sharp decrease in total volume cerebral blood flow and secondary brain tissue hypoxia. At the same time, during an acute myocardial infarction, there is a drop in blood pressure. As for the focal symptoms of the impression of the hemisphere and the stem part of the brain, they are not detected, except for those cases when myocardial infarction is combined with the development of cerebral infarction (heart attack-stroke process). The greatest difficulties arise when differentiating a brain tumor complicated by hemorrhage and cerebral stroke. It should be taken into account that brain tumors are latent for some time, with the absence or late appearance of focal symptoms. The study of cerebrospinal fluid is of some importance for determining the nature of the process - the presence of protein-cellular dissociation, xanthochromia. Computer tomography data of the brain are of decisive importance for the topical diagnosis and for determining the nature of the disease.

Hyperglycemic coma is observed in severe forms of diabetes. It differs from brain hemorrhage by a drop in blood pressure, Kussmaul breathing with the smell of acetone, and the presence of hyperglycemia and glycosuria. In case of hypoglycemic coma, motor excitement and an increase in blood pressure are noted. Fibrillar twitching occurs. Seizures, focal neurological symptoms, which regress after infusion of glucose solution, may develop. The diagnosis is confirmed by a blood sugar test.

With uremic coma, there are also focal symptoms of loss in the form of paralysis, speech disorders. Severe epileptiform seizures occur. For the diagnosis of uremia, the examination of urine and the determination of residual nitrogen in the blood are of crucial importance. Uremic coma is recognized by the smell of ammonia from the patient when he breathes.

The diagnosis of epilepsy with the development of a neurological deficit after an attack is confirmed or rejected based on the analysis of anamnestic data and EEG indicators.

Modern approaches to the treatment of cerebral stroke involve the fastest possible hospitalization of patients, early periods of therapeutic intervention after the development of a stroke, when regeneration of affected areas begins in damaged cells, and other neurons form new synaptic connections to compensate for lost ones. To a large extent, the success of stroke treatment is determined by the time factor. Most patients with hemorrhagic and ischemic strokes are subject to hospitalization in a specialized neurovascular or neurological department of a city (district) hospital. In the event of a violation of vital functions, they are hospitalized in the intensive care unit, in the case of sub-, epidural, and intracerebral hematomas - in the neurosurgery department. They are transported on a stretcher in a lying position, and patients with a hemorrhagic stroke are transported in a position with the head end of the body slightly raised. If the patient is in a state of deep coma, he is hospitalized only after being brought out of it. On the way to the hospital, a complex of non-differentiated therapy measures are carried out, aimed at normalizing the body's vital functions. The existing methods of treatment of cerebral stroke are generally based on modern ideas about the pathogenetic mechanisms of the development of this disease. They provide for a complex of emergency treatment measures for patients with cerebral stroke regardless of its nature (undifferentiated therapy) and differentiated stroke treatment. Undifferentiated therapy includes measures aimed at the treatment of cardiovascular disorders, prevention and treatment of

respiratory failure, maintenance of normal water-electrolyte balance and acid-alkaline balance in patients in a comatose state, treatment of cerebral edema, and the fight against hypertension. Treatment of acute cardiovascular disorders involves the use of measures aimed at normalizing systemic blood pressure and improving cardiac activity. With a significant increase in blood pressure, one of the following drugs is administered intravenously: dibazol in a dose of 4-5 ml of a 1% solution, pyroxan in a dose of 1-2 ml of a 0.25% solution, clofelin in a dose of 1 ml of a 0.01% solution. They also use diuretics (furosemide 40-60 mg), which are administered intravenously or intramuscularly, depending on the level of blood pressure. (Due to the possible development of the phenomenon of intracerebral theft, the appointment of myogenic vasodilators, such as papaverine, is inappropriate). Hypotensive therapy should be carried out carefully, maintaining blood pressure at the optimal level for each patient. With a sharp drop in blood pressure, glucocorticoids (dexamethasone in a dose of 4-8 mg, prednisone in a dose of 50-120 mg), adrenomimetics (ephedrine, mezaton, dopamine) and caffeine-sodium benzoate are prescribed intravenously. They are injected intravenously in 250 ml of 5% glucose solution, or isotonic sodium chloride solution, or 4% sodium bicarbonate solution. When the activity of the heart is weakened, intravenous strophantin is administered in a dose of 0.25-1 ml of a 0.05% solution or corglycon in a dose of 0.5-1 ml of a 0.06% solution, cordiamine in a dose of 1-2 ml intramuscularly or sulfocamphocaine in a dose of 2 ml subcutaneously. In case of heart rhythm disorders, antiarrhythmic drugs are prescribed. Tactics of their treatment must be coordinated with a cardiologist. Prevention and treatment of respiratory failure. In the presence of respiratory failure, start active oxygen therapy (through a nasal catheter, a mask). In patients with impaired consciousness, measures are taken to maintain the patency of the respiratory tract (suction of mucus, introduction of an airway in the presence of trismus-rotordilator, etc.). In acute II-III degree respiratory insufficiency, tracheal intubation and transfer of the patient to artificial lung ventilation are indicated. Maintenance of normal water-electrolyte balance and acid-base balance in patients in a comatose state. Violation of water-electrolyte metabolism and acid-base balance is the main manifestation of homeostasis disorders in comatose states. To correct these changes, it is necessary to inject 2000-2500 ml of liquid parenterally during the day in 2-3 doses (at least 30 ml/kg of weight). From electrolyte solutions, the most often used isotonic Ringer-Locke solution, glucose-potassium mixtures. To eliminate acidosis, a 4-5% solution of sodium bicarbonate, or lactate, or a 3.6% solution of trisamine is prescribed. With metabolic alkalosis, hypokalemia and hypochloremia are corrected. In addition to electrolyte solutions, the water balance is replenished. As for plasma replacement drugs (polyglukin, reopolyglukin), they should not be used by patients in a comatose state, with intracranial hypertension, or renal failure. Infusion therapy should be carried out under the control of indicators of the electrolyte composition, blood plasma, acid-base status, the content of plasma proteins, urea, residual nitrogen, sugar and other biochemical indicators. Treatment of cerebral edema. Hypertonic solutions, saluretics, and corticosteroid hormones are used to combat cerebral edema. Among hypertonic solutions, osmotic diuretics are quite widely used, which, when administered intravenously, increase the osmotic pressure of the plasma, promoting the flow of fluid from the brain. The osmotic diuretic mannitol is effective, which is prescribed at a dose of 1-1.5 g/kg of body weight per day in the form of a 15-20% solution prepared in isotonic sodium chloride solution or distilled water. Intravenous drip is administered at a rate of 80-90 drops per minute during the day in 2-3 doses. Glycerin (glycerol) is also used - triatomic alcohol, which is prescribed internally at 1-2 g/kg of the patient's weight in a mixture with water or fruit juice in a ratio of 1:2 or 1:3. The intravenous drip drug is administered at a dose of 1 g/kg of body weight during the day in 2-3 doses in the form of a 10% solution prepared in an isotonic sodium chloride solution. However, there are data that do not confirm the effectiveness of the use of glycerin in stroke. Saluretics are also used to combat cerebral edema in stroke. Among the drugs of this group, furosemide (Lasix) is prescribed, which is administered intravenously or intramuscularly in a dose of 2-4 ml of a 1% solution. When using saluretics, it is important to prevent hypokalemia and timely replenish potassium deficiency. To enhance effective dehydration, saluretics can be combined with osmopreparations. A moderate but steady decrease

in intracranial pressure is caused by corticosteroid hormones: dexamethasone at a dose of 16-30 mg or prednisone at a dose of 60-120 mg per day intravenous drip in 200 ml of isotonic sodium chloride solution. Drugs of this group have a stabilizing effect on cell membranes and reduce the production of cerebrospinal fluid. It should be noted that glucocorticoids should not be prescribed for high blood pressure, stomach and duodenal ulcers, and diabetes. In order to reduce CSF hypertension, eufilin is widely used in a dose of 10 ml of a 2.4% solution intravenously twice a day. Antihistamines are also prescribed, which block the action of histamine at the level of cerebral receptors: Suprastin or Diphenhydramine in a dose of 2 ml of a 1% solution intramuscularly. The use of magnesium sulfate 25% solution and glucose 40% solution to combat cerebral edema has a slight dehydrating effect. Fight against hyperthermia. If hyperthermia develops, analgin is prescribed intramuscularly in a dose of 1-2 ml of a 50% solution, lytic mixtures. Cold is applied to the areas of the carotid arteries, axillary, inguinal areas. In order to prevent pneumonia on the first day after a stroke, patients are returned to bed every 2 hours. If pneumonia is suspected, antibiotics are prescribed. It is necessary to prevent bedsores, monitor the emptying of the bladder and intestines. In case of urinary retention - catheterization of the urinary bladder.

Differentiated treatment of ischemic stroke. Treatment measures for ischemic stroke should include: 1) timely and adequate restoration of blood flow in the area of ischemic stroke; 2) correction of rheological and coagulation properties of blood, improvement of microcirculation and collateral circulation; 3) prevention of cascading disorders of cerebral metabolism at various stages of brain infarction formation; 4) reducing the size of irreversible brain damage; 5) increasing the resistance threshold of brain tissue to hypoxia and ischemia.

In the zone of the infarct core, where the blood supply is completely stopped due to vessel blockage, neurons die more often and are unlikely to be saved by any means without immediate removal of the thrombus. But even with surgical restoration of cerebral circulation, the time factor must be taken into account. If more than eight hours elapse between vessel occlusion and thrombus removal, then during this period most neurons even in areas of the brain adjacent to the stroke die. In order to dissolve the thrombus in cerebral vessels and restore blood flow in the ischemia zone in the first hours of an ischemic stroke, thrombolytic agents are used, they can be divided into two groups: exo- and endogenous. Exogenous enzymes include streptokinase and urokinase. They contribute to the transition of plasminogen into active plasmin, which leads to the dissolution of the thrombus. At the same time, these drugs cause a decrease in coagulation factors, especially V and VIII, that is, a state of anticoagulation. Since streptokinase and urokinase remain active in the bloodstream for several hours, there is a danger of hemorrhagic complications. Therefore, thrombolytic agents are not widely used in the treatment of ischemic stroke. Recently, an endogenous tissue-type plasminogen activator has been introduced into the practice of treating brain infarction, which has a more limited effect on thrombus dissolution and does not cause a significant decrease in coagulation factors. The drug remains active in the bloodstream for 10 minutes, so its hemorrhagic effect is less likely than that of streptokinase or urokinase. Now used in the clinic is the preparation of tissue plasminogen activator - actylize, which stimulates fibrinolysis only in the area of the thrombus and does not activate it in the general bloodstream. One of the methods of improving cerebral blood flow is the normalization of the rheological properties of blood by reducing its viscosity. Blood viscosity is influenced by the value of hematocrit, fibrinogen, aggregation properties of platelets, ability of erythrocytes to deform. Of the antiplatelet agents, the most widely used is pentoxifylline (Trental) in a dose of 5 ml (0.1 g) of a 2% solution intravenously by drip or jet with a gradual increase in the dose of the drug to 10-15 ml within 10 days and orally in a dose of 1 tablet (200 mg) 3-4 times a day for three to four weeks. Sermion is also prescribed in a dose of 1-2 ml intravenously for 8-10 days and by mouth in a dose of 1 tablet three times a day, tiklid in a dose of 250 mg twice a day orally during meals. In the treatment of acute ischemic stroke, the method of hemodilution is used - dilution of blood, which is used to improve cerebral blood flow by normalizing the rheological properties of blood and increasing its fluidity. Hemodilution increases the duration of functioning of neurons in the zone of ischemic "penumbra", averts its transformation into brain infarction. In clinical practice,

hypervolemic hemodilution is most often used, for which low molecular weight dextrans (polyglukin, reopolyglukin) are used during the first 5-7 days at the rate of 10 ml/kg of body weight. A single dose of reopolyglukin or polyglukin (400 ml) is administered intravenously. As for cerebral vasodilators, their use can lead to the occurrence of the "robbing" syndrome, that is, increased ischemia of the brain in the area of the lesion. Cerebral blood circulation depends not only on the state of the vascular wall, the composition and properties of blood, but also on the functional state of the heart, indicators of central hemodynamics. Therefore, the justification is the appointment of cardiac glycosides, drugs that regulate blood pressure. Rapid restoration of blood supply to the ischemic brain in the first 6 hours after the development of a stroke (within the so-called "therapeutic window") can block the cascade of pathobiochemical reactions, disorders of cerebral metabolism, and thus prevent the death of the brain substance and reduce the volume of neurological deficit. One of the means of inhibiting the glutamate cascade is to block the release of glutamate by neurons. To achieve it, craniocerebral hypothermia is used. When it is used in an ischemic brain, the imbalance between metabolism and cerebral blood circulation in conditions of oxygen and glucose deficiency disappears. In addition, it has a membrane-stabilizing effect. Calcium channel blockers (CCBs) also improve cerebral blood circulation and have a protective effect in focal brain ischemia. Their use is aimed at preventing or weakening the metabolic cascade, the trigger of which can be an increase in the intracellular content of calcium ions. This is a diverse group of drugs with a significant calcium-blocking effect (nimodipine, flunarizine, nimotop). In particular, nimodipine, nimotop reduce the size of brain infarction, prevent the development of spasm of cerebral vessels, metabolic disorders; nicardipine prevents the development of cytotoxic damage to cortical neurons during reperfusion, and flunarizine has neuroprotective properties. Calcium antagonists have a beneficial effect on the metabolism of neurons in the ischemic "penumbra" zone, and also increase blood flow in the affected hemisphere due to the redistribution of blood from areas of hyperperfusion to the zone of ischemia. A negative effect of nimodipine, nimotop is a decrease in blood pressure. It is not advisable to use them in cases of severe cerebral edema. The effectiveness of drugs of this group requires further study and clarification. When choosing treatment methods for patients with ischemic stroke, it is necessary to take into account that in certain periods of the development of ischemic stroke, the nature of cerebral blood flow changes can be diverse: from significant hypoperfusion to hyperperfusion of brain tissue. Antioxidants are used to increase the threshold of brain tissue resistance to hypoxia in clinical conditions, namely alpha-tocopherol acetate (vitamin E) in a dose of 1 ml intramuscularly, which inhibits the process of lipid peroxidation in the focus of ischemia. The use of hypoxia protectors is justified: Cerebrolysin in a dose of 30 ml intravenous drip in 100 ml of isotonic sodium chloride solution daily for the first ten days, and then in a dose of 5 ml after day No. 5 or nootropil (piracetam) in a dose of 5-10 ml in 100 ml of isotonic sodium solution chloride intravenous drip. Therefore, for the purpose of correcting circulatory and metabolic disorders in acute cerebral ischemia, drugs of various pharmacological groups are used. In the complex treatment of ischemic stroke, hyperbaric oxygenation is also used, especially in the first days after its development. Recently, surgical treatment of ischemic disorders of cerebral blood circulation in the presence of pathology of the main vessels of the head (occlusion, stenosis, pathological tortuosity) has been carried out. Neurosurgical treatment can be carried out at the stage of the stroke itself or immediately after it. Nursing care, early rehabilitation (for 2-4 days), prescribing antidepressants for depression, as well as measures aimed at preventing pulmonary and other somatic complications, pulmonary embolism, are of great importance in the treatment of stroke patients. The rates of recovery of neurological disorders in patients with ischemic stroke are different: with a small ischemic stroke, the recovery of lost functions occurs from the first days, their complete regression is possible on the 7th or 14th day of treatment or in the period up to 21 days. Patients with a completed ischemic stroke are in a moderate or severe condition in the first 7-8 days. Moderate clinical improvement occurs from 10-14 days of treatment. Some increase in the volume of active movements and strength in paretic limbs occurs in the period from 14 to 30 days. Significant improvements may not occur in patients with a severe course of cerebral

infarction. In such cases, stable stabilization of neurological symptoms is observed. Mortality in ischemic stroke is 20% of cases.

Hemorrhagic stroke. Depending on the localization of the hemorrhage in relation to the substance and membranes of the brain, parenchymal, subarachnoid, subdural, and epidural hemorrhages are distinguished. There are also mixed forms of hemorrhages: subarachnoid-parenchymal, parenchymal-subarachnoid, parenchymal-ventricular.

Etiology. Hemorrhage in the substance of the brain (parenchymal) most often develops in hypertensive disease, symptomatic arterial hypertension caused by kidney diseases, pheochromocytoma, as well as in systemic vascular processes of an allergic and infectious-allergic nature. Hemorrhage in the brain can occur with a congenital angioma due to the rupture of an aneurysm. Hemorrhage is much less often caused by atherosclerosis of blood vessels, blood diseases (Werlhoff's disease, leukemia) and other reasons.

Pathogenesis. In the pathogenesis of hemorrhagic stroke, arterial hypertension is of the greatest importance. The severe course of hypertensive disease with crises is the main cause of anatomical changes in intracerebral vessels, as well as vessels of the heart and kidneys. As a result of angiodystonic changes and increased permeability of vascular walls, subendothelial serous infiltration develops first, accompanied by perivascular transudation. Such rapid intramural saturation can cause acute aneurysmal expansion of blood vessels, rupture of structural elements of the wall, its elastic membranes. Plasma saturation of vessel walls can also contribute to their compaction due to fibrinoid substance, microhyalinosis. As a result of fibrinoid-hyalinous degeneration of arterial walls, exfoliating aneurysms can develop, which are the main cause of bleeding when a vessel ruptures. Rupture of arterial and arterial-venous aneurysms can also occur against the background of normal blood pressure. Diapedesis of blood plasma and erythrocytes from small arteries, capillaries and veins due to increased permeability of vascular walls caused by dystonia, hypoxia, stasis, prestasis or intoxication is important in the mechanism of hemorrhage. Hemorrhage through diapedesis occurs in uremia and diseases manifested by hemorrhagic diathesis, etc. Now it is believed that the main mechanism of development of hemorrhagic stroke is the rex mechanism (85% of cases). Hemorrhage by diapedesis occurs in 15% of hemorrhagic strokes.

Pathomorphology. In a hemorrhagic stroke, hemorrhages of the hematoma type and hemorrhagic seepage type are distinguished. A separate group consists of hemorrhages that occur when congenital aneurysms of brain vessels rupture. Hemorrhage centers mainly occur in the basin of the middle cerebral artery. With a hematoma-type hemorrhage, a cavity with clear edges filled with dark liquid and blood clots forms in the area of the internal capsule and subcortical nodes. The area of hemorrhagic seepage occurs as a result of the fusion of multiple small cells, it does not have clear boundaries, it is characterized by the presence of brain matter of a loose consistency, imbibed with elements of blood. In some cases, two or more foci of hemorrhage may occur at the same time.

By localization, lateral hemorrhages are distinguished, which are located laterally from the internal capsule, in the area of the semioval center and occur most often (40%); the second place in terms of frequency (16%) is occupied by mixed hemorrhages that spread to the subcortical nodes, the internal capsule, and the thalamus; Hemorrhages in the visual hump are called medial, they make up 10% of the total number of intracranial hemorrhages. With a medial, as well as a mixed location of the hemorrhagic center, breakthroughs of blood into the ventricular system of the brain may occur. Hemorrhages in the cerebellum occur at autopsies in 6-10%, in the brain stem, mainly in the bridge - in 5% of all hemorrhagic strokes. Haemorrhages in the cerebral cortex and mesentery oblongata, as a rule, do not occur. Very rarely, primary hemorrhages occur in the ventricles of the brain. With significant hemorrhages, swelling of the brain develops, as a result of which dislocation of the brain stem occurs with the development of secondary hemorrhages in it. Such disorders are one of the most frequent causes of death of patients. With hemorrhages, death can also be caused by a massive cell with a breakthrough of blood into the ventricular system of

the brain. The direct cause of death can be cells in the stem part of the brain that destroy vital centers.

Clinic. Hemorrhage into the substance of the brain (parenchymal) begins acutely, without warning, most often during the day, during the period of active activity of the patient, after emotional or physical overload, sometimes - at rest, during sleep. It is characterized by the development of brain-wide and focal symptoms. There is a sharp headache, vomiting, often repeated, sometimes with an admixture of blood. An important diagnostic and prognostic sign is a disturbance of consciousness (from mild stupor to deep coma). Severe vegetative disorders: profuse sweating, hyperemia of the skin of the face and cyanosis of the mucous membranes; the pulse is strained, accelerated or slowed down, blood pressure is increased. Breathing becomes hoarse, like Cheyne-Stokes, with difficulty inhaling or exhaling. Hyperthermia is noted, especially pronounced when blood enters the ventricles of the brain. Pupils are often changed in size, sometimes there is an expansion of the pupil on the side of the hemorrhage. They have no reaction to light. There may be "floating" or pendulum-like movements of the eyeballs, divergent strabismus. The eyes, and sometimes the head, are turned towards the lesion, "the patient looks at the cell." (paresis of gaze). The nasolabial fold on the side opposite the focus is smoothed, the corner of the mouth is lowered, the cheek "sails" when breathing (the "sail" symptom). Raised limbs fall "like whips". However, it is possible to note that on the side opposite to the focus, the tone of the limbs is lower, the foot is turned outward, the Babinski reflex is evoked, although it is quite often bilateral; deep and skin reflexes on are caused. In this condition, involuntary emptying of the bladder or rectum is observed, but retention of urine and bowel movements is also possible. A few hours or on the second day after the hemorrhage, due to swelling of the brain and its membranes, meningeal symptoms appear: stiffness of the muscles of the back of the head, symptoms of Kernig, Brudzinsky, more noticeably expressed in the non-paralyzed leg. The development of bedsores in the areas of the sacrum, buttocks, and heels is possible. A fairly frequent complication of hemorrhage into the brain substance is the breakthrough of blood into the ventricles, which is accompanied by a sharp deterioration of the patient's condition, hyperthermia, respiratory failure, deepening of other autonomic disorders, the development of hormonal syndrome, which is manifested by a constant change in the tone of the limbs, when they go from a state of hypertension with a sharp extension in a hypotonic state. Paroxysmal increase in muscle tone, especially if it predominates in the extensors, is similar to decerebrate rigidity, which is observed not only when blood breaks into the ventricles of the brain, but also when the oral parts of the brainstem are affected (S.M. Davydenkov). With hemorrhage in the right hemisphere of the brain, violent movements of non-paralyzed limbs can be observed - parakinesis, or automated gesticulation syndrome (M.K. Bogolyepov). Hemorrhages in the cerebral hemisphere are often complicated by a secondary stem syndrome. Clinically, this is manifested by a change in the size of the pupils, strabismus, floating or pendulum-like movements of the eyeballs, a violation of muscle tone, and the presence of bilateral pathological reflexes. With hemorrhage in the brain stem, tetraparesis or tetraplegia, alternating syndromes (Weber, Fauville, Millard-Hubler, Benedict), oculomotor disorders, floating movements of the eyeballs, nystagmus, cerebellar symptoms, bilateral pathological reflexes are observed. Hemorrhage in varolii of the bridge is manifested by miosis, paresis of the gaze towards the cell (eyeballs are turned towards the paralyzed limbs). With hemorrhage centers in the lower parts of the brain stem, the functions of breathing and blood circulation are disturbed, muscle hypotonia or atony occurs. Hemorrhage in the cerebellum begins with dizziness, headache in the back of the head, uncontrollable vomiting. Characteristic oculomotor disorders, miosis, Hertwig-Majandi syndrome, which is manifested by divergent strabismus in the vertical plane, and Parino syndrome (vertical paresis of gaze, violation of convergence and reaction of the pupil to light). Nystagmus, slurred speech or dysarthria, muscle hypotonia, ataxia, and stiffness of the neck muscles are also observed. There are no paresis of the limbs. In cases of rapid course of hemorrhage in the cerebellum, focal symptoms "overlap" with general cerebral symptoms. The condition of patients with brain hemorrhage is very serious, most of them die. Death reaches 75-95%. The prognosis of hemorrhages in the ventricles of the brain is

even more difficult. With a favorable course of the disease, patients gradually emerge from a comatose state, which turns into a soporose state.

Subarachnoid hemorrhage mostly occurs as a result of rupture of aneurysms of vessels of the arterial circle of the cerebrum. Physical or emotional stress, blood pressure fluctuations, and angiodystonic disorders contribute to this. Among other causes, congenital defects of vascular walls or systemic vascular diseases (hypertensive disease, atherosclerosis, blood diseases) affecting the vascular system are noted. However, they often occur without any apparent external cause. In younger children, hemorrhage into the subarachnoid space can develop against the background of septic diseases that cause structural changes in the vascular walls. Clinic. Diseases begin acutely, stroke-like, often without warning signs. A sharp headache appears, creating the impression of a dagger blow to the occipital region. At the same time as the headache, dizziness and vomiting occur. There may be loss of consciousness for a short time (from a few minutes, rarely hours). Characteristic psychomotor excitement. For some time, the patient remains disoriented, euphoric, sometimes, on the contrary, lethargic and apathetic. Seizures often occur due to irritation of the cortical motor areas of the brain. After a few hours or on the second day, a meningeal symptom complex develops (rigidity of the muscles of the back of the head, symptoms of Kernig, Brudzinsky, Bekhterev's zygomatic phenomenon, general hyperesthesia). At the basal localization of the hemorrhage, there are signs of damage to certain cranial nerves (ptosis, strabismus, diplopia, paresis of the eye muscles). There are usually no gross focal neurological symptoms. When the hemorrhage spreads to the upper lateral surface of the brain, there may be attacks of Jacksonian epilepsy, monoparesis, aphatic disorders, reflexes of Babinski, Oppenheim, Gordon, Schaefer, which pass quickly. In severe subarachnoid hemorrhage, suppression of tendon and periosteal reflexes is observed. In many patients, the development of symptoms of focal brain damage can be caused by local ischemia caused by spasm of arteries. Spasm of blood vessels often develops on the 3-5th day of the disease and lasts for 2-4 weeks. Arterial spasm in subarachnoid hemorrhage is associated with the direct effect of spilled blood on the sympathetic plexuses of arteries, the toxic effect on the arteries of hemoglobin breakdown products. Among the humoral factors, catecholamines, products of the breakdown of platelets, have spasmogenic properties. Leukotrienes, eicosanoids (prostaglandins, mainly fraction E, thromboxanes) have an antispasmodic effect. Almost always, with subarachnoid hemorrhage, the body temperature rises within 37.5-38° on the 2nd-3rd day of the disease. Hyperthermia is accompanied by moderate leukocytosis and a shift of the white blood formula to the left. The course of subarachnoid hemorrhage is characterized by relapses. They mostly occur 2-4 weeks after the first hemorrhage. In most cases, repeated hemorrhages are due to aneurysm rupture. Their course is more severe and often ends unfavorably. In case of subarachnoid-parenchymal hemorrhage, in addition to the membranes, the process extends to the substance of the brain. They are usually accompanied by persistent focal neurological symptoms. The diagnosis of subarachnoid hemorrhage is made taking into account the stroke-like onset of the disease, the subsequent development of cerebral and meningeal symptoms. The absence of gross neurological deficit is also typical. Lumbar puncture is of decisive importance in diagnosis. Cerebrospinal fluid in the first days has a bloody appearance, flows out under increased pressure. Later, on 3-5 days, it becomes yellowish, xanthochromic, with lymphocytic pleocytosis. On the fundus, it is often possible to detect hemorrhages in the retina, congested discs of the optic nerves. According to the development and clinical course, subarachnoid hemorrhage must be differentiated from meningitis. The study of cerebrospinal fluid solves questions about the etiology of the disease.

Order of the Ministry of Health of Ukraine dated April 17, 2014 No. 275

Unified clinical protocol of emergency, primary, secondary (specialized), tertiary (highly specialized) medical care and medical rehabilitation "Hemorrhagic stroke (Intracerebral hematoma, aneurysmal subarachnoid hemorrhage)"

(extract)

ORGANIZATION AND PROVISION OF MEDICAL AID AT THE PRE-HOSPITAL STAGE

1. The pre-hospital stage includes the provision of first medical and emergency medical aid to patients with HPMK from the moment of identification of the patient or the application of such a patient (patient's relatives or witnesses) for medical help until the moment of hospitalization.

2. Provision of first medical or emergency medical aid at the pre-hospital stage is carried out:

2.1. Emergency (emergency) medical aid teams of Centers of emergency medical aid and disaster medicine, emergency (emergency) medical aid stations, doctors of emergency (urgent) medical aid departments of multidisciplinary hospitals that are part of the emergency medical aid system.

2.2. Brigades of emergency medical care points for adults and children.

2.3. General practitioners - family doctors (in cases of detection of such patients in residences or at the reception), as well as doctors of other specialties.

3. Medical care at the pre-hospital stage should be provided to patients with HPMK in the first minutes from the onset of the development of clinical signs of the disease.

4. For patients with HPMK, it is necessary to ensure urgent hospitalization in the appropriate health centers that provide secondary medical care, in the period of greatest therapeutic opportunities.

5. Rapid diagnosis of clinical signs of HPMK at the pre-hospital stage reduces the time for establishing the diagnosis and transporting the patient to the appropriate healthcare facilities.

6. All patients with suspicion of HPMK, regardless of gender, age and other factors, after providing emergency medical care, are subject to urgent hospitalization in multidisciplinary hospitals (provided the creation of hospital districts - in multidisciplinary intensive care hospitals).

7. In order to ensure the consistency of providing medical care to patients with a diagnosis of HPMK, it is expedient to develop and implement local medical care protocols (LPMD), which define the patient's clinical route and the scope of treatment and diagnostic measures in accordance with material, technical and personnel support in each health care facility. In addition, the interaction between health centers that provide emergency, primary, secondary and tertiary medical care is determined by the order of the territorial authority on health protection and coordinated by a joint agreed Local protocol of medical care (regional level of the local protocol).

EMERGENCY (FAST) MEDICAL AID BRIGADES

Provisions of the protocol

1. The arrival of the emergency (ambulance) medical aid team at the scene of the incident takes 10 minutes in cities, and 20 minutes in settlements outside the city limits from the moment the request is received by the dispatcher of the operational dispatch service of the center of emergency medical aid and disaster medicine. Taking into account meteorological conditions, seasonal features, the epidemiological situation and the condition of the roads, the specified standards may be exceeded, but not by more than 10 minutes.

2. The patient's diagnostic and clinical examination is carried out in full and is recorded in the emergency medical aid exit card (form 110/o). Rationale Early diagnosis and hospitalization of patients with signs of HPMC in specialized healthcare facilities improves the results of treatment of patients with stroke.

Necessary actions of the head of the emergency (urgent) medical care team 15 Mandatory: 1. Collection of anamnesis 1.1. Collection of anamnesis of the disease: 1.1.1. Establish the exact time of the onset of the disease (or the time when the patient was last without symptoms of HPMC); 1.1.2. Record the patient's complaints (presence of headache, nausea, vomiting, impaired speech, sensitivity and active movements in the limbs) at the time of the onset of the disease (if the patient's condition allows), or find out the specifics of the onset of the disease from the words of relatives (witnesses). 1.1.3. The speed of increasing symptoms of the disease; 1.1.4. If there is a history of

stroke, find out which residual neurological disorders persisted and which appeared at the time of examination. 1.2. Collection of life anamnesis (thorough, but quick): 1.2.1. Collect a general allergy history and find out if there are allergic reactions to taking medicines; 1.2.2. Determine what medicines the patient takes daily; 1.2.3. Find out what medicines the patient took before the first signs of the disease developed; 1.2.4. To detect the presence in the anamnesis of disorders of cerebral blood circulation, other accompanying diseases: arterial hypertension, diabetes, diseases of the cardiovascular system, arrhythmia and bad habits.

2. Carrying out a physical examination (thoroughly, but quickly) 2.1. Assessment of the general condition and vital functions: consciousness, breathing, blood circulation according to the ABCDE algorithm (Appendix No. 2). 2.2. In accordance with the indications, eliminate violations of the vital functions of the body - breathing, blood circulation.

3. Assessment of the patient's neurological status (thoroughly, but very quickly, urgently) 1. Level of consciousness according to the Glasgow coma scale (Appendix No. 3). 2. Signs of a stroke according to the scale "Language-Hand-Face-Time" (FAST) (Appendix No. 4).

4. Conducting a laboratory examination: determination of the blood glucose level using a portable glucometer.

5. Instrumental examination

Обов’язково: 1. Визначення артеріального тиску на обох руках; 2. Вимірювання температури тіла; 3. ЕКГ або передача біометричних ЕКГ-сигналів у консультативний телеметричний центр для вирішення термінових питань інтерпретації ЕКГ. Бажані: Пульсоксиметрія (визначення сатурації крові киснем, норма ≥ 95%).

3. Treatment tactics

3.1. Non-pharmacological medical interventions Provisions of the protocol 1. Providing the correct position of the patient's body to prevent aspiration of the respiratory tract. 2. Ensuring patency of the respiratory tract. 3. Oxygen therapy with signs of hypoxia. Rationale In patients with a stroke, the risk of swallowing disorders increases, so it is necessary to prevent aspiration complications and the development of aspiration pneumonia in the future. Necessary actions of the head of the emergency (urgent) medical care team Mandatory:

1. Provide the correct position of the patient's body: on the back, with the head end of the medical stretcher raised 300 or with the help of improvised means.

2. Apply standard methods to restore airway patency and prevent aspiration. 1. To improve the patency of the upper respiratory tract, it is necessary to: 1.1. Ensure free breathing: unbutton a tight collar, prevent throwing the head back or excessive bending of the head); 1.2. Turn the patient's head to the side; 1.3. Remove dentures from the patient's oral cavity. 2. To carry out measures for the prevention of aspiration: 2.1. Carry out rehabilitation of the upper respiratory tract and oropharynx, and if it is ineffective, insert an airway or a laryngeal mask in patients with preserved independent breathing to prevent the tongue from sinking in and facilitate the suction of saliva. If breathing remains inadequate, it is necessary to perform tracheal intubation followed by sanitation of the respiratory tract. In case of significant respiratory insufficiency, transfer the patient to mechanical ventilation (if the patient's condition is severe according to the SHCG score of 8 points and below - intubation and mechanical ventilation).

3. Carrying out oxygen therapy It is advisable to carry out oxygen therapy to patients who have clinical signs of impaired external breathing and/or blood oxygen saturation (saturation) below 95%. 1. In case of loss of consciousness, inhalation of oxygen is mandatory. 2. Ventilate for bradypnea (BP less than 12 per minute), tachypnea (BP more than 35-40 per minute), if peripheral blood saturation is lower than 95%, and cyanosis of the skin increases.

4. Provision of venous access Provision of venous access is carried out by performing a standard venipuncture procedure with adherence to aseptic/antiseptic measures with an intravenous puncture catheter, which is carefully fixed with a bandage. 17 3.2. Provision of emergency medical care Provisions of the protocol 1. Blood pressure control. 2. Determination and correction of blood glucose level. Rationale A sharp decrease in blood pressure can lead to a worsening of the patient's condition, therefore, due to the difficulty of adequately controlling the

rate of blood pressure reduction, the use of antihypertensive therapy in patients with acute cerebrovascular accident at the pre-hospital stage is not recommended in most cases. An uncorrected high or low blood glucose level negatively affects the further course of the disease. Necessary actions of the head of the emergency (urgent) medical care team Mandatory: 1. Control and correction of JSC NB! BP correction is not carried out in case of detection of: BP syst. no more than 220 mm Hg. Art. and/or BP diast. no more than 120 mm Hg. Art. In the case when the patient has a significant increase in blood pressure: blood pressure syst. more than 220 mm Hg. Art. and/or BP diast. more than 120 mm Hg. Art. antihypertensive therapy can be prescribed under BP control. NB! Blood pressure can be reduced by no more than 10-15%. A slow decrease in blood pressure over the course of an hour is shown. Lowering blood pressure can be carried out using the following drugs: Alpha-adrenergic blockers: urapidil; Beta-blockers: esmolol, metoprolol, labetalol; ACE inhibitors enalapril. 2. Infusion therapy 0.9% sodium chloride solution is administered. 3. Correction of glucose level If the blood glucose level is less than 3.0 mmol/l, it is necessary to inject 40-80 ml of 20% glucose solution IV bolus. 4. Convulsive syndrome 1. Diazepam IV, in case of need for repeated administration - intramuscularly or IV drip. 2. Magnesium sulfate (for complex therapy in convulsive syndrome) - to be administered intravenously slowly bolus or drip in a solution of 0.9% sodium chloride. 18 NB! Contraindicated and non-recommended interventions in patients with suspected HPMC: 1. Prescribing acetylsalicylic acid or other antiplatelet therapy before neuroimaging. 2. Sublingual and intramuscular administration of antihypertensive drugs due to an unforeseen pharmacodynamic effect. 3. The use of short-acting nifedipine is contraindicated, since the speed and degree of blood pressure reduction when taking it (especially sublingually) is difficult to control, which increases the risk of ischemia of cerebral or coronary vessels. 4. The use of furosemide is contraindicated for the treatment of brain edema due to a possible sharp decrease in blood pressure and deepening of brain hypoxia. 5. Administration of papaverine hydrochloride is contraindicated. 6. Administration of glucose solutions and other infusion solutions containing glucose is contraindicated. 7. Administration of unfractionated heparin, low molecular weight heparin and heparinoids is contraindicated. 8. Contraindicated use of calcium chloride, ethamsylate, menadione or ascorbic acid to stop bleeding in case of suspected hemorrhagic stroke (they begin to act after a few days, effectiveness in HPMC has not been studied). 9. To date, there is no evidence of the effectiveness of treatment of HPMK with the help of neuroprotective and nootropic drugs, as well as succinic acid, the administration of these drugs at the pre-hospital stage is not recommended. 4. Transportation of the patient to the hospital Provisions of the protocol 1. Urgent transportation of the patient to the designated secondary medical care facility Necessary actions of the head of the emergency (ambulance) medical team Mandatory: 1. The patient is transported in an ambulance with the head end of the medical stretcher raised gurney in a lying position on the back, with the head end raised to 300. (Stretchers are functional and meet state standards: since 10/01/2009, the National Standard of Ukraine DSTU 7032:2009 (EN 1789:2007, MOD) "Ambulances and their equipment" has been put into effect on the territory of Ukraine. 2. Urgent the patient is hospitalized in a health care institution: in the emergency (emergency) medical care department of a hospital, specialized stroke departments, an emergency medical care hospital 19 (or in a multidisciplinary hospital (clinical hospital) of intensive care). 3. Patients with transient ischemic attacks are subject to mandatory urgent hospitalization. 4. The patient is hospitalized accompanied by relatives or representatives (if possible). 5. The head of the medical team must inform the health center about a patient with HPMK who is within the "window of therapeutic possibilities" as a possible candidate for cTLT in the manner agreed upon in the LPMD.

3.1.3. FOR DOCTORS OF EMERGENCY MEDICAL ASSISTANCE CENTERS FOR ADULTS AND CHILDREN POPULATION Provisions of the protocol When receiving a call from a patient (his relatives or witnesses) with complaints that may indicate symptoms of HPMK, a doctor of an emergency medical aid center for adults and children should facilitate the rapid access of such patients to emergency medical care and ensure urgent hospitalization. Necessary actions of the doctor Mandatory: 1. Make an exit on call as a matter of urgency. 2. Carry out a

quick history collection: 2.1. Establish the exact time of the appearance of the first signs of the disease. 2.2. Record the patient's complaints at the time of the onset of the disease (if the patient's condition allows, or find out the specifics of the onset of the disease from the words of relatives (witnesses). 2.3. Collect the general allergic history and find out whether there are allergic reactions to taking medicines. 2.4. To establish what medicines the patient takes daily. 2.5. To find out what medicines the patient took before the development of the first signs of the disease. 2.6. To find out the presence in the anamnesis of disorders of cerebral blood circulation, other concomitant diseases: arterial hypertension, diabetes, diseases of the cardiovascular system, arrhythmias, and bad habits. 3. Conduct an examination of the patient's clinical condition and the necessary instrumental diagnostics. 3.1. Physical examination (thoroughly but quickly): 1. Assessment of the general condition and vital functions: consciousness, breathing, blood circulation according to the ABCDE algorithm (Appendix no. 1). 2. If necessary, provide first aid (or carry out a set of resuscitation measures). 20 3.2. Assessment of the patient's neurological condition (thoroughly but quickly). Signs of stroke using the Language-Hand-Face-Time (FAST) scale (Appendix No. 4). 3.3. Laboratory examination: Desirable: Determination of blood glucose level using a portable glucometer; 3.4. Instrumental examinations: Mandatory: 1. Determination of blood pressure on both hands; 2. Measurement of body temperature. Desired: ECG or transmission of biometric ECG signals to a consultative telemetry center for urgent ECG interpretation issues. 4. Provide medical assistance 1. Provide the correct position of the patient's body: 1.1. The correct position of the patient's body: on the back, with the head end of the body raised by 300, so that the head end is in a stretcher - gurney 1.2. To prevent aspiration, the head must be turned to the side. 1.3. In case of life-threatening respiratory and circulatory disorders, act according to the ABCDE algorithm (Appendix No. 1). 5. To ensure the organization of urgent hospitalization of the patient in a health center that provides secondary (specialized) medical care. 1. Make an emergency medical aid call by calling the single emergency medical aid telephone number 103 or by the single emergency medical aid telephone number 112. 2. Be sure to wait for the arrival of the emergency (ambulance) medical aid team and ensure the monitoring of the state of consciousness and greetings before its arrival functions of the patient, carrying out treatment measures and readiness to carry out resuscitation measures. 3. To save time, provide the head of the emergency (urgent) medical care team with information from the patient's anamnesis and indicate the time of onset of the first symptoms of HPMK. 21 3.1.4.

FOR GENERAL PRACTICE DOCTORS - FAMILY DOCTORS Provisions of the protocol 1. Primary and secondary prevention of stroke development is considered from the standpoint of general prevention of hypertension and consists in identifying and correcting proven risk factors (FR). Necessary actions are determined by the unified clinical protocol of medical care "Hypertension" (2012), approved by the Ministry of Health of Ukraine. 2. Primary medical care is provided by general practitioners - family doctors and district therapists: 2.1. In dispensaries, dispensaries of general practice-family medicine, rural medical dispensaries, paramedic stations, paramedic-midwifery stations (nomenclature according to the order of the Ministry of Health of October 28, 2002 No. 385). 2.1.1. In primary health care centers, paramedic and midwifery centers, paramedic centers in outpatient settings (at a doctor's appointment, in a day hospital) or at the patient's place of residence (stay). Provisions of the protocol The general practitioner-family doctor must facilitate the rapid access of patients with suspected HPM to emergency medical care and urgent hospitalization in the appropriate health care facility. Necessary actions of the doctor Mandatory: Call emergency medical assistance 103 or call the single emergency assistance telephone number 112. 1. Conduct a quick medical history collection: 1.1. Establish the exact time of the appearance of the first signs of the disease. 1.2. Record the patient's complaints at the time of the onset of the disease (if the patient's condition allows, or find out the specifics of the onset of the disease from the words of relatives (witnesses). 1.3. Collect the general allergic history and find out the presence of possible allergic reactions to taking medicines. 1.4. To establish what medicines the patient takes daily. 1.5. To find out what medicines the patient took before the development of the first signs of the disease. 1.6. To find out the presence in the anamnesis of

disorders of cerebral blood circulation, other concomitant diseases: arterial hypertension, diabetes, diseases of the cardiovascular system, arrhythmias, and bad habits. 2. Conduct an examination of the patient's clinical condition and the necessary instrumental diagnostics. 2.1. Physical examination (thorough, but quick): 22 2.1.1. Assessment of the general condition and vital functions: consciousness, breathing, blood circulation according to the ABCDE algorithm (Appendix No. 1). 2.1.2. If necessary, provide first aid (or carry out a set of resuscitation measures). 2.2. Assessment of the patient's neurological condition (thoroughly but quickly). Signs of stroke using the Language-Hand-Face-Time (FAST) scale (Appendix No. 4). 2.3. Laboratory examination: Desirable: Determination of blood glucose level using a portable glucometer; Instrumental examinations: Mandatory: 1. Determination of blood pressure on both hands; 2. Measurement of body temperature. Desired: ECG or transmission of biometric ECG signals to a consultative telemetry center for urgent ECG interpretation issues. 3. Provide medical assistance 3.1. Provide the correct position of the patient's body: 3.1.1. Position of the patient's body: on the back, with the head end raised to 300 with the help of improvised means. 3.1.2. To prevent aspiration, it is necessary to turn the head to the side. 3.1.3. In case of life-threatening respiratory and circulatory disorders, act according to the ABCDE algorithm (Appendix No. 1). 4. To ensure the organization of urgent transportation of the patient to a health center that provides secondary (specialized) medical care 1. Call for emergency medical care by calling the single telephone number for emergency medical assistance 103 or by calling the single telephone number for calling emergency medical assistance 112. 2. Be sure to wait the arrival of the emergency (ambulance) medical team and, before its arrival, to ensure the monitoring of the patient's state of consciousness and vital functions, the implementation of medical measures and the readiness to carry out resuscitation measures. 3. To save time, provide the head of the emergency (urgent) medical care team with information from the patient's anamnesis and indicate the time of onset of the first symptoms of HPMK. 23 3.2.

FOR INSTITUTIONS PROVIDING SECONDARY (SPECIALIZED) AND TERTIARY MEDICAL CARE Organization of medical care provision 1. Interaction between health care facilities that provide emergency, primary, secondary and tertiary medical care is determined by the order of the territorial health authority and is coordinated by a jointly agreed Local protocol medical care (regional level of local protocol). 2. Each health care facility develops and implements its own Local Medical Care Protocol (patient route), which defines the interaction of the structural divisions of primary and secondary care in this health care facility (department), the necessary actions and time during hospitalization, diagnostic procedures, and specialized treatment in stroke patient. 3. Organization of departments for providing specialized care to patients with stroke, which are created in health care facilities, which have in their structure reception departments of emergency medical care, diagnostic, laboratory and other units for providing such care; a staff of specially trained specialists with higher medical (neurologists, anesthesiologists, neurosurgeons) and pedagogical (physical rehabilitation specialists, speech therapists) education, as well as secondary medical education, specially trained to provide assistance to stroke patients. 4. Organization of medical care for patients with hemorrhagic stroke involving specialists of the multidisciplinary team (MTC), which, if necessary, ensures timely provision of neurosurgical care. MDC consists of trained medical and pedagogical staff, provides an interdisciplinary approach to treatment, rehabilitation and prevention (including complications). Participants of the MDC meet regularly (at least once a week) to discuss the patient's problems, determine individual tasks and the scope of rehabilitation interventions, based on the patient's clinical condition and functional capabilities, plan the patient's discharge for further stages of treatment and rehabilitation. Provisions of the protocol 1. All patients with suspicion of HPMC should be urgently hospitalized, regardless of age, gender and severity of the disease. 2. Patients with suspected HPMK are hospitalized in multidisciplinary hospitals (provided hospital districts are created - in multidisciplinary intensive care hospitals). 3. Hospitals that accept patients with suspected HPMK should be able to provide 24/7: 3.1. CT/MRI neuroimaging 3.2. monitoring of welcome functions

24 3.3. laboratory control of homeostasis indicators 3.4. provision of necessary assistance by medical personnel specially trained for the problem of stroke. 3.5. consultation of a neurosurgeon. 4. The available LPMD will allow to organize effective use of time for quick diagnosis and selection of treatment strategy and tactics, will ensure timely provision of neurosurgical assistance to patients with hemorrhagic stroke according to indications. 5. Every medical worker involved in the provision of medical care to patients with hemorrhagic stroke must be guided by the LPMD. Treatment of patients in specialized stroke units reliably reduces mortality, total mortality and disability. Timely neurosurgical treatment of patients with cerebral aneurysms, intracerebral hemorrhages, and AVMs prevents the development of hemorrhagic stroke complications and improves treatment outcomes. The main components of medical care at the hospital stage: 1. Diagnostic examination to determine the type of stroke, location and volume of hematoma, to determine the cause of intracranial hemorrhage. 2. In case of a confirmed hemorrhagic stroke - assessment of the patient's condition and determination of the necessary type of treatment - drug, microsurgical or endovascular. Determination of indications for CT or MR angiography examination, selective cerebral angiography, indications for surgical and/or endovascular treatment of patients with intracranial hemorrhages and their complications (arterial spasm, fluid dynamics disorders). Ensuring the performance of neurosurgical interventions in the presence of appropriate indications. In the absence of conditions for the provision of neurosurgical care - ensuring the timely transfer of patients with intracranial hemorrhages who need it to the appropriate hospitals. 3. If the VMC is not caused by a vascular lesion of the brain (aneurysm, AVM, cavernoma) and does not cause dislocation-compression syndrome and liquefaction disorders, medical treatment is carried out, which includes correction of blood pressure, maintenance of vital functions, correction of disorders of the blood coagulation system, normalization of intracranial pressure, prevention of secondary angiospasm and measures to prevent possible somatic complications. 4. Surgical treatment is carried out according to the specified indications in the presence of dislocation-compression syndrome and fluid-dynamic disorders due to IMC, hemorrhage in the cerebellum or breakthrough of blood in the ventricles of the brain, as well as in case of intracranial hemorrhage due to the rupture of an aneurysm or arterio-venous malformation, in case of hemorrhage into a tumor or cavernous . For surgical treatment, the patient is transferred to the neurosurgery department. If a rupture of a cerebral aneurysm is suspected or if there are fluid-dynamic disorders, transfer to the neurosurgical department is carried out urgently. Types and features of surgical and endovascular interventions are outlined in tertiary care protocols. 5. In the departments where the patient is, constant monitoring of the patient's condition is carried out, necessary medical assistance is provided. With the stabilization of vital functions and the general condition of the patient, rehabilitation measures are provided with the participation of the coordinated work of a multidisciplinary team of qualified specialists. 6. Preparing the patient for discharge and creating an individual plan for the prevention of repeated stroke and correction of identified cardiovascular risk factors 7. Development and provision of a long-term (at least one year) multidisciplinary individual rehabilitation program (IRP) after discharge from the hospital. 3.2.1. Reception department Provisions of the protocol 1. The initial examination of a hospitalized patient with suspicion of HPMC is carried out by a neurologist, if necessary - jointly with an anesthesiologist and a neurosurgeon. 2. In the reception department, the patient's general and neurological condition is assessed, differential diagnosis is made, and further examination and treatment tactics are determined. 3. The reception department must be able to provide 24/7: 3.1. conducting CT/MRI neuroimaging methods; 3.2. carrying out laboratory diagnostics; 3.3. medical and instrumental support for providing emergency care in case of violations of the patient's vital functions; 3.4. technical support for monitoring welcome functions; 3.5. urgent consultation of a neurosurgeon and other necessary specialists. 4. The available LPMD will allow to organize effective use of time for quick diagnosis and selection of treatment strategy and tactics, will ensure timely provision of neurosurgical assistance to patients with intracranial hemorrhages according to specified indications. Rationale A complete neurological examination should be carried out urgently. The clinical picture of a hemorrhagic stroke depends on the location

and volume of the hemorrhage. The most important sign of HPMC is the acute development of 26 general brain and focal neurological symptoms (within minutes, hours). Usually, with a hemorrhagic stroke, the patient's condition is severe, general brain symptoms prevail, and often there is a disturbance of consciousness in the form of pronounced muting, sopor or coma. The formation of hemispheric stroke-hematoma is accompanied by the appearance of contralateral motor and sensory disorders according to the hemitype, aphatic disorders with lesions of the dominant hemisphere. An increase in the volume of a stroke-hematoma causes an increase in the clinical manifestations of intracranial hypertension and the appearance of symptoms of compression of the upper parts of the trunk in the form of increased headache intensity, the appearance of vomiting and deepening of disorders of consciousness, impaired respiratory function and cardiovascular activity. As a result of dislocation and compression of the brain stem, a comatose state - hormetonia can develop within a short period of time without prior manifestation of focal neurological disorders. When the hemorrhage spreads to the upper parts of the trunk or their compression due to dislocation disorders in the acute period, oculomotor and pupillary motor disorders are determined (paresis of vertical gaze, convergence disorder, diverging strabismus, paresis of sideways gaze, anisocoria, mydriasis, lack of reaction of the pupils to light). With hemorrhages in the bridge of the brain, the clinical picture includes a sudden loss of consciousness up to coma, tetraplegia, pronounced decerebration rigidity, miosis with no reaction of the pupils to light, the symptom of "doll's eyes", a decrease in blood pressure, bradycardia. With hemorrhages in the cavernous bridge of the brain, clinical symptoms may be less pronounced and limited to mild paresis of the limbs, disorders of the functions of the cranial nerves. Characteristic symptoms of cerebellar hemorrhage are pain in the occipital region, repeated vomiting, diffuse muscular hypotension, dizziness, decreased blood pressure, bradycardia. With further suppression of consciousness to the point of coma, symptoms of brain stem damage occur: hormetonia, decerebration rigidity. When blood breaks into the ventricular system, the patient's condition deteriorates rapidly within a short period of time: consciousness deepens, hyperthermia develops, hormetonia appears, cardiac activity and vascular tone become labile, breathing disorders occur. Subarachnoid hemorrhage (SAH) is characterized by the sudden onset of a severe headache that patients describe as the worst pain of their lives. At the same time, there is radiation of pain in the cervical and/or lumbar regions of the spine, the appearance of nausea, vomiting, developing meningeal symptoms, photophobia. At the onset of the disease, there may be a loss of consciousness, sometimes a generalized convulsive attack. In the future, recovery of consciousness is most often observed. Repeated loss of consciousness is associated with recurrence of aneurysm rupture. Meningeal symptoms are the leading clinical manifestation of the disease. In 27 most cases, patients with SAH do not have focal neurological disorders. Sometimes in the acute period of SAH, compression of the cranial nerves by an aneurysm is possible, which is accompanied by the appearance of oculomotor disorders. A sudden deterioration of vision due to hemorrhages in the retina or in the vitreous body is quite specific for SAH due to the rupture of an aneurysm

Necessary actions of the doctor of the reception department

Mandatory: 1. 3 history collection: 1.1 Collection of disease history. 1.1.1. The doctor who examines the patient must accurately determine the time of the onset of the disease (if there are no eyewitnesses to the development of stroke symptoms, the time of the development of a stroke should be considered the time when the patient was last seen in a "normal" state - without stroke symptoms). 1.1.2. Determine the complaints (if the patient's condition allows) and the clinical course of the symptoms of the disease. 1.2 Collection of life history. Identification of vascular risk factors for stroke, such as hypertension, cardiac arrhythmias, heart disease, diabetes, myocardial infarction, seizures, or history of CHD. 1.3 Collection of family history. 1.4 Collection of medication anamnesis. List of medications taken by the patient (oral anticoagulants; antiplatelet agents, NSAIDs, antihypertensive agents, statins, etc.) and the presence/absence of drug allergies. 1.6 Collection of information about past injuries, surgical interventions and bad habits (smoking, alcohol abuse, drug use).

2. Physical examination 2.1 Measurement of blood pressure on both arms. 2.2 Heart rate

measurement. 2.3 Measurement of body temperature (T body). 2.4 Measurement of respiratory rate (RB). 2.5 Measurement of body weight. 2.6 Measurement of height, waist circumference.

3. Examination of organs and systems During the examination, special attention should be paid to: 3.1 examination of the head and neck (signs of injury), 3.2 examination of the tongue (bites), 3.3 examination of the legs (edema), 3.4 examination of skin color (jaundice, bruises, petechiae, cyanosis), 3.5 auscultation of the heart and carotid arteries (arrhythmias, murmurs), 3.6 palpation of the pulse on the arteries of the feet.

4. Neurological examination 28 4.1. Peculiarities of conducting a neurological examination in a patient with intracerebral hemorrhage

4.1.1. Determining the degree of impaired consciousness is carried out according to the Glasgow coma scale

4.1.2. Determining the severity of a stroke is carried out using the Stroke Severity Scale (NIHSS) (Appendix No. 5) 4.1.3. Repeated assessments of neurological status using the Stroke Severity Scale (NIHSS) are performed 3 and 6 hours after the patient's arrival at the department, and then daily. Assessment of the dynamics of the neurological condition helps determine the nature of the clinical course of the disease, timely prevent the development of complications and predict the consequences of treatment. Stroke severity points are entered in the medical history according to Appendix No. 6. 4.1.4. Conducting a test to detect swallowing disorders (Appendix No. 7). 4.2. Peculiarities of neurological examination in case of subarachnoid hemorrhage due to rupture of cerebral aneurysms: 4.2.1. Determining the degree of impaired consciousness is carried out according to the Glasgow coma scale (Appendix No. 3) 4.2.2. The assessment of the severity of a patient with aneurysmal SAH is carried out according to the scale of Hunt and Hess (Appendix No. 8), which makes it possible to determine the further tactics of the patient's treatment: I-III stages. – surgical treatment, IV-V centuries. - intensive therapy aimed at stabilizing the patient's condition and preparing for surgical treatment. The expediency of urgent surgery and its type for patients in critical condition (IV-V stage according to the scale of Hunt and Hess) is determined individually, taking into account the factors that cause dislocation- compression or fluid-dynamic disorders (presence of VMC or hydrocephalus). 4.2.3. Repeated assessments of the patient's severity are carried out according to the Hunt and Hess scale after 3 and 6 hours from the moment the patient arrives at the department, then daily (for SAH according to the Hunt and Hess scale, for VMI according to the Glasgow coma scale). 4.2.4. Conducting a test to detect swallowing disorders (Appendix No. 7). 3.2.2.

Diagnostics Provision of the protocol

1. Neuroimaging of the brain is performed for all patients with HPMC as a matter of urgency.

2. When signs of an ischemic stroke are detected during visualization, the necessary actions are taken in accordance with the UCPMD "Ischemic Stroke", approved by the order of the Ministry of Health of Ukraine dated August 3, 2012 No. 602.

3. Laboratory and instrumental examinations are carried out for all patients with HPMK as a matter of urgency in order to confirm the diagnosis of a stroke and determine its type, establish the cause of intracranial hemorrhage for the selection of adequate treatment tactics, the appointment of appropriate therapy, the prevention of possible complications, as well as measures of differentiated secondary prevention of stroke.

4. Differential diagnosis is carried out taking into account clinical symptoms, data of laboratory and instrumental research methods, consultations of necessary specialists.

Rationale 1. Prior notification of the medical institution and proper communication with the radiology department (x-ray diagnostic department) facilitates access to diagnostic neuroimaging methods and saves time for timely provision of further medical care. 2. Urgent neuroimaging (CT or MRI) is the primary diagnostic measure to establish the type of stroke and determine the treatment tactics of a patient with HPMC. Non-contrast CT is the method of choice for urgent neuroimaging. 3. Modern neuroimaging tools make it possible to non-invasively assess the state of cerebral vessels (CT or MR angiography) 4. Digital subtraction angiography (DSA)

remains the "gold standard" in the diagnosis of cerebral vascular pathology and is performed when cerebral vascular pathology is suspected, if CT results or MR angiography inconclusive or negative, as well as, if necessary, choosing a certain method of neurosurgical treatment (microsurgical or endovascular). 5. Neuroimaging examinations of patients with HPMC have priority over routine examinations, as time is of the essence for effective treatment. 6. Laboratory examinations are important for the appointment of adequate therapy in case of detection of homeostasis disorders.

Instrumental examinations (ECG) are important for the diagnosis of acute cardiac (or coronary) pathology, which quite often accompanies HPMC. Necessary actions of the doctor

1. Neuroimaging of the brain 1.1. Carrying out a neuroimaging study of the brain (CT/MRI). 1.2. For patients with meningeal symptoms and no signs of intracranial hemorrhage according to the results of a neuroimaging study, a lumbar puncture is indicated to rule out SAH.

2. Instrumental studies 2.1. EKG. 2.2. Pulse oximetry (monitoring).

3. Laboratory tests 3.1. General blood test (with determination of the number of platelets). 3.2. Determination of glucose level. 3.3. Determination of the MVN indicator (in patients who took warfarin or other anticoagulants). 3.4. AChT, electrolyte composition of blood serum, kidney tests (creatinine and urea in blood serum). 3.5. General analysis of urine.

4. Urgent consultation of a neurosurgeon in case of: 4.1. intracranial hemorrhage according to the results of neuroimaging studies; 4.2. signs of SAH according to the results of a lumbar puncture. If necessary, the patient may be assigned an urgent consultation with a cardiologist and other specialists.

5. Differential diagnosis is carried out taking into account clinical symptoms, data from laboratory and instrumental research methods. If the diagnosis is doubtful, the following diseases should be excluded: 1. ischemic stroke; 2. craniocerebral injury; 3. metabolic disorders (hypo- or hyperglycemia, electrolyte disorders, uremia); 4. tumor lesions of the brain; 5. idiopathic neuropathy of the facial nerve (Bel's palsy); 6. comas of various etiology; 7. acute intoxication (medicines, alcohol, narcotics, other toxins); 8. infectious and inflammatory diseases of the brain and/or its membranes, brain abscess; 9. acute peripheral vestibulopathy; 10. focal neurological deficit after an epileptic attack (Tod's palsy); 11. syncopal state; 12. transient global amnesia; 13. secondary neurological disorders against the background of somatic pathology (pneumonia, heart failure, liver and kidney failure); 14. fluid dynamics disorders.

6. Planned diagnostic actions: 6.1.1. Neurosurgeon (performed when vascular pathology of the brain is detected by the results of neuroimaging studies, without signs of intracranial hemorrhage or in the case of tumor lesions of the brain). 6.1.2. Cardiologist (carried out to identify cardiovascular pathology and, if necessary, prescribe additional laboratory and diagnostic interventions). 6.1.3. Ophthalmologist (conducted to detect changes in the retina (the presence of 31 hemorrhages in the retina can confirm the hemorrhagic nature of a stroke) and congested optic nerve discs (is evidence of intracranial hypertension, and is a contraindication for performing a lumbar puncture). 6.1.4. Speech therapist (conducted to detect speech disorders (aphasia, dysarthria) and/or swallowing (dysphagia), detection of accompanying neurological disorders (dyscalculia, dyspraxia), prevention of secondary speech disorders, provision of primary speech therapy assistance, selection of the necessary communication tool for further communication with the patient in case of detection of aphasia. 6.1.5 Specialist in physical rehabilitation (performed to prescribe: a movement regimen for an immobilized patient to prevent the formation of bedsores, correct positioning of the affected limbs and prevent the formation of pathological postures, conduct a rehabilitation examination and prescribe an individual early rehabilitation program). 6.1.6. If necessary, consultations of a vascular surgeon, endocrinologist, gynecologist, urologist, psychiatrist, etc. can also be carried out. 6.2. Instrumental methods (according to indications): 6.2.1. CT or MR angiography, digital subtraction angiography. 6.2.2. Invasive monitoring of ICH. 6.2.3. Transcranial dopplerography (diagnosis of arterial vasospasm). 6.2.4. X-ray of lungs. 6.2.5. Echocardiography (transthoracic or transesophageal). 6.2.6. Ultrasound of abdominal organs. 6.3. Laboratory studies: 6.3.1. General analysis of urine 6.3.2. Toxicological screening. 3.2.3.

Treatment Treatment of hemorrhagic stroke can be effective only if the cause of hemorrhage is quickly established using modern instrumental (non-invasive and invasive) and laboratory diagnostic methods. Positive dynamics in the clinical course of the disease (improvement of the general condition, regression of meningeal symptoms, etc.) after prescribing drug treatment without establishing the cause of hemorrhagic stroke cannot be a criterion for evaluating its effectiveness and a favorable prognosis.

Protocol provisions: Treatment of patients with hemorrhagic stroke includes:

I. Basic therapy

II. Specific (differentiated) therapy, including

III. Intensive therapy of patients who are in critical condition 32 IV. Surgical treatment Rationale 1. Effective basic therapy improves the general condition of the patient and affects the effectiveness of specific therapy and neurosurgical treatment. 2. Medicinal treatment of hemorrhagic stroke (basic, specific and intensive therapy) is aimed at normalizing and maintaining the functioning of organs and systems, eliminating the consequences of brain tissue damage and their recovery, preventing the development of complications of the disease, including somatic ones, and optimizing the conditions of neurosurgical treatment in case of it necessity 3. All patients with acute cerebrovascular accident who are in serious condition are subject to intensive therapy. These include all patients with impaired consciousness by the type of coma, as well as patients in whom the course of HPMC is accompanied by the development of clinically significant complications, both on the part of the CNS and on the part of other organs and systems. 4. Ruptures of arterial cerebral aneurysms, hemorrhages in the ventricular system and in the posterior cranial fossa with the development of fluid-dynamic disorders and, in certain cases, intracerebral hematomas require immediate neurosurgical treatment aimed at eliminating the source of hemorrhage and controlling intracranial hypertension. 5. To determine the surgical tactics for intracranial hemorrhages, the feasibility of dynamic observation of the patient's condition and the course of the disease in the conditions of a neurosurgical hospital is possible. Provision of specialized neurosurgical care is carried out in third-level hospitals. In all cases, after instrumental confirmation of a hemorrhagic stroke, patients should have a consultation with a neurosurgeon.

І. БАЗИСНА ТЕРАПІЯ Необхідні дії 1. Підтримка дихальної функції та захист дихальних шляхів. 2. Підтримка серцево-судинної функції та корекція АТ. 3. Контроль та корекція водно-електролітного балансу. 4. Корекція рівня глюкози. 5. Корекція температури тіла (пахвова температура вище 380 С). − Призначення нестероїдних протизапальних лікарських засобів. − Призначення парацетамолу, метамізолу натрію (при значній гіпертермії внутрішньовенно), антигістамінних засобів короткої дії. − Призначення фізичних методів охолодження (обертання пацієнта холодними простирадлами, холод на магістральні судини, заливання холодних розчинів в шлунковий зонд). NB! Пацієнти з геморагічним інсультом, у яких розвивається гіпертермія, мають гірші шанси на виживання і відновлення неврологічного дефіциту. 33 Гіпотермія знижує метаболізм мозку, церебральний кровоток і об'єм крові мозку. Помірна гіпотермія (33-350 С) може використовуватись тільки в спеціалізованих центрах за наявності спеціального обладнання для охолодження крові. Лікувальна гіпотермія не була систематично досліджена у пацієнтів з ВМК. NB! Методи зовнішнього охолодження слід застосовувати з обережністю, тому що вони часто супроводжуються виникненням м'язового тремтіння, яке призводить до підвищення потреби мозку у кисні. 6. Забезпечення адекватної оксигенації крові 7. Призначення протиепілептичних препаратів пацієнтам із судомними нападами.

II. SPECIFIC (DIFFERENTIATED) THERAPY Necessary actions 1. Control of blood coagulation disorders. 2. Correction of hemostasis in patients who have taken anticoagulants and antiplatelet drugs and have elevated MVN - their withdrawal, followed by the administration of intravenous vitamin K preparations, prothrombin complex concentrate, and fresh frozen plasma. 2.1. Low molecular weight (in prophylactic doses) or unfractionated heparin in low doses (5000 IU every 8-12 hours) should be prescribed to bedridden patients from the 1st to the 4th day - provided that the intracerebral bleeding has stopped, which is confirmed by the absence of an

increase in the size of the hematoma according to the CT scan/ MRI dynamics. 2.2. Patients with a high risk of thromboembolism (presence of permanent or persistent atrial fibrillation in patients over 60 years of age, with accompanying diabetes, hypertension, heart failure) resume warfarin intake from 7-14 days - in the absence of a diagnosed source of bleeding (aneurysm, AVM) . 2.3. For the prevention of venous thromboembolism and DVT - wearing elastic stockings in combination with intermittent pneumatic compression. 3. To correct intracranial hypertension, the following is performed: • ICP monitoring (according to indications) • Adequate respiratory support, • Administration of hypertonic saline solutions, osmodiuretics, • Installation of ventricular CSF drainage in case of ICH, etc. (see section III.1.2 "Correction of intracranial hypertension") . 4. Prevention and treatment of secondary arterial vasospasm in case of aneurysmal subarachnoid hemorrhage (see sections III.1.4 and III.1.5).

III. ІНТЕНСИВНА ТЕРАПІЯ ПАЦІЄНТІВ З ГЕМОРАГІЧНИМ ІНСУЛЬТОМ Положення протоколу 34 Основним спрямуванням інтенсивної терапії є підтримка порушених функції органів та систем, а також заміщення їхніх втрачених функцій. Найбільш ефективною є інтенсивна терапія, за допомогою якої досягають необхідної мети з використанням мінімальної кількості медикаментозних та інших засобів. При підозрі на підвищення ВЧТ діагностичні зусилля повинні бути спрямовані на встановлення його причини (неврологічне обстеження, КТ, МРТ) і на уточнення його ступеня виразності. При відсутності об'ємного внутрішньочерепного процесу або гідроцефалії, які потребують невідкладного нейрохірургічного втручання, зниження ВЧТ проводять медикаментозними методами. Обґрунтування Критеріями ефективності інтенсивної терапії є швидкість корекції клінічно значимих порушень функцій органів та систем та стабільність підтримки цих функцій у пацієнтів, що перебувають в критичному стані. Дієвими засобами зниження ВЧТ за відсутності гіпернатріємії вважають осмотично активні препарати, зокрема, манітол, комбіновані розчини електролітів, що містять сорбітол, гіпертонічний розчин натрію хлориду у комбінації з іншими препаратами. Протипоказами для використання осмотичних діуретиків є гіперосмолярність плазми. Необхідні дії 3.1. Проведення інтенсивної терапії у пацієнтів, які перебувають у коматозному стані 3.1.1. Діагностика внутрішньочерепної гіпертензії (при можливості інвазивний моніторинг ВЧТ у пацієнтів з порушеннями свідомості менше 8 балів за ШКГ) 3.1.2. Корекція внутрішньочерепної гіпертензії: 1. Основні дії лікаря наведені в Алгоритмі корекції внутрішньочерепної гіпертензії (ст. 46). 2. NB! Алгоритм використовується у пацієнтів із встановленим контролем над дихальними шляхами (інтубація чи трахеостомія), які перебувають на ШВЛ і в яких виключена можливість неврологічного погіршення внаслідок гіпоксії, гіперкапнії, артеріальної гіпотензії та відсутній внутрішньочерепний крововилив або, які потребують хірургічного втручання. Кожен наступний крок алгоритму здійснюють при неефективності попереднього. 3. Положення головного кінця ліжка під кутом 300 , помірна гіпервентиляція, використання внутрішньовенних анестетиків. 4. Призначення манітолу, комбінованих розчинів електролітів, що містять сорбітол, гіпертонічного розчину натрію хлориду за відсутності гіпернатріємії (Na+ не вище 145-148 ммоль/л). 4. Виведення ліквору через вентрикулярний дренаж; 35 Покази для моніторингу ВЧТ: 1. Пацієнти з ВМК, у яких оцінка тяжкості стану за ШКГ менше 8 балів. 2. Пацієнти з ознаками внутрішньочерепної гіпертензії, які не потребують нейрохірургічного лікування (ВМК з ознаками дислокації, набряку головного мозку, компресії базальних цистерн при нейровізуалізації, ліквородинамічними порушеннями), у яких неможливо оцінити неврологічний стан внаслідок проведення глибокої седації та нервово-м'язової блокади. Методи вимірювання ВЧТ: вентрикулярний, паренхіматозний, субдуральний або епідуральний датчики. 3.1.3.Діагностика вазоспазму при САК • Поява нової та/або поглиблення неврологічної симптоматики (починаючи з 4- 6 доби від початку захворювання); • Поява вогнищ ішемії в головному мозку при нейровізуалізації, зниження регіонального МК за даними перфузійних режимів КТ або МРТ; • Прискорення лінійної швидкості кровоплину по мозкових артеріях за даними транскраніальної допплерографії; • Ознаки спазмування

мозкових артерій за даними КТ/МР – ангіографії або цифрової субтракційної ангіографії (ЦСА). 3.1.4. Лікування вазоспазму при САК: • забезпечення нормоволемії; • застосування німодипіну ентерально під контролем артеріального тиску; • проведення аналгоседації (пропофол, фентаніл, дексмедетомідин під контролем АТ) у пацієнтів, які перебувають на ШВЛ; • контрольована артеріальна гіпертензія (якщо дозволяє стан серцевої функції і артеріальний тиск відповідає нормальним величинам). Нейрохірургічні ендоваскулярні методики застосовуються для лікування вазоспазму у випадку появи відповідної неврологічної симптоматики при неефективності специфічних інфузійних лікувальних заходів в комплексі інтенсивної терапії. Ендоваскулярні операції балонної ангіопластики застосовуються при сегментарному вазоспазмі, а селективне інтраартеріальне введення вазодилятаторів (фармангіопластика) виконують при дифузній формі вазоспазму. 3.1.5. Особливості анестезіологічного забезпечення при хірургічному лікуванні 1. Введення в наркоз із застосуванням анестетиків із коротким періодом напіввиведення: тіопентал натрію, пропофол, дексмедетомідин (при відсутності артеріальної гіпотензії), оксибутират натрію (за наявності артеріальної гіпотензії) у пацієнтів з неглибоким порушенням свідомості (за ШКГ > 8 балів) до операції. 2. Введення в наркоз - індукція "швидкої послідовності" використовується у пацієнтів не натще (після прийому їжі). • Анестетики (один з перерахованих): тіопентал натрію, пропофол; 36 •Анальгетики: фентаніл (не менше, аніж за 3 хвилини до інтубації при наявності артеріальної гіпертензії). 3. Проведення міорелаксації: • недеполяризуючими релаксантами (рокуронію бромід); • преведення прекураризації (для попередження підвищення ВЧТ) у випадку застосування деполяризуючих міорелаксантів (суксаметоній). NB! Ретельний контроль АТ на етапі інтубації у пацієнтів із розривами артеріальних аневризм! Підтримання анестезії • Анестетики: тіопентал натрію, пропофол, інгаляційні - севофлуран • Анальгетики: фентаніл. Підтримання міорелаксації • Піпекуронію бромід або інший недеполяризуючий релаксант. NB! На етапах втручання до розрізу твердої мозкової оболонки характерно підвищення ВЧТ та підвищення АТ на фоні гіповолемії. 3.1.6. Контроль та корекція параметрів гемодинаміки Обов’язково: 1. Корекція підвищеного АТ: 1.1 Адекватна анестезія та аналгезія. 1.2. Зниження АТ 1. Якщо АТ сист.> 200 мм рт.ст. або середній АТ > 150 мм рт.ст., слід розглядати активне зниження АТ з безперервною внутрішньовенною інфузією з частим моніторуванням АТ кожні 5 хв. 2. Якщо АТ сист.> 180 мм рт.ст. або середній АТ > 130 мм рт.ст. та існує ймовірність підвищеного ВЧТ слід розглядати моніторинг ВЧТ та зниження артеріального тиску з використанням періодичного або безперервного внутрішньовенного введення при збереженні церебрального перфузійного тиску > 60 мм рт.ст. 3. Якщо АТ сист.> 180 мм рт.ст. або середній АТ становить> 130 мм рт.ст. і немає підвищення ВЧТ, слід розглядати доцільність помірного зниження АТ (наприклад, середній АТ – 110 мм рт.ст. або цільового АТ 160/90 мм рт.ст.) з використанням перервної або безперервної внутрішньовенної інфузії для контролю АТ з проведенням клінічної оцінки стану пацієнта кожні 15 хв. 1.3. Для зниження АТ застосовується внутрішньовенне введення урапідилу, есмололу, еналаприлу. NB! Після розтину твердої мозкової оболонки можливе зниження ВЧТ при цьому на фоні гіповолемії можливе зниження АТ до критичного рівня. 2. Контроль ЧСС, центрального венозного тиску (ЦВТ); Бажано: Проведення інвазивного моніторингу АТ. 3.1.7. Гемодинамічна та вентиляційна підтримка у періопераційному періоді 37 Проведення катетеризації центральної вени, а при наявності масивної кровотечі ще й периферичної вени, до або під час інтубації трахеї. Завчасне приготування розчину вазопресору. Забезпечення помірної гіпервентиляції чи нормовентиляції ($PaCO_2$ - 30- 35 мм. рт.ст.). Проведення агресивної корекції артеріальної гіпотензії (за наявності сист. АТ < 120 мм рт.ст. або серед. АТ < 70 мм рт.ст.) (бажано у хворих в критичному стані проводити інвазивний контроль АТ): − Проведення інфузійної терапії (розчини натрію хлориду, за відсутності гіпернатріємії; розчини колоїдів – 10-20% альбумін, розчини гідроксиетильованого крохмалю) під контролем ЦВТ (ЦВТ < 60 мм вод.ст.); − Призначення вазопресорів (допамін, фенілефрин, норепінефрин (норадреналін)).

Призначення для корекції підвищеного АТ бета-адреноблокаторів, блокаторів кальцієвих каналів, внутрішньовенних інгібіторів АПФ (диференційований підхід до корекції артеріальної гіпертензії). 3.1.8. Підтримання респіраторної функції 1. Покази для ШВЛ у пацієнтів з геморагічним інсультом: • порушення свідомості за ШКГ 8 балів і вище; • апное, • тяжкі порушення ритму дихання (брадипное, Чейн-Стокса, Біота, гаспінг), • стійка задишка > 30 дихань/хв., • гіпоксемія (ціаноз, PaO2 ≤ 80 мм.рт.ст. При відсутності газоаналізатора показом є SpO2 < 92%), • гіперкапнія (PaCO2 ≥ 50 мм рт.ст.). 2. Проведення ШВЛ у протективному режимі (Vt=6-7 мл/кг, позитивний тиск в кінці видиху (ПТКВ). NB! Без необхідності не збільшувати ПТКВ вище за 5 см вод.ст., щоб не підвищувати ВЧТ. Трахеостомія у випадку, якщо не прогнозується переведення пацієнта на спонтанне дихання, впродовж найближчих 3-4 днів. 3. Моніторинг показників респіраторної функції легень у пацієнтів, які перебувають на ШВЛ: 1. контроль PaO2 та PaCO2, 2. аускультація легень, 3. призначення рентгенографії легенів не рідше, ніж 1 раз на 3 доби – протягом першого тижня, далі за показами. 4. капнографія (EtCO2 35-45 мм рт.ст.). 3.1.9. Респіраторна підтримка: • Підтримка PaO2 не менше 100 мм рт.ст. чи SpO2 не менше 98% (використання ПТКВ, при потребі – використання підвищеного FiO2); 38 • За умови рефрактерної до інших засобів медикаментозної терапії ВЧГ - проведення короткочасної помірної гіпервентиляції (PaCO2 30- 33 мм рт.ст.). NB! Гіпервентиляція протипоказана у пацієнтів з ВМК, ускладненим розвитком вазоспазму та судомними нападами. • Застосування аналгоседації для адаптації до ШВЛ (при необхідності). III.2. Підтримання нормоволемії (контроль за добовим балансом рідини, ЦВТ, неінвазивні та малоінвазивні методи контролю центральної гемодинаміки). III.3. Підтримання нормоглікемії (у хворих на цукровий діабет доцільно підтримувати незначну гіперглікемію до 8-8,5 ммоль/л). NB! особливо слід уникати гіпоглікемії (рівень глюкози крові < 2,5 ммоль/л) III.4. Повноцінне якісне харчування (при необхідності через назогастральний зонд) – 3000-3500 ккал на добу.

IV. SURGICAL TREATMENT 1. Neurosurgical interventions for hemorrhagic stroke. Provisions of the protocol. Patients with the following diagnoses are subject to transfer to the neurosurgical department: - subarachnoid hemorrhage; - intracerebral hemispheric and cerebellar stroke-hematomas, membrane hematomas and ventricular hemorrhages caused by pathological changes in cerebral vessels (aneurysms, arterio-venous malformations, cavernous angiomas, neoplasms) - intracerebral stroke-hematomas (hemispheric, cerebellar), including ventricular hemorrhages, with for which neurosurgical treatment is indicated Rationale Surgical interventions for cerebral aneurysms and AVMs are performed in neurosurgical departments equipped with appropriate modern diagnostic equipment (CT or MRI, digital subtraction cerebral angiography, transcranial dopplerography), equipment for microsurgical operations (operating microscope for neurosurgical interventions, microsurgical instruments , an angiograph with a configuration for neurointerventional interventions) and necessary medical consumables (clips, spirals, catheter systems, adhesive embolizing compositions, etc.). The acquisition of a sufficient level of appropriate theoretical and practical training of specialists is of primary importance in achieving positive results during surgical or endovascular treatment of this pathology. Timely surgical removal of aneurysms and AVMs from the blood circulation allows to prevent repeated intracranial hemorrhages and to carry out active infusion therapy aimed at preventing arterial spasm and its consequences. Contraindications for transfer of patients to the neurosurgical department: 1. Severity of the condition according to SHKG 5 points or less. 2. Accompanying chronic somatic pathology with gross violations of vital functions. 3. Terminal condition of the patient. Types of urgent neurosurgical interventions in case of hemorrhagic stroke 1. Removal of a stroke-hematoma of supra- and subtentorial localization in the presence of dislocation-compression syndrome and liquefaction disorders: - in the absence of cerebral vascular pathology as an etiological factor (aneurysm, AVM, cavernoma) removal of hematoma of the cerebral hemispheres brain and cerebellum; - in the presence of cerebral vascular pathology as an etiological factor (aneurysm, AVM, cavernoma), surgical treatment includes removal of hematoma and elimination of the cause of hemorrhage (clipping or endovascular embolization of aneurysm,

removal of AVM or cavernoma). 2. External drainage of the ventricular system during the development of fluid-dynamic disorders: - intraventricular or parenchymal-ventricular hemorrhage, according to indications of ventricular fibrinolysis; - hemorrhage in the trunk structures and cerebellum; - acute aresorptive hydrocephalus after SAH. 3. Exclusion of arterial aneurysms in the acute period of SAH: - microsurgical (reconstructive or deconstructive); - endovascular (reconstructive or deconstructive); - combined types of treatment. Planned stroke-hematoma removal in the presence of cerebral vascular pathology (AVM, cavernoma) and elimination of the cause of hemorrhage (AVM or cavernoma removal) - compensated dislocation syndrome and controlled intracranial hypertension; Observation, medication, symptomatic therapy – stroke-hematomas of supra- and subtentorial localization, in which surgical treatment is not indicated; Exclusion of AVM (after hemorrhages in which urgent intervention is not indicated): - microsurgical; - endovascular (superselective embolization); - radiosurgical irradiation; - combined types of treatment. Surgical treatment of hemorrhagic stroke complications (arterial vasospasm, occlusive or aresorptive hydrocephalus) 40 In the case of the development of symptomatic segmental vasospasm resistant to specific measures of intensive therapy - endovascular balloon angioplasty operation, in the case of diffuse vasospasm under similar conditions - selective intra-arterial pharmangioplasty. The presence of progressive hydrocephalus (occlusive or aresorptive), resistant to drug therapy and repeated removal of SMP requires valvular bypass surgery or endoscopic ventriculocisternostomy of the III ventricle. Prevention of the development of a repeated stroke Provisions of the protocol 1. All patients who have suffered a stroke should receive educational information about the danger of developing a repeated stroke, signs and symptoms of the onset of the disease, and measures to be taken when they develop. 2. The leading role in the secondary prevention of a stroke is played by the correction of the patient's risk factors. 3. Drug prevention and correction of identified risk factors, which was started immediately after a vascular event, can reduce the frequency of recurrent stroke, disability and mortality. 4. If, after neurosurgical operation, the aneurysm or AVM is not completely excluded from the bloodstream, it is necessary to carry out repeated similar or additional interventions to eliminate the risk of repeated hemorrhagic stroke. Rationale Patients who have suffered a hemorrhagic stroke in the form of VMC have a high risk of recurrent stroke, medication, prevention and correction of individual risk factors reduce the frequency of recurrence of the disease. Patients who did not succeed in completely eliminating the cause of hemorrhagic stroke by surgical means need the supervision of a neurosurgeon; according to the indications, the sequence and technique of surgical interventions aimed at the radical exclusion of an aneurysm or AVM is determined. Even after the complete exclusion of the aneurysm or AVM by the endovascular method and after the complete obliteration of the AVM by the radiosurgical method, the risk of recurrence of the disease remains and, accordingly, the risk of intracranial hemorrhage, which determines the need for observation by a neurosurgeon. 4.1.1. Non-medicinal methods of prevention 1. Identification and correction of risk factors for the development of a repeated stroke. 2. All risk factors for cerebrovascular diseases should be aggressively corrected with both pharmacological and non-pharmacological measures to ensure their optimal control. Recommendations for the correction of other factors of the development of a repeated stroke 41 Mandatory: 1. Smoking Those patients who have a habit of smoking should be recommended to completely quit. 2. Correction of hypercholesterolemia and glucose level 1. Recommend adherence to the principles of a healthy diet and lifestyle correction to maintain the level of cholesterol 5.0 mmol/l and LDL cholesterol >3.0 mmol/l), which is not amenable to non-pharmacological correction, it is possible to prescribe statins an individual regimen for pharmacological correction of hypercholesterolemia. 4.1.3 Surgical methods for the prevention of repeated stroke. Provisions of the protocol. Patients with a history of hemorrhagic stroke must be referred to a vascular neurosurgeon for consultation. If CT/MRI or CT/MRI angiographic changes suggestive of an aneurysm, AVM or cavernoma are detected, the patient must be consulted by a neurosurgeon. During an outpatient examination, general practitioners or neurologists may observe symptoms or identify data from the anamnesis that may be a clinical manifestation of a

cerebrovascular disease that threatens the development of a hemorrhagic stroke. If these signs are detected, it is advisable to consult a neurosurgeon to determine the indications for additional instrumental studies (non-invasive - MRI, MRI angiography, CT angiography, invasive - digital subtraction cerebral angiography). The following symptoms may occur most often: 1. An attack of severe headache in the anamnesis, which cannot be compared with any other in its severity and which was accompanied by nausea and vomiting, phono- or photo-phobia, pain in the neck-occipital region, possibly a short-term loss consciousness – a clinical manifestation of SAH; 2. Transient or persistent oculomotor disorders (arterial aneurysms of paraclinoid localization, cavernous sinus, vertebro-basilar basin); 3. Pulsating noises in the head (cerebral AVMs, dural AVMs); 4. Epileptiform syndrome (AVM, cavernoma). 5. All patients who underwent surgical treatment for a hemorrhagic stroke require constant observation by a neurologist and continued medical control of stroke risk factors (hypertension, diabetes, hypercholesterolemia, etc.). 6. After the surgical treatment of arterial aneurysms or AVMs of the brain, the neurosurgeon determines the terms and type of instrumental diagnostic measures to control the possible recurrence of the disease, or to carry out repeated operations in case of incomplete exclusion from the circulation of the source of hemorrhage. 43 Extract and recommendations Provisions of the protocol 1. All stroke patients who are discharged from the hospital should be given individual recommendations on the prevention of recurrent stroke and rehabilitation. 2. After discharge, patients must be under the constant supervision of a family doctor at their place of residence, continue to follow the instructions of the MDC and be under the dispensary supervision of a neurologist. 3. After surgical treatment, the patient is under the supervision of a neurosurgeon to control the effectiveness of excluding the source of the hemorrhagic stroke, to determine the terms for conducting control angiographic studies and repeated operations in the case of incomplete exclusion of vascular lesions from the blood flow, as well as to monitor the development of fluid-dynamic disorders for their timely correction. Rationale Discharge planning and provision of an individual rehabilitation and stroke prevention program have a positive effect on the results of treatment and the effectiveness of the patient's further rehabilitation. Necessary actions of the doctor Mandatory: 1. Assess the patient's degree of independence in everyday life according to the modified Rankin scale. 2. Provide the patient with an individual plan (program) of secondary prevention measures and correction of risk factors upon discharge. 3. Provide the patient with recommendations on rehabilitation measures to be performed at the secondary outpatient level of medical care. 4. Give the patient after a stroke a printed "Information sheet for a patient who has suffered a stroke" (Appendix No. 10). 5. Involve the patient and their caregivers in participating in programs for non-pharmacological correction of stroke risk factors (if available) - Health schools, "Life after a stroke" School, smoking cessation programs, psychological and physical rehabilitation programs, etc. Desired: 1. Fill out the "Card of a patient with an acute cerebral circulation disorder"

Order of the Ministry of Health of Ukraine dated August 3, 2012 No. 602

"On the approval and implementation of medical and technological documents on the standardization of medical care for ischemic stroke"

(extract)

Prevention

Prevention of recurrent stroke

Provisions of the protocol

1. All patients who have suffered a stroke should receive educational information about the danger of developing a second stroke, the signs and symptoms of the onset of the disease and the actions that should be taken.

2. Patients after ischemic stroke or TIA have an increased risk of recurrent stroke.

3. Drug prevention and correction of identified risk factors, which was started immediately after a vascular event, can reduce the frequency of recurrent stroke, disability and mortality.

Justification

Patients who have suffered a stroke have a high risk of a second stroke, the appointment of

drug prophylaxis and correction of individual risk factors reduces the frequency of the development of a second stroke.

Medical methods of prevention:

Necessary actions of the doctor

Mandatory:

Appointment of antiplatelet therapy

1. All patients with II immediately after exclusion of a hemorrhagic stroke with the help of neuroimaging, but no later than 48 hours. from the onset of the disease, acetylsalicylic acid should be prescribed in a dose of 160-325 mg (in the absence of dysphagia - per os, in the case of dysphagia - in a nasogastric probe, intravenously or per rectum).

2. ASA therapy in the specified dose should be continued for 2 weeks, after which the dose can be reduced to prophylactic (usually 75-100 mg per day). Instead of ASA, another antiplatelet agent (clopidogrel or a combination of ASA with modified-release dipyridamole) can be prescribed for long-term secondary prevention.

3. All patients with TIA should receive 160-325 mg of ASA per day as soon as possible, unless contraindicated.

Appointment of anticoagulant therapy

1. Long-term treatment with anticoagulants is prescribed for patients with cardioembolic II, especially against the background of atrial fibrillation. It is not recommended to prescribe a combination of anticoagulants and antiplatelet agents for a long time, unless there are special indications (for example, stenting).

2. The standard drug is warfarin.

3. The dose of warfarin is selected individually, under the control of the Ministry of Health.

4. The ESR should be monitored regularly and maintained at the level of 2.0-3.0.

Prescribing statins

1. Patients with acute stroke who received statins before the vascular event should continue to take the drug in the future.

2. Prescribing statins is recommended primarily for patients with atherothrombotic ischemic stroke to reduce the risk of recurrent vascular events.

3. Patients with II or TIA with elevated cholesterol levels should be treated with statins.

1.1. If a patient with II has CHD, the target LDL cholesterol level should not exceed 2.7 mmol/L (100 mg/dL).

1.2. In patients with II who have a combination of multiple risk factors (eg, CHD and DM), the target cholesterol (LDL) level should be less than 1.8 mmol/L (70 mg/dL).

2. Appointment recommendations:

2.1. The appointment and selection of the medicinal product is carried out in accordance with the relevant medical and technological documents.

2.2. Statins are prescribed once preferably with an evening meal or twice (morning and evening)

Appointment of antihypertensive therapy

If there are indications, antihypertensive drugs and statins should be prescribed to almost all patients with II before discharge from the hospital in accordance with the medical and technological documents that regulate the provision of medical care for arterial hypertension.

Non-medicinal methods of prevention

1. Identification and correction of risk factors for the development of a repeated stroke.

2. All risk factors for cerebrovascular diseases should be aggressively corrected with both pharmacological and non-pharmacological measures to obtain optimal control.

3. It is recommended to use the ABCD and ABCD-2 scale to identify patients at high risk of stroke (after TIA) (Appendix No. 14, Appendix No. 15).

Recommendations for the correction of other factors of the development of a repeated stroke

Mandatory:

1. Smoking

Those patients who have a habit of smoking should be advised to give it up completely.

2. Correction of hypercholesterolemia.

1. Recommend adherence to the principles of a healthy diet and lifestyle correction to maintain the level of cholesterol <5.0 mmol/l and low-density lipoprotein cholesterol (LDL cholesterol) <3.0 mmol/l.
2. Consider the feasibility of prescribing pharmacological correction of hypercholesterolemia with statins to patients who have high and very high SSR and cholesterol metabolism disorder that is not amenable to non-pharmacological correction.
3. For high- and very high-risk patients, it is recommended to maintain the level of cholesterol <4.5 mmol/L and LDL-C <2.5 mmol/L.

3. Body weight control

1. Patients with excess body weight should be recommended to follow a healthy diet, limit the energy value of food, and increase the level of physical activity.
2. Рекомендувати всім пацієнтам контролювати ІМТ та ОТ. Підтримувати ІМТ в межах 18,5 - 24,9кг/м2, ОТ≤88см (у жінок), ≤102см (у чоловіків).

4. Glucose level control

1. Patients with diabetes have a high risk of recurrent stroke.
2. Glucose level control should be carried out with the help of diet and selection of antidiabetic drugs.
3. The target level of glycosylated hemoglobin should not exceed 6.5%.

5. BP control

1. The recommended blood pressure level is no more than 140/80 mm Hg.
2. In patients with accompanying diabetes - no more than 130/80 mm Hg.
3. When choosing antihypertensive drugs, preference should be given to long-acting drugs, to achieve the target blood pressure, combined therapy should be used in accordance with the medical and technological documents regulating the provision of medical care for hypertension.

6. Physical activity

1. For patients who have suffered a stroke and have limitations in movement, safe physical activities are determined in an individual rehabilitation program.
2. For non-disabled patients and patients after TIA, walking for 30-40 minutes at least 4-5 times a week is recommended.

7. Treatment of sleep apnea syndrome

To correct the sleep apnea syndrome, the patient should be referred to a specialized center

Surgical methods of prevention of repeated stroke (carotid endarterectomy, angioplasty and stenting)

Provisions of the protocol

1. Surgical treatment for stenotic lesions of cerebral arteries can be performed in specialized neurosurgical and vascular departments with the necessary experience of relevant surgical interventions.
2. When treating a patient in a vascular or X-ray surgical department, the participation of a neurologist in justifying the indications for surgery is mandatory.
3. The main criteria that are taken into account in determining the indications and choosing the type of surgical treatment (direct or endovascular) include: the degree of narrowing of the artery, the clinical course of the disease, the risk of intervention for the patient's life and the risk of developing postoperative neurological and cardiac complications.
4. Concomitant somatic pathology, localization of the stenotic lesion, the condition of

collateral cerebral blood flow in the patient, adequate training and experience of the surgeon performing the operation, constitute the main criteria on which the safety of direct surgical and interventional neuro-radiological interventions depends.

5. Operations on the vertebral arteries (SAR) are performed only in the case of a symptomatic course of the disease and under conditions of ineffectiveness of conservative therapy.

6. The expediency of surgical intervention in case of stenotic lesion of the CA is considered only under the conditions of decompensated blood flow in the opposite spinal artery (occlusion, atresia, hypoplasia) or due to the anatomical variant of the transition of the opposite spinal artery into the posterior-inferior cerebellar artery.

7. In the case of a combination of stenotic lesion of the CA with hemodynamically significant (>70%) stenosis of the ICA, first of all, it is necessary to perform an operation on the carotid artery and evaluate the dynamics of the clinical course of the disease.

8. Operative interventions for elongations (loop formations, bends) of the main arteries of the neck are performed under the conditions of a symptomatic course and ineffectiveness of drug therapy, in the presence of segmental stenosis exceeding 70%, acute angles at the bends, and Doppler-proven insufficiency of blood circulation in the blood supply zone of the artery.

9. All patients who underwent surgical treatment require constant monitoring by a neurologist and continued constant drug control of ischemic stroke risk factors (hypertension, diabetes, hypercholesterolemia, etc.), constant antiplatelet therapy, and, if indicated, constant anticoagulant therapy.

Statement and recommendations

Provisions of the protocol

1. The duration of inpatient treatment depends on the severity of the stroke and ranges from 7 to 28 days (of which 7-14 are in a specialized stroke department).

2. Early discharge of the patient from the stroke department to home is possible in the case of a stroke of mild or moderate severity, provided that the rehabilitation of the MDK is continued in the health center that provides secondary outpatient medical care.

3. After discharge, patients must be under the constant supervision of a neurologist of the ZOH, who provides secondary outpatient medical care, a family doctor at the place of residence and continue to follow the instructions of the MDC.

4. All stroke patients who are discharged from the hospital should be given individual recommendations on the prevention of repeated stroke and rehabilitation.

Justification

Discharge planning and provision of an individual program of rehabilitation and prevention of recurrent stroke have a positive effect on the outcomes of medical care and rehabilitation at the secondary outpatient and primary levels of medical care.

Necessary actions of the doctor

Mandatory:

1. Assess the patient's degree of independence in everyday life according to the modified Rankin scale (Appendix No. 13).

2. Provide the patient with an individual plan (program) of secondary prevention measures and correction of risk factors upon discharge.

3. Provide the patient with an individual program of rehabilitation measures to be performed at the secondary outpatient level of medical care.

4. Give the patient after a stroke a printed "Information sheet for a patient who has suffered a stroke" (Appendix No. 19).

5. Involve the patient and their caregivers in participating in programs for non-pharmacological correction of stroke risk factors (if available) – Health Schools, Life After Stroke School, smoking cessation programs, psychological and physical rehabilitation programs, etc.

Materials for self-control of training quality

Tests

1) What arteries supply the brain with blood?

Answer options:
a) internal carotid artery;
b) main artery;
c) Adamkevich's artery;
d) Zulch's artery.
Correct answer: a, b)

2) What arteries provide blood supply to the cerebellum?

Answer options:
a) anterior cerebral artery;
b) Adamkevich's artery;
c) main artery.
Correct answer: c)

3) What artery provides blood supply to the frontal lobe?

Answer options:
a) anterior cerebral artery;
b) posterior cerebral artery;
c) middle cerebral artery;
Correct answer: a, c)

4) Transient disorders of cerebral circulation last until:

Answer options:
a) 3 days;
b) 1 week;
c) 24 hours;
d) 3 weeks;
e) 3 months.
Correct answer: c)

5) Subarachnoid hemorrhage is accompanied by the following symptoms:

Answer options:
a) decrease in blood pressure;
b) meningeal symptoms;
c) muscular hypertension by hemityp;
d) ataxia;
e) spastic quadriparesis.
Correct answer: b)

Tasks for self-control :

1. Patient E. is 67 years old. She was brought to the reception department with complaints of a squeezing headache, mainly in the back of the head, in the ears and in the head, flickering "flies" in front of the eyes, nausea and weakness in the left arm and leg. He has been suffering from hypertension for five years. She felt a sharp deterioration in her condition two hours ago after a psychotraumatic situation. Objectively: Hyperemia of the face and sclera. BP 220/120 mm. mercury Art. Pulse 92 beats. per minute, rhythmic, tense. Consciousness and sensitivity are not lost. Decreased muscle strength, volume of active movements in the left limbs. Tendon and periosteal reflexes are animated, above left. Abdominal reflexes are reduced on the left. On the left, a pathological Babinski reflex is evoked. Performs coordination tests vaguely to the left. The right performs well. There are no meningeal symptoms. After 1.5 hours, on the background of

hypotensive and antispasmodic therapy, the patient felt better: the headache, nausea, and neurological symptoms disappeared. Additional studies: Blood and urine analysis are normal. Fundus: optic nerve discs are pale pink, with a clear border. Arteries are narrowed, tortuous, veins are dilated, Salus-2 symptom. CT scan of the brain without focal pathology.

Question:

1) Name the diagnosis and justify it.
2) What vascular pool is affected?

Right answer:

1) TIA
2) Basin of the right middle cerebral artery.

Materials for classroom self-training:

List of educational practical tasks:

1. Master research methods:
   a) presence of paralysis and determine its type;
   b) violation of sensitivity and establish the type of defeat;
   c) language disorders and make a topical diagnosis;
   d) presence of agraphia, alexia, acalculia, apraxia.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

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Electronic information resources

1. Medical Books On-line Library (Neurology) – free download
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Practical lesson No. 17

Topic: Epilepsy and non-epileptic paroxysmal states.

Purpose: to acquaint applicants with the modern classification of epilepsies and epileptic syndromes, etiology, pathogenesis, main clinical manifestations of symptomatic, idiopathic epilepsy, as well as criteria and assessment of clinical symptoms and syndromes of seizures and epilepsy, disorders of consciousness and psyche.

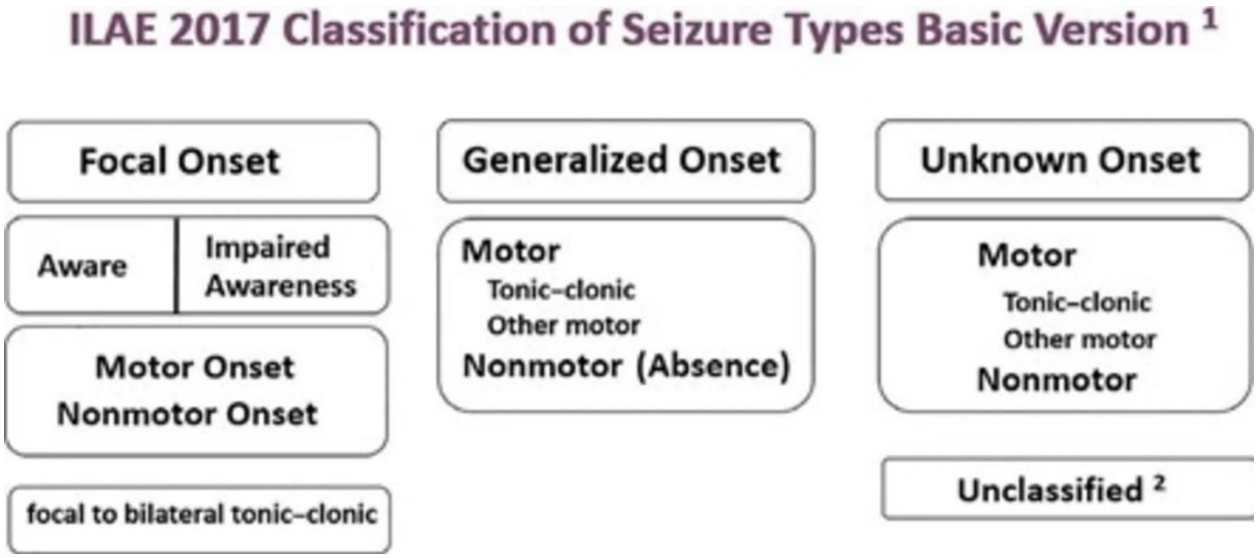
Basic concepts: the prevalence of epilepsy and similar paroxysmal disorders of consciousness makes it necessary to study and identify them in order to provide emergency care for convulsive and paroxysmal conditions, to carry out rehabilitation and social restorative measures with the aim of improving the body, ensuring the opportunity for such patients to live actively. Late recognition and treatment of these conditions often does not lead to a clinical effect, causes disability, and leads to a chronic course of the disease. Timely diagnosis makes it possible to successfully treat these diseases, to carry out effective preventive measures. For the correct assessment of various clinical forms of these diseases, thorough knowledge is required.

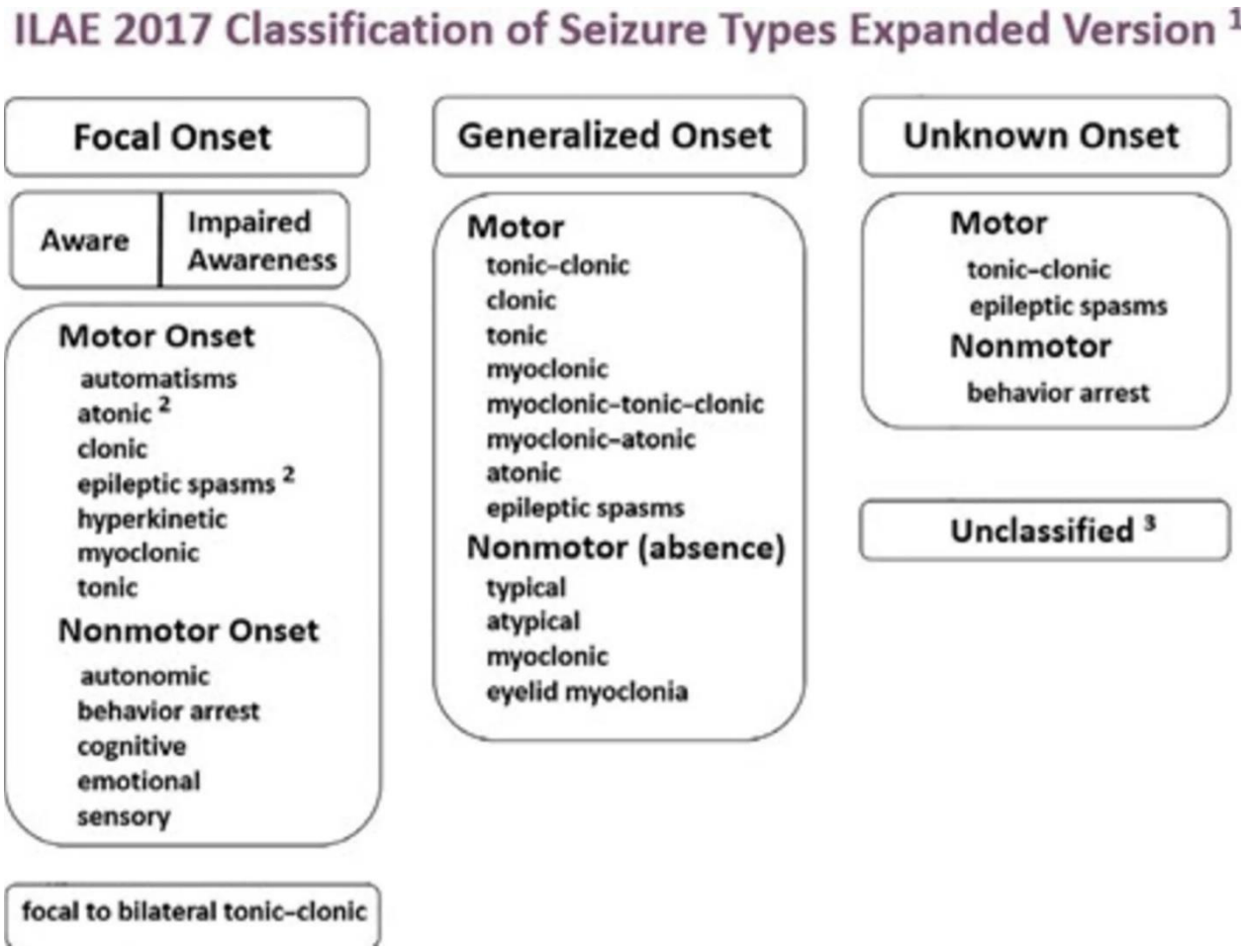
Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content





Materials for self-control of training quality

1. Where is cerebrospinal fluid produced and how is it exchanged?

Answer: CSF is produced in the choroidal plexuses of the lateral ventricles of the brain. Through the III and IU ventricles and the openings of Lyushka and Mozhandi, it enters the subarachnoid space of the brain (between the soft and arachnoid membranes), circulates around the brain and is absorbed by the pachyon granulations of the brain and spinal cord.

2. What is the composition of cerebrospinal fluid?
Answer: Protein - 0.33 ‰, cells - 1.7 per cubic meter. mm; glucose - half of the patient's blood glucose level; chlorides – 112 mg/%

3. What modern anticonvulsant drugs do you know?
Answer: luminal, benzonal, difenin, ethosuximide, eunoctin, valproic acid preparation (decapine, convulsafin, orphyril, acediprol, etc.), seduxen, carbomazepine, lamictal.

4. Etiological factors of epileptic attacks?
Answer: increased convulsive readiness of the cerebral cortex, epileptic focus, external epileptogenic stimulus.

5. Provocative factors of epileptic attacks?
Answer: alcohol, various infections, overwork, operations, abortion, childbirth, exposure to the sun, somatic diseases, craniocerebral injuries.

Instructional material for mastering professional skills

1. Possess the skills of examining patients:

a) For epilepsy (with major convulsive attacks).
b) With small epileptic attacks.
c) With epileptic status.
d) Prescribe treatment for status epilepticus and epileptic seizures.
e) Carry out differential diagnosis between epilepsy and hysteria.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

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Practical lesson No. 18

Topic: Headache.

Purpose: to create an idea among students about the main types of headache, differential diagnosis of headache, treatment of headache, to teach students to diagnose the main types of headache, to carry out differential diagnosis of headache, to prescribe treatment for headache.

Basic concepts: familiarity with the problem of sleep disturbance is relevant due to the fact that it can be observed at any age and with any pathology. The acquirer must recall the physiology of sleep, its clinical phases and bioelectrical characteristics; study different types of sleep disorders, learn to determine adequate therapy according to the etiology of the disease. The ability to correctly establish a diagnosis and prescribe adequate therapy is essential for reducing the suffering of many patients.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Classification of headache

Migraine.
Tension headache.
Cluster headache and chronic paroxysmal hemicrania.
Various forms of headache not associated with structural damage.
Headache associated with head injury.
Headache associated with vascular disorders.
Headache associated with nonvascular intracranial disorders.

Headache associated with the use of certain substances or refusal to take them.

Headache associated with infection.

Headache associated with metabolic disorders.

Headache or facial pain associated with pathology of the skull, neck, eyes, nose, sinuses, teeth, mouth, or other facial or cranial structures.

Cranial neuralgia, neuropathies and deafferentation pain.

Unclassified headache.

Two types are most common among all headaches: migraine - 38% and tension headache - 54%, as well as post-traumatic headache.

Migraine is a paroxysmal recurrent headache of a pulsating character, usually one-sided (hemicrania - pain in 1/2 of the head). It occurs in 2-6% of the population, mainly in women. Occurs at the age of 10 to 30 years.

In the pathogenesis of migraine, a hereditarily determined violation of vasomotor regulation of extra- and intracranial arteries is of leading importance. During an attack, 4 phases of vasomotor disturbances successively replace each other: spasm mainly of intracerebral and retinal vessels; dilatation of extracerebral arteries; edema of the vascular wall; reverse development of changes. In the first phase, an aura may occur, in the second - a headache. Disruption of the metabolism of serotonin and other biologically active substances (histamine, prostaglandins, tyramine, glutamate, etc.) plays an important role in the pathogenesis of migraine. Recently, the triggering factor of an attack is considered to be not biochemical, but neurophysiological changes.

According to the international classification, migraine is divided into 2 types: migraine without aura and migraine with aura. Precursors of migraine can be periodic syndromes of childhood: spasm-like pains in the stomach (abdominal migraine), paroxysmal dizziness ("vestibular migraine"), attacks of vomiting, tendency to pump, less often alternating paresis of the limbs.

Migraine without aura (simple migraine). Its main manifestation is a throbbing unilateral headache. More often, it does not occupy the entire half of the head, but, as a rule, the fronto-temporal or parietal-occipital region, less often it is bilateral, the side of the pain may alternate. The intensity of the pain is average or significant, at the end of the attack the pain is dull. During the attack, general hyperesthesia, intolerance to light, loud sounds develops. The patient wants to lie down in bed and not move, this relieves the pain, physical exertion intensifies it. In most patients, the attack is accompanied by nausea, often vomiting. The duration of attacks varies from 4 to 72 hours.

Migraine with aura. Aura is a focal neurological symptom that precedes a headache. Headache occurs immediately after the end of the aura or after a short light interval, less often - during the aura, especially a prolonged one. The most typical visual aura, manifested by a flickering scotoma, blurring of vision, a zigzag line in the homonymous fields of vision. It lasts 5-20 minutes, and then a headache attack occurs (ophthalmic, classic migraine). The second most frequent place is an aura in the form of paresthesias, which occur first in one finger of the hand, then pass to others, rise up the hand and spread to the face, tongue (this sometimes causes dysphasia, even with left-sided paresthesias). Rare types of aura include hemiparesis, motor aphasia, and ophthalmoparesis. Migraine with aura in the form of neurological disorders was previously called associated. In rare cases, usually in elderly men, the aura is not followed by a headache (dissociated migraine, "migraine without migraine"). Aura is caused by local ischemia. Unlike transient ischemic attacks, which are mistakenly diagnosed in these cases, the main and intracranial arteries are intact and the prognosis in most patients is favorable.

Of great interest is "familial hemiplegic migraine" associated with the pathology of the gene mapped on the 19th chromosome. It is characterized by a prolonged aura in the form of hemiparesis, paresthesia, and speech disorders. The duration of the aura varies from 2-3 hours to

3 days, and the headache develops during the aura, as in other cases with a prolonged aura.

Complications of migraine. These include migraine status and migraine stroke.

Migrainous status. Sometimes migraine attacks follow one another without a break, accompanied by repeated vomiting and dehydration. If the attack lasts more than 72 hours, migraine status is diagnosed. It requires hospitalization and emergency treatment, including corticosteroids.

Migrainous stroke (brain infarction). Recently, it has been proven that in rare cases, a migraine attack ends with the development of a brain infarction, which leads to the occurrence of a neurological deficit that persists for more than 3 days and is not always reversible. Brain infarcts in migraine are usually localized in the back of the brain.

Chronic daily headache. In some patients with migraine, a constant headache appears against the background of typical migraine pains, which differs in character from migraine (pain is not pulsating, diffuse, less intense, without nausea and vomiting). Its cause was considered to be a combination of migraine with another type of headache, most often psychogenic. Recently, it has been proven that the basis may lie in the abuse of medicines, such a headache is called an abuse headache.

The diagnostic criteria for migraine are given in the International Classification of Headache.

Diagnostic criteria for migraine without aura.

The presence of at least 5 attacks lasting from 4 to 72 hours (without treatment).

Cephalgia has at least 2 of the indicated signs: one-sided, pulsating, moderate or strong, aggravated by physical exertion.

Cephalgia is accompanied by at least one of the listed symptoms: nausea and/or vomiting, photo- and phonophobia.

It is also important to alternate the side of cephalgia, since a one-sided headache for a long time requires the exclusion of other diseases.

Another classification criterion that applies to all other types of primary headache is compliance with one of the following three conditions: either the anamnesis, somatic and neurological examination exclude the presence of diseases in which cephalgia is symptomatic, or these diseases are expected, but excluded by detailed examinations, or the patient has these diseases, but migraine attacks are independent and not related to them in terms of time of occurrence.

Diagnostic criteria for migraine with aura.

The presence of at least 2 attacks, characterized by at least three of the listed signs: there is one or more aura symptoms, completely reversible, which indicate focal cerebral dysfunctions of the cortex and / or trunk;

none of the aura symptoms lasts more than 60 minutes (but if there are more symptoms, a proportionately longer duration is allowed);

headache follows the aura with a light interval of less than 60 minutes, but may begin before the aura or simultaneously with it;

at least one of the aura symptoms develops gradually in more than 4 minutes or 2 or more symptoms develop simultaneously;

have one of the conditions listed above (migraine without aura).

Diagnostic criteria for abuse headaches (caused by chronic administration or drug overdose):

headache occurs after taking the drug daily for 3 months or more;

it is possible to accurately establish the minimum dose that brings relief;

headache is chronic (15 days or more per month) and disappears within 1 month after stopping the drug.

Ergotamine abuse tooth pain occurs at a dose of 2 mg per day orally and 1 mg rectally.

Analgesic overuse pain occurs when taking 50 g of aspirin per month (or an equivalent dose of another non-narcotic analgesic), or when taking combined analgesics (with caffeine, barbiturates, tranquilizers, etc.) in the amount of 100 tablets or more per month, or when taking narcotic analgesics .

Tension headache is one of the main forms of primary headache. It is manifested by cephalic episodes (several minutes - several days). The pain, as a rule, is bilateral, compressive in nature, of moderate or light intensity, does not increase with ordinary physical exertion. Sometimes photo and phonophobia is possible.

The term "tension headache" also corresponds to: GBN, muscle tension headache, stress headache, psychomyogenic headache, psychogenic headache, idiopathic headache.

Classification of tension headache

There are several types of tension headache, some of which in turn have subtypes:

episodic (occurs no more than 15 days within 1 month)

chronic (occurs more than 15 days a month)

In addition, both forms of tension headache are divided into "tension headache" and "tension headache without pericranial muscle tension."

Etiology and pathogenesis of tension headache

In modern medicine, tension headache is considered exclusively as a neurobiological disease. Presumably, not only central, but also peripheral nociceptive mechanisms are involved in the etiology of tension headache. The leading role in the pathogenesis of tension headache is played by increased sensitivity of pain structures, as well as insufficient function of the descending inhibitory pathways of the brain.

The main provoking factor of a tension headache attack is emotional stress. It has been proven that switching attention or positive emotions can reduce the intensity of a headache up to its complete disappearance. However, after a while the headache returns. Another provoking factor is the so-called the muscular factor, i.e. being in tension for a long time without changing the posture (forced position of the head and neck when working at a desk and driving a vehicle).

There are also factors that form a chronic pain pattern. One of these factors is depression. In addition to traumatic life situations, the development of depression is also facilitated by personality characteristics, you or other behavioral characteristics. Another factor of chronicity is drug abuse (abuse of symptomatic painkillers). It has been proven that in case of consumption of a large number of painkillers, chronic tension headache is formed twice as often. To treat drug abuse, it is necessary to cancel the drug that caused this complication as soon as possible.

Clinical picture of tension headache

Typically, patients describe a tension-type headache as a mild to moderate, non-throbbing, bilateral squeezing headache that squeezes the head in a "hoop." The intensity of such a headache does not depend on physical exertion, and is very rarely accompanied by nausea. It appears, as a rule, some time after waking up and continues throughout the day.

Diagnosis of tension headache

There are several criteria for diagnosing a tension headache:

Duration of headache from 30 minutes to 7 days

The presence of at least two of the following signs:

pain intensity does not depend on physical exertion;

bilateral headache;

mild or moderate intensity of pain;

the nature of the pain is not pulsating, but squeezing (squeezing the head with a "hoop");

absence of nausea and vomiting;

headache is not a symptom of another disorder of the body's functions;

increasing pain against the background of strong emotional stress;

pain relief against the background of positive emotions and psychological relaxation.

Since, in addition to the above signs indicating a tension headache, patients often complain of a feeling of discomfort and even burning in the back of the head, back of the neck and forearm ("coat hanger" syndrome), when examining the patient, it is necessary to examine the cranial muscles . It has been proven that the most sensitive diagnostic method for detecting pericranial muscle dysfunction in patients with GBN is palpation. This dysfunction is revealed when pressing in the area of the frontal, masticatory, sternoclavicular-mammoid and trapezoidal muscles, as well as when palpating with rotational movements of the second and third fingers in the area of the same muscles. The presence of pericranial muscle dysfunction is taken into account in the future when choosing a treatment strategy. Hypersensitivity of the pericranial muscles during palpation means the presence of "chronic (or episodic) tension headache with tension of the pericranial muscles."

In addition, in addition to the signs listed above, manifestations of anxiety and depressive disorders in the form of low background mood, apathy or, on the contrary, increased aggressiveness and irritability often coexist. The degree of such disorders in tension headache can vary from mild to severe.

Differential diagnosis

To rule out an organic cause of tension headache (tumors, inflammatory processes, impaired blood circulation of the brain), a full set of neurological examinations is performed: EEG of the brain, ECHO-EG, if there are indications - CT scan or MRI of the brain.

The main difference between episodic tension-type headache and chronic tension-type headache is the number of days (days/month) in which this headache occurs.

Treatment of tension headache

In the treatment of tension headaches, neurologists use a complex approach. Firstly, it is necessary to normalize the emotional state of the patient, and secondly, to eliminate the dysfunction of the pericranial muscles. In addition, it is necessary to take measures to prevent drug abuse. The result of such treatment is the reduction of pain and muscle-tonic syndrome, prevention of transformation of episodic tension headache into chronic GBN.

The following groups of drugs are used as drug treatment for tension headaches:

antidepressants (selective serotonin reuptake inhibitors, selective serotonin and norepinephrine reuptake inhibitors);

muscle relaxants (tolperisone, tizanidine), NSAIDs (diclofenac, naproxen, ketoprofen);

drugs for the preventive treatment of migraine (in case of combination of tension headache with migraine)

Acupuncture, manual therapy, massage are used as non-medicinal methods of treatment for tension headaches.

Cluster headache. Synonyms: cluster headache, migraine neuralgia Harris, histamine cephalgia Horton, etc. This type of headache unites several previously distinct forms: migraine neuralgia, ciliary neuralgia, etc.

In the International Classification, 3 forms of cluster pain are distinguished depending on the frequency of their manifestation: with an undefined periodicity, episodic and chronic.

Together with cluster headaches, chronic paroxysmal hemicrania and cluster-like headaches are considered.

Cluster cephalgias are rare, men are affected 5-6 times more often, the onset of the disease is in 20-40 years. Etiopathogenesis is not known, it is assumed that vascular mechanisms are the basis of pain.

Episodic cluster headache.

This disease is characterized by attacks of very strong one-sided headache, which repeats daily (1-2, rarely 5-8 times) for several weeks or even months. After that comes a long remission (months and years). The intensity of pain and the duration of the attacks change during one cluster period (a series of attacks) from lighter and shorter to stronger and longer, and then the attacks become light and disappear. The pain occurs suddenly without warning, is localized in the eye area, periorbital zone and in the temple, radiation to the ear, neck, and hand is possible. The nature of the pain is burning, and the force is so great that it wakes up sleeping patients. The duration of the attack (without treatment) is 15-180 minutes. At the beginning of the series, attacks more often develop at night, in the morning at the same time ("alarm clock" headache), but they can also occur during the day. Psychomotor excitement is noted during the attack. Attacks are accompanied by pronounced vegetative disorders, redness and lacrimation from one eye, nasal congestion, rhinorrhea, sweating in the face or forehead, ptosis, miosis. According to the modern classification, the diagnostic criteria for cluster episodic headache are the presence of at least 5 attacks of the nature and location described above and at least one of the listed vegetative symptoms, as well as a serial course of daily attacks.

Chronic cluster headaches occur in two variants: without remission from the moment of their appearance or transforming from an episodic headache. Attacks are shorter and less severe, but are characterized by high frequency (up to 20-30 per day) and lack of remission.

Chronic paroxysmal hemicrania is a rare type of paroxysmal one-sided headache, localized in the eye-frontal-temporal area (above and below the eye, with migraine - above the eye), of great intensity, drilling. Attacks last 10-30 minutes, repeat 10-20 times a day and are accompanied by vegetative symptoms from the eye and nose. They are distinguished from chronic cluster cephalgia by the predominance of women and the "dramatic" effect of indomethacin, which quickly relieves headache ("indomethacin" headache).

Various headaches not associated with structural damage. This group includes rare forms of non-organic headache. These include idiopathic shooting pain, which manifests itself as a sharp lightning (fraction of a second) severe pain in certain parts of the head, its pathogenesis is unclear.

Headache associated with hypothermia can be caused by any cold stimulus, even in the pharynx.

The headache associated with coughing and physical exertion (effort) is throbbing in nature, but is never accompanied by any other symptoms. By nature, this is a benign pain, but in some patients, the appearance of pain during tension can be caused by structural processes that occur with hypertensive syndrome (brain tumors, etc.). Therefore, headaches with coughing and physical exertion require a detailed examination, including a CT scan of the brain.

Diagnosis of headache. At the first stage of diagnosis, it is necessary to determine whether the headache is related to a structural lesion (organic brain disease). It is most important to rule out tumors, acute disorders of cerebral circulation, especially subarachnoid hemorrhage, hematomas, complications of acute TBI (epi- and subdural hematomas, etc.), inflammatory diseases of the brain, its membranes, paranasal sinuses, eye, glaucoma, etc.

In the International Classification, "danger signals" in headache that cause suspicion of structural damage are highlighted:

Occurrence of headache for the first time after 50 years.

Sudden severe ("thunderous") headache (subarachnoid hemorrhage, first migraine attack, temporal arteritis).

"Tide" to the head (intracranial hemorrhage).

Increase in headache during coughing, straining, physical exertion (intracranial hypertension, migraine).

Increase in headache over time: over hours-days (meningitis, encephalitis), days-weeks (tumor, temporal arteritis).

Headache is the cause of night awakenings (tumor, attack of cluster headache, migraine).

In the morning, nausea, vomiting, hiccups, dizziness (tumor).

An indication of a structural headache is also the presence of focal neurological symptoms, changes in additional research methods, ineffectiveness of therapeutic effects, non-compliance of the headache with the criteria of the International Classification of Headaches.

In favor of a primary headache (migraine, tension headache, cluster and rare variants) is evidenced by the compliance of the headache with the diagnostic criteria, normal well-being and stable body weight, duration of the pain for more than two years and the absence of deviations from the norm during the examination (this sign is the most significant) .

Scheme of examination of a patient with a headache:

- According to the indications, visualization of vessels by non-invasive methods (extra- and transcranial dopplerography) or with intra-arterial injection of a contrast agent (angiography) can be performed to clarify the diagnosis.

These studies more accurately than CT allow to establish the presence and localization of an aneurysm, occlusion of an artery, the state of the vascular bed, the displacement of vessels in the case of bulky processes, to detect vascular tumors, the peculiarities of its blood supply or avascular areas with a violation of the topography of vessels (cyst, abscess, some tumors, hematomas, infarction with perifocal edema).

Other special methods - evoked potentials, nystagmography, etc. - are used to finally specify the nature of the structural process.

At the second stage of diagnosis, after excluding the symptomatic nature of headaches, the type of primary headache is specified. Here, compliance of the headache with the diagnostic criteria of the International Headache Society is crucial. The given diagnostic criteria are very effective in distinguishing the type of headache. Next, it is necessary to assess the frequency of attacks, which is important for the choice of treatment tactics, for example, abortive (in case of rare migraine attacks) or preventive (in case of frequent attacks) treatment.

To work out the tactics of therapy, the choice of drugs and their dosages in patients with chronic pain, it is recommended to keep a diary, which reflects the type of headache, its intensity, duration and frequency of attacks (similar to the diary of a patient with epilepsy). The diary also displays previous and accompanying headache symptoms, provoking factors, response to stress, which drug and which dose stopped the attack. After clarifying the type of headache and its features, the patient is given a calendar in which the dates of the attacks are noted and their frequency is taken into account.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 19

Topic: Professional and household neurointoxications. Damage to the nervous system under the influence of physical factors

Purpose: to create in the applicants an idea about the etiology, pathogenesis of professional and household neurointoxications, the clinical picture of professional and household neurointoxications, modern methods of treatment of professional and household neurointoxications, etiological factors of damage to the nervous system due to physical influences, modern methods of treatment of damage to the nervous system under the influence of physical factors, teach applicants to diagnose and treat various neurointoxications and vibrational disease, recommend preventive measures when working with physical factors.

Basic concepts: Professional and domestic neurointoxication, damage to the nervous system under the influence of physical factors are serious diseases that mainly affect the young working population and require timely diagnosis and urgent treatment.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Neurotoxicity

Mercury is a liquid metal that has the property of evaporating at room temperature and entering the human body by inhalation. Mercury poisoning most often occurs among workers whose work is related to its extraction or use in the production of measuring devices, X-ray tubes, mercury-containing medical preparations, etc.

Pathogenesis. In case of accidental consumption of metallic mercury, it passes through the digestive tract and does not cause poisoning. Inhalation of vaporized mercury in the lungs leads to its accumulation in the blood, where it remains unchanged for a certain time. Gradually, it forms compounds with blood proteins that are partially excreted with urine, biological secretions, through the intestines.

A significant amount of mercury albuminate is deposited in parenchymal organs, the brain (this is where the highest concentration of this substance is observed). Almost exclusive accumulation of metal is observed in the subcortical nuclei, in the area of the midbrain. The vascular plexuses and the actual cortex of the cerebral hemispheres are irritated (due to the ability of mercury to circulate in the cerebrospinal fluid). A significant role in the pathogenesis of poisoning is played by disorders of neuropeptide metabolism, disruption of oxidative phosphorylation processes in the mitochondria of neurons.

Pathomorphology. Histological changes consist in focal destruction of the cortex, subcortical nuclei, thalamus and brain stem. The most severe changes are observed in the area of the ammonium horn, motor centers, vascular system.

Clinic. Acute inorganic mercury poisoning is rare and is the result of accidents with a significant release of vaporized mercury into the production area. Its symptoms develop immediately after the entry of metal into the body and are manifested by headache, drowsiness, metallic taste in the mouth, fever, vomiting, diarrhea. Within a few days, gingivitis and ulcerative stomatitis appear, which sometimes spreads to the upper respiratory tract with the development of severe toxic pneumonia and pulmonary edema.

In mild cases, asthenovegetative syndrome prevails: general weakness, red diffuse dermographism, sweating, tachycardia.

Timely evacuation of the victim from the contaminated area and adequate therapy ensure almost complete regression of symptoms and recovery of the patient within 2–3 weeks.

Chronic mercury intoxication develops slowly, several years after the beginning of contact with mercury.

Several consecutive stages of pathology are distinguished (Kussmaul, 1861).

1. Irritable weakness syndrome — emotional instability, increased fatigue, memory impairment, sleep-wake cycle disorder, sleep inversion. A tremor of the fingers of the outstretched hands is typical for this stage, initially transient, with an increase in amplitude and duration. The excitability of the autonomic nervous system increases (lability of the AT pulse, violation of the ocardiac reflex, bright red dermographism). The early symptoms of intoxication include mercury stomatitis, gingivitis with the formation of a blue-black border on the gums, hair loss, brittle nails. Intoxication, which has a latent course, can be aggravated in case of an acute infection or other various stressful effects on the body. Symptoms are reversible if timely treatment is available.

2. Asthenovegetative syndrome (psychovegetative, mercurial neurasthenia): loss of body weight, constant headache, depression, offensiveness, self-doubt. A symptom of mercurial eretism is the appearance of strong excitement, anxiety in the presence of strangers, inability to perform usual work, vasovegetative reaction (redness of the face, sweating, palpitations). In this stage, the tremor intensifies, becomes permanent, spreads to the lower limbs, and in a state of excitement has the appearance of hyperkinesis. Somatically, hyperplasia of the thyroid gland, dysmenorrhea, bleeding gums, alveolar pyorrhea, copper-red coloration of the mucous membrane of the pharynx and soft palate, gastroenterocolitis with abdominal pain, and profuse diarrhea are observed. Timely active treatment reduces symptoms.

3. Mercury encephalopathy: complaints of intense headache, insomnia. High-amplitude tremor of the upper and lower limbs, head, chorea-like hyperkinesis, dynamic ataxia, slurred speech, nystagmus, hyperreflexia with the presence of pathological reflexes are observed. In severe cases, amyostatic syndrome (mixed form) develops. Psychoorganic syndrome is represented by schizophrenia-like disorders (hallucinatory-delusional phenomena, reduced intelligence), mercurial psychosis leading to dementia. In some cases, polyneuropathy occurs with a predominant lesion of the ulnar nerve. Mercury intoxication in pregnant women is accompanied by a neurotoxic effect on the fetus with the development of prenatal lesions of the central nervous system. In the stage of mercury encephalopathy, the changes are almost irreversible, so the treatment is almost completely symptomatic.

Diagnosis and differential diagnosis are based on the features of the clinical picture (sleep disorders, erethism, tremors, gingivitis with a blue-black stripe on the gums, vegetative disorders), professional history, determination of mercury concentration in urine and feces. On the other hand, the presence of metal in secretions without clinical symptoms of poisoning is considered as a carrier of mercury. There is often no clear correlation between the level of mercury in the urine and clinical symptoms, because a large part of the metal is retained in the depot.

Treatment includes the following measures: 1. Evacuation of the victim from the contaminated area. 2. Hospitalization in the department of occupational pathology or toxicological or neurological, regardless of the condition. 3. Administration of 5 ml of 5% unitiol solution subcutaneously (5 injections), 20 ml of 20% sodium hyposulfite solution intravenously (12–15 injections), 20 ml of 40% glucose solution with 5–10 ml of 5% ascorbic acid solution intravenously (10–15 injections). 4. Prescribing vitamins of group B, psychotropic drugs, glutamic acid, sodium adenosine triphosphate. 5. Sanatorium-resort treatment with balneotherapy (hydrogen sulfide baths No. 12–15 with a break of 6–8 months) — Khmelnyk, Slovyansk, Solony Liman.

Intoxication with organic compounds of mercury

Organic compounds of mercury - insecticide fungicides - are intended for pickling seeds. These include granozan, ethylmercury phosphate, and mercurane. Pathogenesis. Organic compounds of mercury enter the body by inhalation (absorption - 75%), through the digestive tract (absorption is almost 75%), intact skin (2–5%). Highly toxic, quickly deposited. In the depot, they gradually turn into inorganic compounds of mercury. They pass through the BBB well. They are enzyme poisons that block thiol groups of active enzyme loci. Disrupt protein and lipid metabolism, cause demyelination of peripheral nerves and spinal cord, mostly at the height of mercury removal from the body. Pathomorphology. The changes have a multi-organ character. In the nervous system, there is degeneration of cortical ganglion cells, demyelination of nerve trunks with preservation of axial cylinders.

Clinic. Acute poisoning: fever, nausea, uncontrollable vomiting, diarrhea appear after short-term general weakness and illness. In the case of mild poisoning, the symptoms regress quite quickly. Moderate poisoning: on the 2nd to 3rd day after poisoning, headache, staggering while walking, pain and paresthesias in the lower limbs, psychomotor excitement, hypersalivation, metallic taste in the mouth appear. Along with neurological symptoms, arterial hypertension, proteinuria, and hematuria are observed. In the case of timely and adequate treatment, symptoms regress within 1.5–2 months. Severe poisoning is characterized by an extensive clinical picture of toxic polyneuropathy with gross motor, sensory and trophic disorders. The course can be according

to the type of paralysis of Landry; periodic deterioration of vision, amiostatic and cerebellar disorders, hypothalamic syndrome of varying degrees of severity are observed. Chronic poisoning develops after a latent period of 1–1.5 months. in the form of ulcerative stomatitis, gingivitis, toxic gastroenterocolitis, myocarditis, liver and kidney damage. The neurological status is characterized by the presence of polyneuropathic disorders, often in the form of encephalopolyneuropathy accompanied by hypothalamic syndrome. Treatment consists in evacuating the victim from the contaminated environment, washing the stomach with a weak solution of potassium permanganate, using activated carbon or protein water (2 egg whites per 250 ml of water), saline laxative. Further measures are the same as in case of poisoning by inorganic mercury compounds.

Prevention includes the following measures: 1. Compliance with safety techniques when working with mercury and its compounds, periodic medical examinations of workers who come into contact with these compounds. 2. Periodic rinsing of the oral cavity with a solution of potassium permanganate, use of multivitamin preparations with an increased amount of thiamine, ascorbic acid. 3. Enhanced nutrition, additional leave.

Poisoning by lead and its inorganic compounds

Lead is a metal that evaporates at a temperature higher than 400 °C. Contact with lead is possible in the conditions of mining and processing of lead ores, production of paints in the printing industry, production of hunting shot, bullets, etc.

Pathogenesis. Lead enters the body through the respiratory tract (aerosols) and the digestive tract (swallowing lead dust and eating food with dirty hands), 54 and 41% are absorbed, respectively.

In the blood, lead circulates in the form of highly dispersed albuminate and phosphate, is deposited in bones, liver, kidneys, is excreted with urine and feces, as well as with biological secretions of the body. In the later stages of the disease, lead enters the nervous tissue through the BBB.

Lead is a thiol poison, blocks the sulfhydryl groups of the corresponding enzymes and thus disrupts the course of many metabolic processes.

Lead irritates the interoreceptors of the vascular wall and internal organs, reflexively causes neurodynamic disorders in the cortex and cortical-subcortical connections. In the first phase, excitation processes prevail: the threshold of the analyzers' excitability decreases, the concentration of biologically active substances in the blood increases. The second phase is characterized by the development of inhibition, a decrease in the excitability of the taste and smell analyzers, and the torpor of vegetative reflexes.

Lead disrupts porphyrin metabolism, which causes hyperporphyrinemia and a decrease in hemoglobin in the blood, reactive reticulocytosis.

Severe intoxication causes significant dysregulation of the central and peripheral vegetative apparatus, a kind of vegetative crisis in the form of lead colic.

Pathomorphology. Saturnism is characterized by degenerative changes in the cortex, subcortical nuclei, trunk, anterior horns of the spinal cord. Degeneration of the myelin sheath of peripheral nerves occurs at more pronounced stages of intoxication. In severe cases, vascular disorders (hyperemia, hemorrhages), atrophic processes in the fronto-parietal area of the cortex and striatum, trunk and dentate nucleus of the cerebellum are observed (diffuse breakdown of myelin, vacuolization of Purkinje cells in the cerebellum with proliferation of neuroglia). The maximum vascular changes are observed near the lymphatic vessels and in the area of the solar plexus.

Classification. Inorganic lead intoxication is divided into the following forms according to the severity of poisoning and the prevailing clinical syndrome:

1. Lead carrier - presence of metal in urine without clinical manifestations.
2. Mild lead poisoning:

— some mild symptoms, a lead border on the gums; hemoglobin level is normal;

— mildly expressed asthenic syndrome.

3. Moderate lead poisoning:
- anemia;
- mild colic;
— toxic hepatitis, clinically expressed;
— asthenovegetative syndrome;
— sensory polyneuropathy.
4. Severe lead poisoning:
- anemia;
— pronounced lead colic;
— encephalopathy;
- lead paralysis.
Clinic. The leading symptoms of intoxication are:
— a lead border on the gums in the form of a purple-aspid narrow strip along the edge of the gums with the presence of carious teeth;
— significant reticulocytosis;
— the presence of erythrocytes with basophilic granularity in the blood;
— hyperporphyrinemia;
— pale gray ("lead") color of the skin of the face;
— lead content in urine is more than 0.05 mg/l;
— lead colic (convulsive abdominal pain, absence of peritoneal symptoms, persistent, up to 7–10 days, constipation, increased AT, sinus bradycardia, vagus effect when trying to induce pericardial reflex).
Neurological signs:
- lead neurasthenia (headache, general weakness, sleep disorders, hypoesthesia, increased threshold of excitability of analyzers, permanent parasympathetic autonomic dysfunction - muscle weakness, increased sweating, pericardial reflex, low pulse lability during functional tests - tremor of the fingers, painful flexion of the limbs. The syndrome has diagnostic value in the presence of other signs of saturnism. The condition is reversible if timely treatment is started and contact with lead is broken; - lead encephalopathy (severe headache, mnestic disorders, reduced criticism of one's own condition, psychosensory disorders - derealization, asomatopoagnosia, hallucinations, insufficiency of the function of III, VII, VIII pairs of cranial nerves, tremor of the limbs, which becomes intentional, hyperkinesis, dysarthria, ataxia, nystagmus, retrobulbar neuritis with the transition to atrophy of the optic nerves);
— lead meningopathy (pallor of the skin, positive symptoms of Kernig and Brudzinsky, tenderness of Kerer's points, mild CSF hypertension);
— lead epilepsy (develops in severe cases during excessively prolonged colic in the form of status epilepticus, there are also cases of cortical epilepsy, presence of equivalents). This syndrome is quite rare;
— lead encephalomyelopolyneuropathy and polyneuropathy.
According to Dezherin's classification, five syndromes of lead paralysis are distinguished:
- antebrachial - paralysis of the common extensor of the fingers, then - other extensors of the fingers and the hand - the hand hangs at a right angle in a half-pronation position, the thumb is brought to the palm;
— brachial (Duchenne — Erba) — damage to the proximal muscles of the upper limbs;
— Aran-Duchennev — paresis of the thenar and hypothenar muscles with the formation of a "monkey's hand";
— peroneal — observed in children, the muscles of the distal parts of the lower limbs are affected;

— generalized with damage to the muscles of the larynx and cranial nerves, paralysis of the limbs in the form of encephalomyelopolyradiculoneuropathy, motor disorders, especially in subacute cases.

Manifestations of vegetative polyneuropathic disorders and sensitivity disorders are pain in the distal parts of the limbs, vasomotor disorders, distal hypo- and hyperesthesia, elements of hyperpathy.

Acute inorganic lead poisoning (eating food stored in an unglazed clay vessel) is characterized by the rapid development of a comatose state, an epileptic syndrome accompanied by a psycho-organic syndrome, diffuse focal symptoms.

Diagnosis, differential diagnosis. The presence of acute polyneuritis, encephalopathy or other neurological disorders together with the presence of cardinal symptoms of poisoning is a sign of lead intoxication. A significant role is played by professional and epidemiological anamnesis.

Treatment. In order to neutralize and remove lead from the body, complexones are used: tetazincalcium (intravenous drip of 20 ml of 10% solution in 5% glucose solution 2 times a day with intervals of at least 3 hours for 3–4 days with a break of 2–4 days, course treatment — 1 month or orally 1 tablet (0.5 g) 4 times a day (0.25 g 8 times is possible) 3–4 times a week for 20–30 days). A repeated course of treatment is carried out no earlier than after 1 year. Pentacin (intravenously slowly 5–30 ml of an aqueous solution once every 2–3 days, 10–20 infusions in total), you can use the disodium salt of EDTU.

Together with this, iron preparations, B vitamins, ascorbic and nicotinic acids, anticholinesterase drugs, sedatives, vasoactive drugs and diuretics are used. In order to stimulate the removal of lead from the depot, diathermy of the right hypochondrium or intravenous administration of sodium thiosulfate is used. Recommended sanatorium-resort treatment using hydrogen sulfide procedures and mineral water of the appropriate composition (Khmilnik).

Intoxication with organic lead compounds

Tetraethyl lead (TEP) is an oily, colorless liquid, a highly volatile compound. It is used for the production of ethyl liquid, additives to petroleum.

Pathogenesis. It easily enters the body through the respiratory tract and intact skin, because it dissolves in lipoids. It circulates in the blood unchanged from 8 hours to 2-3 days. Freely passes through any histogematic barriers. Breaks down to inorganic lead (complete breakdown) or toxic metabolites (partial breakdown), which are deposited. Tropic to the neurons of the cerebral cortex, hypothalamic area, red nuclei and cerebellum, affects the tone of blood vessels, causes ischemia of brain tissue. TES and its metabolites block pyruvate dehydrogenase, suppress cholinesterase activity and thus deepen brain hypoxia.

Pathomorphology. The hypothalamus and cerebral cortex are mainly affected (microhemorrhages, edema, vacuolization of erythrocytes), hyperemia and petechial hemorrhages in the meninges, swelling of the brain substance are observed.

Clinic. Acute poisoning, subacute and chronic TPP intoxication are distinguished.

Acute poisoning: a rapid form of the disease is possible with the acute development of psychosis, epileptiform attacks and death within a short time. In some cases, a latent period (hours - days) is possible.

The initial stage is asthenovegetative syndrome (headache without specific localization, metallic taste in the mouth, general weakness, euphoria, autonomic dysfunction of a parasympathetic nature: arterial hypotension, bradycardia, hypothermia, hypersalivation; sleep disorders, paresthesias in the form of a sensation of a foreign body on the tongue). In the case of severe poisoning, these symptoms are joined by organic signs: frontocerebellar ataxia, intentional tremor, dysmetria, nystagmus, dysarthria. A pre-delirious state develops: euphoria, non-criticality of one's own state, disorders of memory, thinking, muted state of consciousness, disorientation.

Pre-climax stage: headache intensifies, vomiting occurs. Delirium develops (welcoming terror, hallucinations, delusions of persecution and relation, psychomotor excitement against the background of a muted state of consciousness). A pseudoparalytic syndrome is observed - along

with ataxic manifestations, euphoria and intellectual disorders are present. Statodynamic ataxia, dysarthria, hyperkinesis, pathological reflexes are characteristic.

The culminating stage: psychomotor excitement with inappropriate behavior, hallucinatory-delusional disorders. Epileptic seizures, myoclonic and choreoathetous hyperkinesis, sharp vegetative lability are observed: hypothermia changes to hyperthermia up to 40 °C, bradycardia and tachycardia, arterial hypo- and hypertension, profuse sweating, a possible fatal outcome. Adequate treatment leads to the recovery of the victim, but a significant mental and neurological deficit remains. There may be cases of a wave-like course of pathology with repeated relapses.

Chronic intoxication has three stages according to the severity of the condition:

— asthenovegetative syndrome;

— encephalopathy with hypothalamic syndrome. In this stage, hypothalamic crises with chills, visceral pain, elasticity, characteristic cataplexy of awakening for several minutes, hypnagogic hallucinations prevail;

— toxic encephalopathy with the development of psychoses (the latter have a protracted course and are prone to relapse);

— symptoms of chronic TES intoxication resemble symptoms of acute poisoning, but last longer.

Diagnosis, differential diagnosis. The diagnosis is established taking into account primary autonomic disorders (parasympatheticotonia) with subsequent inversion of symptoms (predominance of ergotropic phenomena), psychotic disorders, professional history.

Treatment. Immediate measures: washing the skin with kerosene, then with soap and water; in the case of enteral administration of TES — gastric lavage with a 2% solution of sodium hydrogen carbonate or a 0.5% solution of magnesium sulfate followed by the use of magnesium sulfate as a laxative. Forced diuresis, ascorbic acid, sodium thiosulfate, sleeping pills, glucose, vitamins of group B, ascorbic acid, cardiovasotonics are also recommended. In the case of psychotic disorders, antipsychotics are used. Contraindications are morphine and bromine drugs, because the cortex of the cerebrum hemispheres is depressed and at the same time the hypothalamic area is excited.

Prevention includes the following measures:

1. Sealing of the zone with the presence of lead aerosols, thermal power plant steam, rational ventilation of work premises.

2. Water washing and treatment of the skin with petroleum or kerosene in case of TPP contact.

3. Appropriate sanitary treatment of things, if they were contaminated by TPP.

4. Not eating in the work area.

5. Compliance with safety rules, periodic examination of persons who work with lead and TES (a neurologist and a therapist examine twice a year).

Carbon monoxide poisoning

Carbon monoxide is a colorless gas produced during the incomplete combustion of various fuels.

Pathogenesis. Enters the body by inhalation, is a blood poison. Interacts with hemoglobin, displaces oxygen, forms carboxyhemoglobin with a sharp decrease in blood oxygen capacity, development of hemic hypoxia. Compensatory hyperventilation leads to hypocapnia and thus inhibits the activity of the respiratory center. Secondary is inhibition of tissue respiration.

Due to the fact that the central nervous system is the most sensitive to hypoxia, disorders of its functions prevail. The cortex and striopallidary system are most affected. Vascular cerebral disorders are also significant.

Pathomorphology. In the case of a quick fatal end, vascular disorders predominate — expansion, stasis, hyperemia of membranes. If the victim has been in a coma for a long time, necrobiotic foci will be observed in the area of the subcortical nuclei and in the white matter of the

cerebral hemispheres, the death of neurons with their replacement by ganglion elements mainly in the area of the globus pallidus; cerebral edema.

Clinic. Acute poisoning. A mild form of intoxication occurs with the clinic of "ring" headache, nausea, vomiting, palpitations. The skin and mucous membranes are pink due to the accumulation of carboxyhemoglobin in the blood. Tachycardia and tachypnea are observed. The content of carboxyhemoglobin in the blood is 20–30%.

Moderate poisoning is accompanied by fainting followed by excitement, visual or auditory hallucinations, or stupor, adynamia, arterial hypertension, tachycardia. Cardiotoxic phenomena (arrhythmia, myocardial ischemia, etc.) may be observed. Often there are trophic skin changes (pigmentation with preceding erythema or bulbous formations filled with transudate, located according to the type of hemiolesion). On the part of the respiratory system — tracheobronchitis, in severe cases — pulmonary edema on the background of toxic pneumonia.

Severe poisoning is characterized by a comatose state with trismus, significant rigidity of the muscles of the trunk and limbs, tonic convulsions, pathological reflexes, impaired external breathing with the possible development of Mendelssohn's syndrome, toxic cardiomyopathy, myocardial ischemia, trophic skin disorders. The concentration of carboxyhemoglobin in the blood is 50% or higher.

Coma lasting more than 48 hours is a poor prognostic sign. The fatal end occurs as a result of paralysis of the respiratory center and progressive cardiovascular disorders. Exit from the comatose state is accompanied by a period of motor excitement, aggressiveness, followed by apalic syndrome. Sometimes after coming out of a coma, psychotic disorders develop, which recur for several years after intoxication. Parkinson's syndrome is characteristic of the residual period.

The clinic of chronic intoxication includes asthenic, vestibular and amiostatic syndromes.

Diagnosis and differential diagnosis are based on the characteristic clinical picture (bright pink color of the skin and mucous membranes, coma, Parkinson's syndrome), the presence of carboxyhemoglobin in the blood in an increased concentration, and professional history.

Treatment. It is necessary to remove the victim to fresh air, provide continuous inhalation of oxygen for 2–3 hours, in severe cases — HBO 2–3 atm for 50–60 min, mechanical ventilation according to indications, inhalation of carbogen. In case of development of pulmonary edema — antifoam agents, ganglioblockers (in case of increased AT). Vitamin therapy and sedatives are also recommended. In order to accelerate the dissociation of carboxyhemoglobin — ultraviolet radiation of blood.

Prevention consists in adequate ventilation of rooms in which furnaces are located and internal combustion engines work; sealing of production zones with the release of carbon monoxide.

Intoxication by organochlorine compounds

The substances of this group include some insecticide fungicides (DDT, hexachloran).

Pathogenesis. The compounds do not dissolve in water, dissolve well in lipids, enter the body through the respiratory tract and intact skin. They accumulate in lipid depots, are capable of cumulation, due to their lipotropic properties, they accumulate in nervous tissue, where they block oxidative processes.

Pathomorphology. Hyperemia and microhemorrhages in internal organs, dystrophic processes in the cerebellum, medulla oblongata, cerebral cortex and subcortical nuclei.

Clinic. Against the background of symptoms of irritation of the mucous membrane of the upper respiratory tract and general intoxication and enterotoxic phenomena, neurological disorders appear in the form of encephalomyelitis syndrome (nystagmus, coordination insufficiency, spastic tetraparesis), polyneuropathic syndrome. In case of chronic intoxication, asthenovegetative, polyneuropathic, cardiovascular and liver syndromes are observed.

Diagnosis, differential diagnosis. It is necessary to carry out differential diagnosis with diffuse encephalomyelitis, amyotrophic lateral sclerosis and other disseminated forms of damage to the central nervous system.

Treatment includes the following measures:

1. Evacuation of the victim from the contaminated area.

2. Treatment of the skin with an alcohol-based solution (25 ml of ammonia per 5 liters of water); mucous membranes are moistened with sodium bicarbonate solution.

3. Stomach lavage using a probe, use of activated charcoal, saline laxatives.

4. Symptomatic therapy depending on the leading syndrome.

Prevention. Compliance with safety rules when working with poisonous chemicals.

Intoxication with organophosphorus compounds

Organophosphorus compounds (FOCs) are effective insecticides. These include thiophos, karbophos, chlorophos, etc.

Pathogenesis. FOS enter the body through intact skin, respiratory tract, digestive tract. Suppress cholinesterase and cause disorders of synaptic transmission. The effects are divided into three groups: muscarinic; nicotinic; damage to the central nervous system. In the latter case, the predominant location of the substance is hypothalamic-stem formations.

Pathomorphology. Hyperemia with microhemorrhages of the membranes and substance of the brain, edema and swelling mainly of subcortical areas.

Clinic. Acute and subacute poisoning go through three stages of development:

the first stage — psychomotor excitement, miosis, nausea, hypersalivation, moist rales in the lungs, hyporeflexia;

the second stage — body temperature up to 40 °C, stagnant enlargement of the liver, diplopia, horizontal nystagmus, individual or generalized muscle fibrillations, clonic-tonic convulsions, soporotic state;

third stage — coma; death occurs as a result of asphyxia, paralysis of the respiratory center, cardiac arrest.

Chronic intoxication is manifested by asthenovegetative syndrome with sharp parasympathotonia.

Diagnosis, differential diagnosis are based on the characteristic parasympathotonic effect of compounds (increased secretion of biological secretions, miosis, bradycardia, hyporeflexia), anamnestic data.

Treatment. It is necessary to evacuate the victim from the contaminated area. Multiple gastric lavage, fatty laxatives, siphon enemas, forced diuresis are recommended. In the second and third stages — early hemodialysis, hemosorption on the first day after poisoning. Antidote therapy: in the first stage — 2–3 ml of 0.1% solution of atropine sulfate intramuscularly, 2 ml of 25% solution of aminazine intramuscularly, 10 ml of 25% solution of magnesium sulfate intramuscularly. Atropinization until dry mouth during the day. In the second stage — 3 ml of a 0.1% solution of atropine sulfate in a 5% solution of glucose intravenously repeatedly until regression of bronchorrhea. In case of severe hypertension and convulsions — benzohexonium, sibazone, dipiroxime. Atropinization for 3–4 days. The third stage is mechanical ventilation, 30–50 ml of 0.1% atropine sulfate solution intravenously. Treatment of toxic shock. Blood replacement on the 2nd — 3rd day after poisoning in case of low cholinesterase activity and cardiac conduction disorders. Atropinization for 6–8 days.

Prevention. Compliance with the rules of personal hygiene, use of insulating overalls.

Gasoline (petroleum) intoxication

Gasoline is a volatile substance. It enters the body through the respiratory tract, sometimes orally, and can also be absorbed through the skin. It is eliminated by the lungs. A significant role is played by the speed of saturation of the blood and the central nervous system. It is a psychotropic poison, in case of exposure to significant concentrations, the brain stem parts are affected, the development of paralysis of the respiratory center is possible.

Pathomorphology — hyperemia and swelling of the meninges, diffuse hemorrhages.

Clinic. In the case of a severe instantaneous form of poisoning, loss of consciousness and reflex respiratory arrest, convulsions, increased body temperature, facial hyperemia, and the smell of gasoline from the mouth are observed.

Mild and moderate intoxication resembles a state of intoxication with dizziness, unsteadiness while walking, tremors of the limbs.

Chronic poisoning is characterized by asthenovegetative syndrome.

Treatment. Stopping contact with gasoline, washing the stomach using a probe with vaseline oil (200 ml), using activated charcoal. Symptomatic therapy according to indications: oxygen inhalation, cardiotonic drugs, vitamin therapy (vitamins of group B, ascorbic acid, tocopherol acetate), respiratory analeptics.

Prevention. The use of insulating clothing in rooms where there are gasoline vapors, a ban on pouring gasoline by forming a siphon system with sucking gasoline by mouth.

Poisoning with ethyl alcohol

Intoxication occurs when drinking too much alcohol.

Pathogenesis. Ethyl alcohol belongs to narcotics of the lipid series. It is quickly (within 40–90 minutes) absorbed in the stomach and small intestine (20 and 80%, respectively).

It is oxidized with the formation of acetaldehyde and acetic acid, excreted by the lungs and kidneys within 7–12 hours. Thiamine is needed for its oxidation, therefore, chronic intoxication is characterized by thiamine deficiency. The process is facilitated to some extent by gastroenteritis, in which hyporesorption of vitamins of group B is observed. Ethyl alcohol directly affects the metabolism of neurons with the secondary involvement of autoimmune and other mechanisms in the event of intercurrent diseases. Ethanol metabolism products also have a toxic effect on the body. In acute cases, subcortical autonomic centers and extrapyramidal nuclei are inhibited, followed by inhibition of their functions.

Pathomorphology. The phylogenetically youngest cortex of the cerebrum is the first to suffer, where degeneration of ganglion cells is observed. Most often, polyneuropathy with manifestations of segmental periaxial neuritis (degeneration of myelin, starting from the outer layer and having an ascending character) develops.

Clinic. Acute intoxication occurs when toxic doses are used with appropriate concentrations (toxic — 15 g/l, lethal — 35 g/l) in the blood.

Initial signs: parasympatheticotonia (facial hyperemia, hypersalivation, hyperhidrosis, mydriasis, polypolakiuria).

Further, the clinical picture is complemented by coordination disorders, hypomimia, oculomotor and cerebellar disorders, the criticality of one's condition decreases. In case of moderate poisoning, subcortical unconscious activity is inhibited. After that comes sleep. In case of severe poisoning, an alcoholic coma occurs, in which three stages are distinguished.

The first stage is superficial coma with hyperreflexia. Facial hyperemia, hypersalivation, tachycardia, shortness of breath are characteristic. The reaction to painful stimuli is undifferentiated. EEG shows disorganized fast-wave activity.

The second stage is superficial coma with hyporeflexia of tendon and periosteal reflexes. Activity with D-wave bursts is recorded on the EEG.

The third stage is a deep coma, characteristic smooth movements of the eyeballs, cyanosis, hypothermia, tonic convulsions, involuntary urination and defecation. EEG shows sinusoidal D-activity.

Acute liver and kidney failure can be a complication of coma.

The fatal end occurs as a result of acute cardiovascular and respiratory failure.

Pathological intoxication is distinguished. It occurs if a small amount of alcohol is consumed. Characteristic mental disorders of the epileptoid (state of pathological affect with aggressiveness without external provoking factors) or paranoid (hallucinatory-delusional symptoms with delusions of persecution) type.

Chronic alcohol poisoning occurs in case of regular alcohol consumption. Dose dependence is completely individual. Chronic intoxication with ethyl alcohol has the following characteristic features:

1. Asthenovegetative syndrome: decrease in spontaneous mental activity, deterioration of memory, deformations of behavior, decrease in emotional tone, parasympatheticotonia.

2. Polyneuropathic syndrome: there are sensitive, pseudo-betic and vegetative forms. It develops after a prodromal period lasting 1–7 days (paresthesias in the distal parts of the limbs, pain in the lower legs). After that, there are distal motor disorders with amyotrophy, the predominant damage to the extensors of the hands and feet. Further, on the background of motor, sensitive disorders develop (hyperesthesia, hypoalgesia with paresthesia, sensitive ataxia). In severe cases, the phrenic and vagus nerves are affected with paralysis of the diaphragm and cardiac disorders. Peripheral vegetative disorders are characterized by changes in the color of the skin of the distal parts of the limbs.

3. Wernicke-Gaye encephalopathy is caused by acute or subacute lesions of the midbrain or hypothalamus due to vitamin B1 deficiency. Early signs are vomiting, nystagmus, dizziness with subsequent disorders of consciousness (confusion, delirium, often against the background of Korsak's syndrome). Paralysis of oculomotor muscles, cerebellar ataxia. Body temperature is reduced due to damage to the posterior hypothalamus. A comatose state occurs, and if therapy is not started in a timely manner, 50% of patients die within 8–14 days. In case of withdrawal from coma, paresis of oculomotor muscles and ataxia are observed.

4. Korsak's syndrome (amnestic) is manifested by a combination of polyneuropathy with impaired memory for the recent past with relative preservation of it for events of the distant past, secondary disorientation in place and time. The essence of the individual is preserved.

5. Alcoholic epilepsy is formed in the presence of alcoholism, according to the clinic of epileptic seizures, it does not differ from genuine epilepsy, it occurs at the height of drunkenness and during abstinence.

6. Alcoholic cerebellar degeneration is characterized by dynamic and static ataxia, mainly in the lower extremities. It progresses for several months, after which it has a stationary course. Morphologically, degeneration of all neuronal elements of the cerebellar cortex and olives is observed.

Diagnosis, differential diagnosis. Acute conditions are diagnosed by the regression of neurological symptoms in accordance with the elimination of alcohol from the blood, the presence of the smell of alcohol from the mouth, the detection of ethyl alcohol in the blood, characteristic changes on the EEG, and the presence of a coma. Chronic intoxication should be differentiated from encephalopathy, polyneuropathy, epileptic syndrome of various genesis.

Treatment. In case of acute poisoning, gastric lavage, saline drinks, laxatives, forced diuresis, if necessary, ventilatory support, analeptics, intravenous hypertonic glucose solution, administration of group B vitamins, ascorbic acid.

In case of chronic intoxication, stop drinking alcohol, detoxification therapy, group B vitamins, ascorbic acid, biostimulants, anticholinesterase agents, physical therapy and spa treatment.

Vibration disease is an occupational disease, characterized by a variety of clinical symptoms, a peculiarity of the course; caused by the influence of local (local) and general vibration.

Etiology and pathogenesis

The main etiological factor is industrial vibration. It is often accompanied by other occupational hazards (noise, cooling, static tension of the muscles of the shoulder girdle, forced tilting of the body, etc.). The disease is manifested by disorders of the nervous, cardiovascular systems, and musculoskeletal system. Local and general vibration, being a strong stimulus, affects the receptor apparatus of the skin and nerve trunks, leads to increased secretion of norepinephrine at the terminals of the sympathetic nervous system. An excess of norepinephrine cannot be

completely taken up and accumulated in the terminals, therefore a significant part of it enters the blood, which causes an increase in the tone of blood vessels, leads to an increase in blood pressure and angio-spasm. Under the influence of vibration, destructive phenomena occur in the bodies of Father Pacino, nerve fibers, neurons of the spinal cord, reticular formation of the brain stem, spinal cord and ganglia of sympathetic border trunks. A decrease in afferent innervation is noted, especially the perception of vibration sensitivity. As pathological changes develop in the somatoneurological and vegetative apparatus, dystrophic changes occur in the skin, muscles, and bone system. The receptors of the large joints of the shoulder girdle are especially affected, which causes their pain. Significant importance in the pathogenesis of the disease has a strong influence of the vibration stimulus on the sympathetic-adrenal system, which also leads to angiospasm already in the initial stage of the disease. During the formation of the decompensated phase, persistent arterial hypertension develops, mediator exchange and other biochemical disorders are disturbed. Vegetative disorders affect the regulation of the activity of the gastrointestinal tract, which leads to gastrointestinal dyskinesia, and in the later period - to more severe pathology.

Pathomorphology

Changes in the nervous system are characterized by destructive phenomena in the bodies of Father-Pacino, encapsulated receptors, various types of deformation at the level of neuromuscular receptors, focal demyelination and disintegration of axial cylinders. In the experiments, dystrophic changes were found in the cells of the lateral horns of the spinal cord and the reticular formation of the brain stem.

Clinical picture.

When exposed to local vibration in persons working with manual mechanized tools, four stages of vibration disease are distinguished. In the I stage, temporary pain, paresthesias and numbness occur in the fingers. In the II stage, pain and paresthesias are persistent, changes in vascular tone (capillaries and larger vessels), pronounced sensitivity disorders (vibration sensitivity is especially reduced). symptoms of vegetative-vascular dystonia and asthenia appear . In the III stage, vasomotor and trophic disorders become pronounced, there are attacks of pain, numbness and paresthesia, a pronounced syndrome of vasospasm - whitening of the fingers, mixed sensitivity disorders (peripheral, often segmental). Characteristic are the complete loss of vibrational sensitivity, suppression of deep reflexes, neuroticism of the personality according to the type of asthenia, vegetative-vascular dystonia with increased blood pressure, hyperhidrosis. Gastrointestinal disorders are noted. X-rays reveal changes in joints and bones. In the IV stage, diffuse organic lesions of the brain and spinal cord (encephalomyelopathy), severe trophic and sensitive disorders develop. Pains in the fingers, along the course of the nerve trunks and in the joints are persistent. There are microfocal neurological symptoms, autonomic paroxysms, which flow mainly along the sympatho-adrenal and less often - of the mixed type. Angiodystonic crisis covers not only the peripheral vessels of the hands, but also the area of coronary cerebral vessels.

Vibration disease, caused previously by the influence of general and local (local) vibration, occurs in those working on vibro-compaction of concrete. In this form, the angiopolyneuropathic syndrome is combined with the development of neurasthenia with a sharp weakening of inhibitory processes. Complaints of irritability, headaches, dizziness, increased sensitivity, aching pains in the legs, numbness, paresthesias prevail. In the later period, headaches become constant, autonomic crises join (feeling of nausea, tachycardia, lack of air, fear of death, thermoregulation disorders). Neurological symptoms are also accompanied by "failures" in memory, tearfulness, and sleep disturbances. Attacks with pallor of the toes, diffuse sweating are often observed. Angiopolyneuropathic symptoms appear mainly in the legs, vibration sensitivity is disturbed, paresthesias, etc. Deep reflexes are first revived, then suppressed, trophic disorders develop in the form of thinning of the skin on the toes, muscle hypotrophy. Blood pressure rises moderately, ECG changes. Foci of epileptiform activity are detected on the EEG.

Vibration disease caused by exposure to general vibration and jolts manifests itself in vestibulopathy (dizziness), headaches, changes in vision and hearing, trunk-spinal symptoms,

gastrointestinal dyskinesias, abdominal pain, especially in the solar plexus, radicular pain, mainly in the lumbosacral region. region

Diagnosis and differential diagnosis

In determining the diagnosis, the main importance is given to professional anamnesis, sanitary and hygienic characteristics of working conditions. A detailed examination of the patient using modern neurophysiological and neuroimaging methods is necessary. A comprehensive examination is especially important during dispensation to identify the initial manifestations of the disease, as well as the functional capabilities of the body. During the examination, pay attention to the color of the skin of the fingers and toes, and measure the skin temperature. Special attention is paid to the study of sensitivity (vibration, pain). Bone-joint, muscular and cardiovascular systems are also studied. Cold tests, capillaroscopy, thermometers, electroencephalography, electroneuromyography, studies of the cardiovascular system, polycardiography, mechanocardiograph, and oscilloscope are used.

Vibration disease should be differentiated from other diseases of non-professional etiology (Raynaud's disease, syringomyelia, vegetative polyneuritis, myositis, etc.)

Treatment

Treatment begins at the first manifestations of the disease. The etiological principle consists in the temporary or permanent elimination of the effect of vibration on the body, a gentle regime with regard to physical exertion and cooling. Pathogenetic therapy should be complex with the use of medicinal and physiotherapeutic effects. Of the drugs, ganglioblocking agents (pachicarpine) in combination with small doses of cholinolytics (gliatilin, spasmolitin, metamyzil) and vasodilators (cavinton, nicotinic acid, calcium channel blockers) give the greatest effect. Pyrroxan is successfully used for vegetative paroxysms. Tonics, administration of 40% glucose solution or calcium gluconate, vitamins of group B, E, nootropic drugs are shown.

Electrophoresis of various drugs (5% solution of novocaine or 2% aqueous solution of benzohexonium) is applied to the hand, feet or collar area. The current strength is 10-15 mA, the duration of the procedure is 10-15 minutes.

With polyneuropathic syndromes, the effect of using high-frequency electrotherapy, needle reflexotherapy is obtained.

The prognosis is favorable with timely detection of the disease and active therapy. Employment is quite an important factor contributing to the full recovery of working capacity. All types of work are shown, excluding vibration, lifting loads and cooling.

Prevention.

An exception to the adverse effect of vibration on the body. Improvement and creation of fundamentally new tools and equipment that limit vibration within permissible limits. Carrying out dispensation, organization of prophylactics at factories, compliance with strict norms of **labor organization.**

Materials for self-control of training quality

Tests

1. Which cranial nerve is affected by methanol intoxication?
A. Lytsovyi
B. Threefold
B. Additional
G. Zorovy
D. Glossopharyngeal Answer: H

2. What kind of polyneuropathy most often occurs in intoxication with salts of heavy metals?
A. Motorna
B. Sensory

B. Vegetative Answer: B

3. What drugs are the most effective for intoxication with salts of heavy metals?
A. Vitamins of group B
B. Haemodesis
V. Sechoghinni
M. Kompleksony Answer: H.

4. What is the methanol intoxication clinic?

A. Damage to the facial nerve
B. Hemiparesis
B. Lesions of the frontal lobes
G. Intentional tremor
D. Damage to the optic nerves
Answer: V, D.

5. What symptoms are characteristic of vegetative polyneuropathy?
A. Nystagmus
B. Absence of abdominal reflexes
B. Discoloration of the temporal halves of the discs of the optic nerves
D. Pronounced pain syndrome
D. Trophic disorders Answer: G, D.

A 25-year-old patient noted a worsening of his health, dry mouth, flatulence, and nausea after consuming canned food.
Objectively: impaired function of the oculomotor nerves, diplopia, mydriasis, unstable in the Romberg position, frequent, threadlike pulse
Question:
1. Name the diagnosis of the disease?
2. Where should this patient be treated?
Answers:
1. Botulism.
2. In an infectious disease hospital.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

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Practical lesson No. 20

Topic: Neurological aspects of brain injury. Spinal injury

Purpose: to create an idea of the classification, pathogenesis of craniocerebral injury, symptoms of damage to the brain and spinal cord in case of a closed injury, to teach applicants to examine a patient with TBI, determine the localization of the pathological focus, assess the severity of TBI and the data of paraclinical examinations, make the correct clinical diagnosis, and prescribe effective treatment.

Basic concepts: closed brain and spinal cord injuries are an important medical problem. Features of the clinical course with the presence of many complications, and the very pathogenesis and pathomorphology of traumatic diseases of the nervous system dictates the need for emergency care, requires a quick diagnosis. Therefore, neuropathologists, resuscitators, neurosurgeons, forensic doctors, as well as doctors of any other specialty must know this pathology, which is urgent, to be able to correctly and timely examine the victims and provide them with effective assistance.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):

- Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

The classification of TBI is based on the determination of the nature and degree of damage to the brain and spinal cord, since the clinical course, treatment and its consequences largely depend on this.

There are 7 main clinical forms of craniocerebral injuries.

1. Concussion.
2. Mild cerebral contusion
3. Contusion of the brain of a moderate degree.
4. Severe cerebral contusion.
5. Compression of the brain against the background of its slaughter.
6. Compression of the brain without accompanying slaughter.
7. Diffuse axonal injury of the brain

When analyzing a closed injury of the brain and spinal cord, attention should be paid to the duration of loss of consciousness, the presence of amnesia, vegetative disorders. To determine focal symptoms, the reflex, motor, sensitive, coordinating, psychoemotional spheres, the presence of meningeal symptoms should be carefully examined.

Early and late complications of traumatic CNS disease should be remembered. Therefore, along with clinical, paraclinical examinations of patients should be widely used. At the same time, considerable attention is paid to radiography, CT, MRI, lumbar puncture, etc., in order to determine the tactics of conservative or operative treatment and further rehabilitation measures

Closed craniocerebral injuries are injuries in which there is no damage to the aponeurosis.

Open craniocerebral injuries are injuries in which there is damage to the aponeurosis.

Penetrating craniocerebral injuries are those in which the dura mater is damaged, or there is a fracture of the base of the skull with discharge from the nose and ears.

Non-penetrating TBI are injuries in which the dura mater is not damaged.

Brain concussion (соммотио сегебги) is the lightest form of brain damage. Changes in the central nervous system are reversible and are manifested mainly by general brain, autonomic and light scattered focal symptoms that quickly disappear.

Pathogenesis. There are a number of theories to explain the impairments caused by concussions. The vibrational molecular theory (Zh. Pti, 1771) explains the mechanism of damage by molecular changes in brain cells that occur as a result of vibration during injury. The vibration from the area of application of force spreads through the entire brain to the opposite side of the skull (principle of countershock). According to the vasomotor theory (Ricker, 1877), the main role is assigned to a long-term disturbance of cerebral blood circulation due to a disorder of the functions of the vasomotor centers (short-term vasospasm and ischemia of the brain are replaced by longer stagnant hyperemia of it). According to the hydrodynamic theory (Dure, 1876), the dynamic force of the shock leads to the movement of cerebrospinal fluid, which at the time of injury enters from the lateral ventricles into the III and then into the IV ventricle, irritating the adjacent centers and sometimes causing congestion, stretching, tearing of the walls of the ventricles brain

Clinic. The rapid development of symptoms after an injury is characteristic. Just as quickly, violations decrease and disappear. The main symptom is fainting, which, as a rule, is shallow and short-lived, often instantaneous, in severe cases lasting 10-20 minutes. In mild cases, reactions to painful stimuli persist against the background of unconsciousness. The absence of such reactions indicates a deep disturbance of consciousness and the severe nature of the injury. The course of the disease is determined by the dynamics of unconsciousness. If fainting is short-lived (patients

often do not notice it), the course is favorable. Short-term unconsciousness is replaced by stupor. At the same time, drowsiness, lethargy, indifference to others are observed, often - vomiting (as a rule, one-time) with previous nausea, which is not related to taking food. Vomiting occurs a few minutes or hours after the injury, less often - after a few days. A concussion in children may not be accompanied by fainting. In this case, the victims are mostly not hospitalized. The clinical picture of concussion is dominated by general brain symptoms: headache, general weakness, dizziness, nausea, vomiting, tinnitus, sleep disturbances. The Gurevich-Mann symptom appears (intensification of headache when opening the eyes and moving the eyeballs). Patients are irritated by bright light and noise. As a rule, a brain concussion is accompanied by vegetative and vestibular disorders in the form of dizziness, rush of blood to the head, increased sweating, especially of the palms, a feeling of heat, pronounced asthenia. A characteristic symptom is amnesia retrograde (loss from memory of events that preceded the injury) and anterograde (loss from memory of events that occurred after the injury). During an objective examination, individual scattered focal symptoms are most often detected, which quickly pass: horizontal small-amplitude, which quickly fades, nystagmus, sluggish reaction of the pupils to light; decrease or absence of abdominal and cremasteric reflexes, slight smoothing on one side of the nasolabial fold, moderate diffuse hypotonia of muscles without particular asymmetry, increase or decrease of deep reflexes, weakly expressed asymmetry of tendon reflexes, presence of pathological reflexes, trembling of the fingertips, difficulty speaking, sensitivity disorders etc. These neurological symptoms are quite dynamic: they can quickly weaken, disappear, be replaced by others. Breathing, as a rule, is not disturbed, sometimes labile with slight slowing or acceleration. Other indicators (body temperature, pulse, composition of blood and cerebrospinal fluid) without special deviations. Cerebrospinal fluid pressure in concussion victims remains normal in 60% of cases. In 30% of victims, its level increases (over 1.96 kPa, or 200 mm of water) and hypertensive syndrome develops. Clinically, it manifests itself as a throbbing headache. The pain is diffuse, intensifies in the morning, sometimes radiates to the eyeballs and temples. Irritability, restlessness, insomnia are observed. A decrease in the pressure of the cerebrospinal fluid (below 0.98 kPa, or 100 mm H2O) and the occurrence of hypotensive syndrome is observed in less than 10% of patients. The reason, most likely, is a decrease in fluid secretion by the vascular plexuses of the ventricles as a result of spasm of brain vessels or arterial hypotension. Intensification of headache and worsening of the condition after changing the patient's horizontal position to vertical are characteristic. Constant headache is significantly reduced if you lower your head. And that is why you can see how the patient hides his head under the pillow or lowers it from the bed to the floor.

The skin is pale, the mucous membranes have a cyanotic tint, sweating is increased. Increased fatigue, mental exhaustion, and lethargy come into view. Patients quickly tire during a conversation, eat. Deterioration of the general condition, increased headache, onset of nausea and vomiting after dehydration therapy are characteristic. Hypotensive syndrome is sometimes persistent and difficult to treat. The level of intracranial pressure changes; in particular, high pressure can change to low as a result of long-term uncontrolled use of dehydration therapy.

Diagnosis of a concussion can be complicated, especially in the acute period. The differential diagnosis of concussion and brain damage is based on the dynamics of symptoms. In the case of a concussion, the prognosis is favorable. Neurological symptoms, as a rule, completely disappear and patients recover, but in some patients residual phenomena of an asthenic or asthenovegetative character persist. Treatment. At the scene of the incident, the patient should be placed with the head slightly raised, unbutton the clothing that interferes with free breathing. In case of vomiting, the patient's head should be turned to the side, free the upper respiratory tract from mucus and vomited masses.

If there is a wound on the head, a bandage must be applied; in case of bleeding from a wound, especially an arterial one, it is necessary to press the wound to the bone with your hand through a bandage until the bleeding stops and hold it, changing your hand during transportation to a medical institution. Patients should be transported in a lying position. Every victim with a brain concussion needs inpatient treatment. Victims who do not need surgical treatment of head

wounds are hospitalized in the neurological department, if necessary, in the surgical profile department (neurosurgical, traumatological, surgical). X-rays of the skull must be performed in the hospital. Detection of cracks or fractures indicates a severe injury. The term of bed rest is set individually, depending on the patient's well-being and objective condition (most often - 7 - 14 days). One of the important factors in the treatment complex is the creation of conditions for physical and mental peace. The duration of sleep should be 10-13 hours a day. If necessary, mild hypnotics are prescribed. Taking into account the high sensitivity of brain cells to hypoxia, it is recommended to periodically inhale oxygen during the acute period of injury. Effective hyperbaric oxygenation. Analgesics are prescribed for headache relief. Agitation, general brain symptoms, insomnia, intense headache, nausea, vomiting in young and middle-aged patients are indications for the appointment of neuroleptics, ganglioblockers. Depending on the nature of vascular disorders in the brain, calcium preparations are prescribed. B vitamins, ascorbic acid, tocopherol acetate, a nootropic drug (aminalon, pyriditol, nootropil), cerebrolysin are prescribed to normalize metabolic and neurodynamic processes. Intravenous injections of 40 — 60 mm of 40% glucose solution are recommended. In case of damage to the soft tissues of the head, in order to prevent purulent complications, antibacterial therapy with prophylactic doses of antibiotics and sulfonamide drugs is carried out for 3-5 days. Lumbar puncture is performed on the 3rd — 5th day after the injury, if the signs of concussion do not decrease or increase or new ones appear. Cerebrospinal fluid pressure is determined and treatment is adjusted depending on its level. In case of increased pressure, dehydrating agents (mannitol, hypothiazide, diacarb, etc.) are prescribed. In case of its decrease, a large amount of liquid is injected (5-10% glucose solution, isotonic sodium chloride solution, polyglukin, reopolyglukin, peptides). To stimulate the production of cerebrospinal fluid by the vascular plexuses of the ventricles of the brain, 20 — 100 ml of distilled water are administered intravenously or vagosympathetic blockade is performed (every 3 — 4 days). To reduce the headache, you should slightly raise the tender end of the bed and lay the patient without a pillow. If such treatment is ineffective, perform a lumbar puncture with the patient in a sitting position and inject 20-30 ml of air without releasing cerebrospinal fluid. Air, having entered the ventricles, irritates the vascular plexus, which stimulates the production of cerebrospinal fluid. With a favorable course of the post-traumatic period, if subjective sensations normalize in the first days and neurological symptoms disappear, patients can be discharged on the 7th - 10th day. If at the end of this period headache, autonomic symptoms, dizziness, general weakness are observed, focal neurological symptoms persist, the patient's stay in the hospital is extended for up to 30 days or more. This applies mainly to victims with arterial hypertension, patients with diabetes, other chronic diseases. It is unacceptable to discharge patients who have not completed a full course of treatment. Often, after hospitalization, patients need semi-bed rest at home. Persons working at heights, in hot workshops, with moving mechanisms, and transport drivers need special attention. In case of dizziness, it is recommended to temporarily transfer them to another job. Sanatorium-resort treatment is indicated after 4 — 6 months. after an injury A feature of craniocerebral trauma, including mild, concussion in particular, is that in some victims, pathological phenomena gradually progress in the distant period. This applies primarily to vegetative-vascular reactions and disorders of mental activity. Chronic pathological processes, against the background of which a craniocerebral injury occurred, are often exacerbated. This can lead to disability, despite the fact that the patients were in good condition when they were discharged from the hospital. This course of the disease is explained by the fact that at the time of the injury and in the acute post-traumatic period, there is a deep violation of the mechanisms of self-regulation of the vital activity of nerve cells of the brain, which is not compensated or restored over time. The pathological process begins to progress with the increase of clinical phenomena. Identifying the mechanisms of these disorders should open to clinicians new ways of pathogenetically based treatment.

Brain contusion (contusio segebgi) is a serious injury compared to a concussion. At the same time, there are always foci (focus) of dead nerve tissue, from small areas of distention to

brain detritus flowing out of the wound. In the affected area, rupture of small vessels, impregnation (imbibition) of the brain substance with blood are observed. The brain tissue is more injured in the areas of application of force, it can be damaged by the anti-shock mechanism, as well as in other places: in the area of bony protrusions of the base of the skull, processes of the dura mater (sickle of the cerebrum, tent of the cerebellum). Most often, foci of damage are located in the area of the base and poles of the frontal, temporal, and occipital lobes. They can be single or numerous both on one side and on both sides. The sizes of foci are different, depending on the intensity of the mechanical impact on the brain. Stroke of the brain can be without compression and with compression.

Pathogenesis. Pathological protective reactions that occur after a blow develop primarily directly around the focus. These are perifocal reactions, the nature of which is determined by the size of the focus, its localization, the age of the victim and individual characteristics of the body. Secondary post-traumatic reactions can develop after some time. Their development in the brain stem structures is especially dangerous. Post-traumatic pathological changes consist of manifestations of a general brain and focal nature. However, if general brain manifestations always develop, then the signs of a focal lesion are unstable and may not even be detected (in the case of beating in the so-called silent zone of the brain). At the same time, the localization of the hammering can be detected only with the help of auxiliary research methods, the degree of severity of the hammering of the brain is mainly characterized by the depth and duration of unconsciousness. In the case of a mild stroke, unconsciousness is short-lived (minutes, tens of minutes), general brain disorders are observed for 2-3 days, focal symptoms, although not very pronounced, are quite persistent: slight paresis or reflex asymmetry, impaired coordination of movements, vestibular disorders, others symptoms, depending on the main lesion. Blood impurities are sometimes detected in the cerebrospinal fluid. Cerebral infarction of an average degree is characterized by the presence of pronounced general brain and focal signs, impaired functions of the cerebral hemispheres, which are often combined with stem disorders. Fainting lasts longer - up to several hours. Many patients have fractures of the skull vault and its base. Cerebrospinal fluid — with pronounced blood impurities. Severe cerebral contusion is characterized by prolonged (several days, even weeks) unconsciousness (deep coma), pronounced trunk symptoms with impaired vital functions, signs of damage to hypothalamic centers (disturbance of thermoregulation, metabolic disorders), gross focal symptoms. Severe brain damage often leads to death.

The clinic of mild and moderate concussion is often quite similar to the clinic of a concussion. For differential diagnosis, dynamic observations and the results of objective research methods are required. A severe beating is characterized by an acute development of phenomena immediately after the injury. In the coming days, due to secondary processes around the focus of the infection, symptoms may increase. Its regression occurs slowly and, as a rule, not completely, which is evidenced by the presence of persistent residual phenomena. All symptoms and syndromes of a concussion can be observed in a brain injury clinic, but persistent focal neurological symptoms of various degrees are necessarily present. Unlike a concussion, sometimes there may be motor agitation at first. In the case of a severe injury, breathing is accelerated, loud, shallow. Congestion in the lungs quickly joins. Violations of cardiovascular activity are less pronounced, stable and most often manifested by tachycardia, arterial hypertension, sometimes by bradycardia, arrhythmia. In response to stress, which is a trauma, there is a disorder of the function of thermoregulation centers, as a result of which the body temperature rises to 38°C or more in the first days. Vomiting is repeated. Meningeal symptoms, dilation or narrowing of the pupils, anisocoria are revealed. In dynamics, these symptoms can change. Pupil reaction to light is absent or weak, corneal, conjunctival, and pharyngeal reflexes are reduced. The condition of these reflexes is important for determining the prognosis. If there is no reaction of the pupils to light and no corneal reflexes during the day, this indicates damage to the brain stem structures and is an unfavorable prognostic sign. Focal symptoms are determined by the location and size of the lesion, the degree of hemodynamic and lycodynamic disturbances. Paresis and paralysis, convulsions

(local, general), loss of functions of cranial nerves, disorders of sensitivity and reflex activity, speech disorders in case of severe beating are characteristic. Along with expressed vegetative reactions, bulbar symptoms are often observed paralysis of sphincters, spinal areflexia.

An increased number of leukocytes is found in the blood in the case of moderate and severe blood clots. After an acute period of trauma, severe headache, marked retrograde and anterograde amnesia, often psychomotor agitation are characteristic. Recovery of consciousness after a long-term disturbance occurs slowly (sometimes after several weeks and even months), due to a period of severe stupor. The longer the unconsciousness, the longer the retrograde amnesia.

Electroencephalographic signs are rather labile, but may reflect cortical neurodynamics. The bioelectrical activity of the brain in the early period of injury is characterized by a diffuse decrease or slowing down of the delta rhythm. Against the background of diffuse changes in electrical activity, focal symptoms can be masked, but gradually, with the improvement of the condition, pathological activity in the form of slow waves is concentrated in the focus of maximum brain damage. As a rule, immediately after the injury, the displacement of the middle M-echo is not noticeable. In case of development of cerebral edema around the focus of the contusion, M-echo shift is possible, due to which there is a need to conduct additional studies (computed tomography, magnetic resonance imaging, angiography) for differential diagnosis with brain compression, which can be caused by an intracranial hematoma. Computer tomography is the most informative method of detecting contusion foci, determining their size and localization. In the case of a moderate degree of cerebral infarction, the majority of injured persons show focal changes in the form of high-density small inclusions not compactly located in the area of reduced density, or a moderate homogeneous increase in density is observed, which indicates the presence of small hemorrhages in the area of the injury or moderate hemorrhagic impregnation of the brain tissue without its gross destruction.

In the case of a severe beating, brain lesions are detected in the form of a zone of heterogeneous increased density (the volume of detritus significantly exceeds the amount of blood) or significant in size and distribution in the depth of the brain foci of intense homogeneous increased density with unclear boundaries (the volume of detritus is less than the amount of spilled blood and the volume of clots).

Diagnosis of cerebral infarction is often complicated, especially during the initial examination of the victim. Sometimes the final diagnosis is established only when the patient is discharged from the hospital. In connection with this, the main task is to conduct a comprehensive examination, compare the obtained results, conduct research into the dynamics of the disease.

Treatment. In the case of a mild stroke, the treatment tactics are the same as in the case of a concussion. In severe cases, medical measures are primarily aimed at normalizing breathing, heart activity, metabolism, especially water-electrolyte metabolism, body temperature, and nervous reactions. It is important to ensure a healthy diet and nutrition. To prevent pronounced perifocal processes, surgically remove all necrotized tissues if possible. Attempts to resect affected areas to the limits of intact brain tissue should be categorically rejected.

In case of respiratory failure, the treatment should be aimed at restoring and stabilizing spontaneous breathing, if necessary, normal ventilation of the lungs should be provided by artificial means. To restore the patency of the respiratory tract, the contents of the oral cavity and the nasal part of the throat are carefully suctioned. If the tongue is sunken, the lower jaw is pushed forward, the tongue is fixed with a tongue holder. If these measures are not effective, intubation is indicated (in the first 1-3 days), sometimes — tracheostomy, which makes it possible to systematically rehabilitate the tracheobronchial tree and, as indicated, artificial ventilation of the lungs. Early prevention of pulmonary complications is extremely important and is often the key to a favorable outcome of a severe injury.

Breathing exercises in combination with airway drainage are effective. To do this, after 2-3 minutes after the injection of a solution of sodium hydrogen carbonate with glycerin into the tracheostomy, the tender end of the bed is raised for 30-50 minutes. In this position, the contents are sucked out, after which a solution of antibiotics is injected into the trachea and inhalation is

carried out. This manipulation is repeated 3-4 times a day. The internal tracheostomy tube is periodically removed, observing asepsis, mechanically cleaned and boiled. Chest massage, as well as turning the patient in bed several times a day, are important in the prevention of pneumonia. Means that stimulate the function of the myocardium and increase vascular tone (corglycon, calcium chloride, ATP, cocarboxylase) are indicated to combat insufficient blood circulation. If tachycardia is pronounced, novocainamide is used. Neuroleptics (droperidol, diprazine, etc.) are used to lower blood pressure. In case of significant persistence of arterial hypertension, ganglioblockers (pentamine), antispasmodic and sympatholytic agents are added.

Since the activity of endocrine glands is inhibited at a body temperature above 380 C, the breakdown of proteins and metabolic disorders increases, antipyretics should be used. A 50% analgin solution is prescribed in combination with antipsychotics, cold on the main vessels (blisters with ice on the inguinal and inguinal areas), wrapping with wet cold sheets, blowing with a fan. It is not advisable to lower the body temperature below normal.

As a result of hypoxia and impaired hemodynamics, metabolic acidosis rapidly develops, for the correction of which 100-200 ml of 4% sodium bicarbonate solution is administered intravenously under control of the acid-base state. In the event of a severe injury, the regulation of the water-electrolyte balance is of great importance, because as a result of the release of potassium ions from the cells, sodium ions take their place, which retain fluid in the tissues and contribute to the development of edema and swelling of the brain. In this case, dehydration therapy is carried out: mannitol is administered intravenously at the rate of 1 g/kg of body weight, furosemide intramuscularly or intravenously — 1 — 2 ml 1 — 2 times a day, albumin, euphilin, magnesium sulfate, diacarb taking into account the administered and excreted fluid, dexazone. In an average patient with a body weight of 60 kg, 2,500 — 3,000 ml of fluid should be administered per day, diuresis should be at least 1,500 ml. The use of a glucose-potassium solution (potassium chloride — 4 g, glucose — 1000 ml of a 10% solution, insulin — 25 units) is effective in overcoming potassium deficiency and eliminating metabolic acidosis. The solution is administered intravenously, preferably twice. To improve microcirculation in the brain, the daily amount of liquid should include polyglukin, reopolyglukin, and reomacrodex. Repeated blood transfusions are recommended to combat anemia, normalize cellular metabolism, increase blood oxygenation, and stimulate enzymatic processes. B vitamins, calciferol, ascorbic acid, rutin are used. From the first days, drugs that contribute to the normalization of metabolic and energy processes in the central nervous system are prescribed: aminalon, cerebrolysin, piracetam (nootropil), pyriditol, later - proserin, aloe preparations, FiBS.

In case of impaired urinary function, it is necessary to stabilize blood pressure and blood flow in the kidneys. Hydrocortisone (300 — 500 mg) or prednisolone (90 mg per day), antispasmodics, anabolic steroids, drugs that improve microcirculation (pentoxifylline or trental, reopoliglucin), as well as heat on the kidney area, paranephric blockade are prescribed.

The patient's nutrition during the acute period is carried out with the help of a thin gastric probe, which is introduced through one nostril into the stomach and left for 1-2 days, then through the other nostril. The amount of food — 250 — 300 ml, the frequency of feeding — 4 — 5 times a day. food should be liquid, easily digestible, its energy value should be 10467-12560 kJ (2500-3000 kcal). Medicines can be administered through a probe. Feeding by mouth is allowed only after the act of swallowing is restored.

Recently, surgical treatment is more and more often used for brain damage. Decompressive trepanation of the skull does not give clearly defined positive consequences. The operation of choice is removal of the focus of the contusion followed by conservative therapy. Surgical removal is mainly subject to lesions in the area of the base, temporal and frontal lobes. The nature of trepanation is determined by the patient's condition. Preference should be given to bone-plastic trepanation. Resection trepanation is performed for patients in serious condition. After dissection of the dura mater and examination of the brain, the distended areas of the brain are washed and removed with an aspirator, bleeding is carefully stopped.

Diffuse axonal injury is characterized by widespread primary and secondary axon breaks in the semioval center, subcortical structures, corpus callosum, and brainstem, as well as point and small focal hemorrhages in these structures.

Clinically, pronounced oculomotor symptoms (paresis of looking up, different position of the eyes along the vertical axis, bilateral suppression or loss of pupillary reactions, violation of the formula or absence of the oculocephalic reflex, etc.) are manifested clinically against the background of a prolonged, multi-day comatose state. Violations of the frequency and rhythm of breathing, phenomena of decerebration, disorders of muscle tone (hormetonia or diffuse hypotonia, pyramidal-bottom-extrapyramidal asymmetric tetraparesis), pronounced autonomic disorders (arterial hypertension, hyperhidrosis, hypersalivation, etc.) are also observed.

In the future, a long coma turns into a persistent or transient vegetative state, the appearance of which is evidenced by the opening of the eyes spontaneously or in response to irritation. The vegetative state lasts from several days to several months. It is characterized by symptoms of functional and (or) anatomical disconnection of the large hemispheres and the brain stem. In the absence of manifestations of activity of the primary grossly damaged cerebral cortex, subcortical, oral-stem, caudal-stem and spinal mechanisms are inhibited, which is manifested by an unusual variety and dynamism of oculomotor, pupillary, oral, bulbar, pyramidal and extrapyramidal phenomena. This is a paradoxical expansion of the pupils to the light, floating eyeballs in the horizontal and vertical planes, a spasm of the gaze (more often down), trismus, synkinesis - chewing, sucking, grinding of the teeth, yawning and swallowing automatisms, adduct tonic spasms in the limbs, rotations of the trunk and head, motor stereotypies, hand tremors, etc.

After leaving the vegetative state, the neurological symptoms of disconnection are replaced mainly by withdrawal symptoms. Chief among them is the extrapyramidal syndrome (stiffness, hypomimia, bradykinesia, hyperkinesis, dyscoordination) in combination with mental disorders (untidiness, akinesia, absent-mindedness, aggressiveness, irritability).

Computed tomographic pattern of diffuse axonal damage changes over time. During the comatose state, an increase in the volume of the brain (due to edema—swelling) with compression of the ventricles and cisterns is determined. Against this background, diffuse small focal hemorrhages in the white matter of the hemispheres, corpus callosum, subcortical structures, and brain stem are detected.

At the stage of the vegetative state and during exit from it, foci of increased density (hemorrhages) become hypodense, edema disappears, and diffuse brain atrophy is observed.

Treatment tactics in the case of diffuse axonal damage are the same as in the case of a severe cerebral infarction.

Compression of the brain occurs at the time of injury (compressed fracture) or develops during hematoma formation. Compression (compression) of the brain is caused by a pressed fracture, intracranial hematoma, subdural hygroma, aggressive edema — swelling of the brain around the focus of the beating, pneumocephalus. 3a, sometimes the appearance of threatening clinical signs after a craniocerebral injury is distinguished by compression of the brain: acute — during the day; subacute - within 2 weeks. (2-14 days); chronic — after 2 weeks. As a rule, compression of the brain is associated with a stroke. Most often, the area of compression and the place of hammering coincide. Sometimes the focus of the brain beating corresponds to the place of the injury, and the hematoma is formed in the opposite part of the brain. An indented fracture is determined by palpation and visually. However, the final diagnosis requires an X-ray examination, which, in addition to the localization of the fracture, helps to establish the size of the fragments and the depth of their penetration into the skull cavity. With significant compression, symptoms of focal brain damage may be observed, in some cases epileptic seizures occur as a result of irritation of the cerebral cortex by bone fragments. If the fragments are pressed no less than the thickness of the bone, and there are even no clinical manifestations, the fracture is subject to surgical correction. This especially applies to patients of childhood, since compression of the brain can cause the emergence of a pathological focus and become a trigger for epileptic seizures

and other complications. The repositioning of the pressed fragments of the skull should be ensured as early as possible, surgical intervention can be delayed only in the case of a severe general condition of the victim with a violation of vital functions. An exception for surgical correction is a pressed fracture of the outer wall of the frontal sinus, since there is no compression of the brain. However, if there is a cosmetic defect, surgical intervention is extremely necessary. Compression of the brain due to a compression fracture (without a hematoma) is usually not life-threatening.

Brain compressions caused by hemorrhages in a limited area of the intracranial space are dangerous - hematomas, which occur in 2-16% of cases of craniocerebral trauma. There is no direct relationship between the severity of the injury and the occurrence of a hematoma. Often, large hematomas occur in the event of a light injury, even without unconsciousness (a blow to the head with a hand or a boxing glove, an accidental hit on a hard object or a sharp movement of the head), and vice versa, with a severe injury, an intracranial hematoma may not be present. An intracranial hematoma occurs in the first minutes or hours after an injury, but brain compression syndrome is not immediately apparent. It is believed that the clinical picture of compression of the brain caused by a hematoma begins to develop when the difference between the capacity of the skull and the volume of the brain becomes less than 8% (normally it is 8-15%). Traumatic hematoma can be single (more often) and multiple. Multiple hematomas can be located in layers (one under the other), mainly in the area affected by the traumatic factor (for example, epi- and subdural or intracerebral hematomas) or in different departments of the intracranial space — next to each other or in diametrically opposite departments. In this case, two or more subdural hematomas may occur at the same time, or one subdural and the other intracerebral, etc. Compression of the brain caused by an intracranial hematoma is characterized by a phasic course of the process: 1) craniocerebral injury with corresponding general brain, autonomic, and focal neurological symptoms; 2) light interval — the time from the start of bleeding to the appearance of symptoms of compression of the brain by a hematoma (its duration depends on the source and speed of bleeding, localization of the hematoma, volume of reserve intracranial spaces); 3) progressive worsening of the patient’s condition with increasing general cerebral (appearance or worsening of consciousness disorders, increased headache, repeated vomiting, psychomotor agitation), focal (appearance or worsening of hemiparesis, unilateral mydriasis, focal epileptic seizures, etc.) and dislocation (oculomotor disorders, bilateral pathological signs, coma, impaired breathing and heart activity) symptoms.

Depending on the background (concussion, brain damage of various degrees), on which traumatic compression develops, the light space may be widened, erased, or it may not be there at all. According to the clinical course, traumatic hematoma is divided into acute, subacute, and chronic. In the case of an acute hematoma, the victim's condition progressively worsens. If a coma develops after an injury, its depth gradually increases. If the victim's condition immediately after the injury was relatively satisfactory, then during the acute course of the hematoma, it quickly deteriorates, the stupor turns into sopor, and then into a coma. Different dynamics are observed in case of subacute and chronic course of hematoma. Here, a rather important clinical feature is the presence of a light interval, which can last several days, weeks, and even months. With the increase of the hematoma, the exhaustion of compensatory mechanisms, the increase of venous stasis, swelling and swelling of the brain, after a light interval, the symptoms of intracranial hypertension appear - headache, nausea, vomiting (more often at the height of the headache in the morning), restlessness, agitation, insomnia, hallucinations, delirium . In the future, the general condition deteriorates quickly, patients become apathetic, lethargic, then fall into pathological drowsiness, which is replaced by a soporose or comatose state. Focal symptoms appear early, primarily mono- and hemiparesis, convulsive seizures, decreased abdominal, then tendon reflexes on the side opposite to the hematoma, pyramidal pathological symptoms, decreased sensitivity. Pathognomonic and the second most important symptom is unilateral dilatation of the pupil, more often on the side of the hematoma. Congestive discs of the optic nerves may appear 3 days after the injury. Cerebrospinal fluid pressure is mostly elevated, sometimes normal or reduced, mainly in older patients. A decrease in pressure can also be caused by dehydration therapy. Thus, an

increase in the pressure of the cerebrospinal fluid gives reason to assume the development of an intracranial hematoma, but a decrease in pressure does not deny its presence. In the cerebrospinal fluid in the first days after the injury, an admixture of blood can be detected, later xanthochromia is determined.

Diagnosis of intracranial hematoma is based primarily on the dynamics of clinical phenomena. Their growth should be a warning about the formation of a hematoma. Particular importance should be attached to the progression of such focal symptoms as anisocoria, anisoreflexia, movement disorders, sensitivity disorder. The place of possible formation of an intracranial hematoma often corresponds to the site of a fracture (usually linear) of the bones of the skull. Differential diagnosis with cerebral infarction is often complicated; in the case of a combination of hematoma with brain damage, recognition becomes even more difficult. The greatest diagnostic difficulties arise in the case of hemorrhage in the posterior fossa of the skull. Reliable definitive signs of a hematoma of this localization are traumatic injuries in the neck-occipital region, violation of the integrity of the occipital bone, rapid increase in symptoms of damage to the brain stem and structures located in the posterior cranial fossa. Of decisive importance when establishing a final diagnosis in the absence of highly effective auxiliary methods of diagnosis (computer, magnetic resonance imaging) is the imposition of milling holes in the occipital region (if there is a bone fracture, the milling hole is placed on the fracture line). The main additional research methods that are used in case of suspicion of hematoma are computer tomography, magnetic resonance imaging, echoencephalography, carotid angiography, overlaying of diagnostic milling holes. Computed tomography helps to establish the number, localization, size of the intracranial hematoma, the degree of displacement and deformation of the ventricles of the brain, the degree of dislocation of the brain structures and compression of the brain stem, as well as the limits of the spread of perifocal edema or secondary hemorrhages around the center of the beating and at a distance from it. On the echoencephalogram, in the case of a lateral location of the hematoma, the displacement of the M-echo signal is determined in 90-95% of the victims, in other localization (in the area of the poles, between the hemispheres of the cerebrum), and in the case of a bilateral hematoma, the displacement is weak. The degree of displacement of the M-echo signal varies from 4 to 13 mm, and the greater it is, the more likely the diagnosis of hematoma. Displacement of blood vessels is detected angiographically. In the occipital projection, if the hematoma is located laterally, in addition to vascular dislocation, the presence of a vascular zone in the form of a sickle or a biconvex lens is characteristic, especially in the venous phase. Intracranial hemorrhages (hematomas) are divided into epidural, subdural, intracerebral, intraventricular, and subarachnoid hemorrhage (not a hematoma). Each species has its own characteristics of the development of the clinical picture. An epidural hematoma is an accumulation of blood between the inner plate of the skull and the dura mater. It accounts for 15% of all intracranial hematomas and occurs most often with fractures of the skull bones with damage to the meningeal vessels, mainly the middle meningeal artery and its branches, the blood peels off the dura mater from the skull bone. Peeling of the shell, as a rule, is limited to the places of the sutures of the skull. Due to the intensity of arterial bleeding, the hematoma increases rapidly, which leads to a life-threatening increase in brain and focal symptoms. The source of epidural hematoma can be damaged sinuses (upper sagittal, transverse and sphenoid-parietal), as well as numerous small vessels that penetrate from the shell into the diploic vessels of the bones of the skull, the latter are damaged in case of detachment of the dura mater at the time of injury, they bleed slowly . The hematoma, as a rule, does not reach large sizes, the clinical picture grows gradually. Epidural hematoma almost always occurs on the side of the injury, most often in the parietal-temporal region. An epidural hematoma, which is localized in the temporal area and posterior cranial fossa, has a particularly unfavorable course and causes the rapid development of dislocation symptoms.

A subdural hematoma is an accumulation of blood between the dura mater and the arachnoid membrane of the brain. It is spent much more often than epidural hematoma. It is formed mainly as a result of bleeding from the veins of the soft shell of the brain, which flow into the sinuses. At the time of injury, they are stretched and torn off in the places of confluence. In rare

cases, a subdural hematoma occurs as a result of an indirect injury (sudden movement of the head, a fall on the buttocks, etc.). Subdural hematoma forms more slowly than epidural hematoma, often spreads over several areas of the brain, sometimes over the entire cerebral hemisphere. Treatment of hematomas is surgical.

Order of the Ministry of Health of Ukraine dated April 25, 2006 No. 245 (excerpt)

Protocol
provision of medical assistance to patients with brain concussion
IKH-10 code: S06.0

Signs and diagnostic criteria

Concussion of the brain (TBI) ranks first in the structure of craniocerebral trauma by frequency, is found in 70-80% of patients with craniocerebral trauma, and refers to mild TBI. Pathomorphologically, SGM is characterized by mosaic microstructural changes only at the cellular and subcellular levels (plasma or cellular membranes, synapses).

In most cases, the severity of a patient with SMG in the acute period corresponds to 14-15 points according to the SHKG.

The clinical picture of SGM depends on individual characteristics and the age factor.

Clinical signs of SGM are:

1. General brain and meningeal symptoms (loss of consciousness after an injury from a few seconds to minutes, while alcoholic, narcotic or other intoxication should be excluded), amnesia, headache, nausea, one or more vomiting, symptoms of Sedan and Gurevich-Man.

2. Vegetative symptoms (most often observed are acrocyanosis, hyperhidrosis (especially palms), "capillary play", dermographism disorders, subfebrility (often with asymmetry phenomena), blood pressure instability, pulse lability - bradycardia, which is replaced by tachycardia, tremors, etc.

3. Microfocal neurological symptoms (mild, transient anisocoria, asymmetry of facial muscles, nystagmus, decrease in corneal reflexes, weakness of convergence, weakening or strengthening of tendon reflexes, weakness of convergence, decrease in abdominal reflexes, muscle hypotonia, static ataxia, Marinescu's symptom- Radovichi), which in the vast majority of patients lasts from a few hours to 3-4 days.

The most important diagnostic criteria of SGM, which make it possible to separate SGM from mild cerebral infarction, are the absence of fractures of the vault or the base of the skull, subarachnoid hemorrhage, as well as focal changes on CT (MRI).

The algorithm for providing medical care to a patient with TBI in the acute period is given in the appendix.

Conditions under which medical assistance should be provided

Patients with SGM are subject to examination and treatment in a neurosurgical, neurological department within the first three days after receiving an injury.

Diagnostics

Diagnostic measures include:

1. Neurological and somatic examination.
2. Ro-graph of the skull in 2 projections (front-back, side).
3. EkhoES.
4. LP with determination of LT and analysis of cerebrospinal fluid.
5. CT scan of the brain (when admitted to the hospital, when the neurological symptoms worsen, the condition worsens).
6. Determination of blood group, Rh factor.
7. General tests of blood and urine, RW.

8. Blood analysis for sugar and alcohol content. Rappoport's test.

Treatment

The main method is conservative treatment. Medical treatment includes dehydration or hydration according to CSF pressure, sedative, nootropic, vascular drugs, symptomatic therapy (according to clinical signs).

In case of damage to soft tissues, PHO wounds are performed, anti-tetanus toxoid is administered.

Efficacy criteria and expected results of treatment

Improvement of the general condition, regression of general brain and focal symptoms.

Approximate duration of treatment in neurosurgical and neurological departments - up to 3 days, for soft tissue injuries - up to 8 days.

Further treatment in outpatient conditions under the supervision of a neurologist.

Protocol
provision of medical assistance to patients with mild cerebral contusion
IKH-10 code: S06.9

Signs and diagnostic criteria

Mild cerebral contusion (CBI) ranks second in frequency in the structure of craniocerebral trauma, occurs in 10-15% of patients with craniocerebral trauma, and refers to mild TBI. Structurally, ZLS is characterized by grouped small-focal point hemorrhages or areas of local edema of the brain substance in the absence of damage to the meninges.

The severity of the patient with ZLS in the acute period according to the SHKG corresponds to 13-14 points in most cases.

Clinical signs of ZLS are:

1. General brain and meningeal symptoms - loss of consciousness after an injury from a few seconds to 30 minutes (at the same time, alcohol, drug or other intoxication should be excluded), amnesia, headache, nausea, one or more vomiting, symptoms of Sedan and Gurevich-Man.

2. Vegetative symptoms - acrocyanosis, hyperhidrosis (especially of the palms), "capillary play", violation of dermographism, hypofebrility (often with asymmetry phenomena), instability of blood pressure, lability of the pulse - bradycardia, which is replaced by tachycardia, tremor, etc. are most often observed.

3. Slightly pronounced focal neurological symptoms (mild, transient anisocoria, asymmetry of facial muscles, nystagmus, decrease in corneal reflexes, weakness of convergence, weakening or strengthening of tendon reflexes, weakness of convergence, decrease in abdominal reflexes, muscle hypotonia, static ataxia, symptom Marinesku-Radovichi), which in most patients lasts up to 14 days.

The diagnostic criteria of SCI, which make it possible to separate SCI from a concussion, are the presence of fractures of the vault or base of the skull, subarachnoid hemorrhage, as well as focal changes on CT (MRI).

The presence of prolonged (hours) loss of consciousness, pronounced focal and/or trunk symptoms, CT (MRI) signs of a widespread focus of slaughter or intracranial hemorrhage indicate a more severe TBI, the criteria for which are described in the relevant protocols.

The algorithm for providing medical care to a patient with TBI in the acute period is given in the appendix.

Conditions in which medical assistance should be provided

Patients with ZLS are subject to inpatient examination and treatment in a neurosurgical or

neurological department.

Diagnostics

Diagnostic measures include:

1. Neurological and somatic examination.
2. Ro-graph of the skull in 2 projections (front-back, side).
3. ECHOES, EEG (on the 5-7th day).
4. LP with determination of LT and analysis of cerebrospinal fluid.
5. CT scan (MRI) of the brain (when admitted to the hospital, when the neurological symptoms worsen, the condition worsens).
6. Determination of blood group, Rh factor.
7. General tests of blood and urine, RW.
8. Blood analysis for sugar and alcohol content. Rappoport's test.

Treatment

The main method is conservative treatment. Medical treatment includes dehydration or hydration according to CSF pressure, sedative, nootropic, vascular drugs, symptomatic therapy (according to clinical signs).

In case of damage to soft tissues, PHO wounds are performed, anti-tetanus toxoid is administered.

Efficacy criteria and expected results of treatment

Criteria by which the decision to discharge from the hospital is made: improvement of the general condition, regression of general brain and focal symptoms.

Approximate duration of treatment in inpatient conditions - up to 8-14 days. Further outpatient treatment under the supervision of a neurologist.

Protocol

provision of medical assistance to patients with cerebral contusion of moderate degree of severity IKX-10 code: S06.3

Signs and diagnostic criteria

Cerebral contusion of a medium degree (SCM-SS) occurs in 8-10% of patients with craniocerebral trauma and refers to severe TBI. Pathomorphologically, ZGM-SS is characterized by foci of hemorrhagic softening or hemorrhagic impregnation of the brain tissue with small focal hemorrhages with preservation of the integrity of the configuration of furrows and convolutions.

In most cases, the severity of a patient with SGM-SS according to the SHKG corresponds to 9-12 points.

Clinical signs of ZGM-SS are:

1. General brain and meningeal symptoms (loss of consciousness after an injury from several tens of minutes to several hours (at the same time, alcohol, drug or other intoxication should be excluded), amnesia, headache, vomiting (in most cases multiple), mental disorders, etc. Kernig and rigidity of the occipital muscles).
2. Pronounced focal symptoms determined by the location of the focus of slaughter (pupillary and oculomotor disorders, limb paresis, sensitivity disorders, speech, etc.).
3. Individual trunk symptoms.
4. Transient, transient disturbances of vital functions are possible (bradycardia or tachycardia, increased blood pressure, tachypnea.
5. A characteristic CT feature of ZGM-SS is the presence of foci of brain lesions. In some cases, there is no focal traumatic substrate on the CT scan of the first day after the injury. With ZGM-SS, as a rule, there are no signs of compression and dislocation of the brain, the lateral displacement of the middle structures does not exceed 5 mm, the basal cisterns are not deformed.
6. Fractures of the bones of the vault and the base of the skull, massive subarachnoid

hemorrhages are often found with ZGM-SS.

The most important diagnostic criteria of ZGM-SS, which make it possible to separate ZGM-SS from milder and more severe lesions of the brain, are the characteristic duration of loss of consciousness (up to several hours, provided that alcohol, narcotic, other intoxication is excluded), the presence of a focus visualized by CT slaughter and persistent (more than 1 day) and pronounced (paresis, paralysis) focal symptoms. The presence of a longer (tens of hours) loss of consciousness, gross trunk symptoms with impaired vital functions, CT signs of compression-dislocation phenomena indicate a severe cerebral contusion, the criteria for which are described in the relevant protocol.

The algorithm for providing medical care to a patient with TBI in the acute period is given in the appendix.

Conditions in which medical assistance should be provided

Patients with ZGM-SS are subject to inpatient treatment in the neurosurgical department.

Diagnostics

Diagnostic measures include:

1. Neurological and somatic examination.
2. Ro-graph of the skull in 2 projections.
3. EchoES (for 1, 3-5, 9-14 days) - in the absence of CT, MRI.
4. CT scan (MRI) of the brain (during hospitalization, as well as again before discharge), EEG (for 7-10 days).
5. LP with determination of LT and analysis of cerebrospinal fluid.
6. Determination of blood group, Rh factor.
7. General tests of blood and urine, RW.
8. Blood analysis for sugar and alcohol content. Rappoport's test.
9. Biochemical examination of blood (electrolytes, total protein) and determination of hematocrit, plasma osmolarity, SpO2.
10. Blood coagulation control.
11. Consultation of an ophthalmologist, otoneurologist (in dynamics). Before discharge – examination by a psychiatrist (according to clinical indications).

Treatment

The main method is conservative treatment according to algorithms of intensive therapy. Infusion therapy with positive fluid balance prevails.

In case of damage to soft tissues, PHO wounds are performed, anti-tetanus toxoid is administered. Repeated LPs before sanitation of the cerebrospinal fluid with measurement of cerebrospinal fluid pressure (in the absence of contraindications) are justified.

Surgical intervention is performed when a contusion center is formed with an increase in compression-dislocation manifestations - decompression trepanation, ventricular drainage, etc. (according to indications).

Medical treatment includes dehydration, nootropic, vascular, anticonvulsant drugs (as indicated).

Efficacy criteria and expected results of treatment

Lethality with ZGM-SS should not exceed 9%. The criteria by which the decision to discharge from the neurosurgical hospital is made - improvement of the general condition, partial regression of general brain and focal symptoms, resorption of the hemorrhagic component of the focal lesion according to CT data. Further treatment is in the neurological or rehabilitation department.

The estimated duration of treatment in the neurosurgical department and the intensive care unit is up to 20 days.

When the patient's condition is stabilized, continuation of treatment in the rehabilitation or neurology department is indicated.

Protocol for the provision of medical care to patients with a severe cerebral contusion, diffuse axonal injury Code IKH-10: S06.3, S06.2

Signs and diagnostic criteria

Cerebral contusion of a severe degree (SGM-TS) occurs in 5-7% of patients with TBI and refers to a severe craniocerebral injury. Pathomorphologically, ZGM-TS is characterized by gross destruction of the cortex and underlying white matter, reaching in some cases subcortical nodes and walls of the ventricles. As a rule, it is accompanied by fractures of the bones of the vault and the base of the skull, ruptures of soft membranes, massive subarachnoid hemorrhages. Intracerebral hematomas and hemorrhages are found in the focus of softening and complete destruction of the brain substance. Widespread swelling of the brain and pronounced hyperemia develop.

Diagnostics

If the patient has clinical signs of severe TBI, it is necessary to conduct a standard set of tests, which includes:

1. Neurological and somatic examination.
2. Ro-graph of the skull in 2 projections.
3. EchoEG (for 1, 3-5, 9-14 days) - in the absence of CT, MRI.
4. CT scan (MRI) of the brain (during hospitalization, as well as again before discharge).
5. Determination of blood group, Rh factor.
6. General tests of blood and urine, RW.
7. Blood analysis for sugar and alcohol content. Rappoport's test.
8. Biochemical examination of blood (electrolytes, total protein) and determination of plasma osmolarity, SpO2.
9. Coagulation monitoring.
10. Consultation of an ophthalmologist, otoneurologist (in dynamics). Before discharge – examination by a psychiatrist (according to clinical indications).

The clinical condition of a patient with ZGM-TS corresponds to 3-8 points according to the SHKG, the clinical picture is dominated by distinct brain and stem movements (floating movements of the eyeballs, gaze paresis, bilateral mydriasis, miosis, divergence of the eyeballs along the vertical and horizontal axes, swallowing disorders, bilateral pathological foot signs overlapping focal hemispheric symptoms) symptoms with impaired vital functions.

The combination of existing neurological changes makes it possible to distinguish clinical forms of ZGM-TS: extrapyramidal, diencephalic, mesencephalo-bulbar, cerebro-spinal.

The algorithm for providing medical care to a patient with TBI in the acute period is given in the appendix.

CT and MRI of the brain are the leading methods of diagnosis of ZGM-TS and diffuse axonal damage.

There are absolute (direct) and relative (indirect) CT features of ZGM-TS.

Direct signs include changes in the density of brain matter. A third of patients have foci of increased (64-74N), decreased (18-25N) density, isodense (the same density as intact brain matter) areas.

CT signs of ZGM-TS are the presence of significant (more than 30 cm3) foci of slaughter. Along with direct CT signs of ZGM-TS, the state of intracerebral structures is assessed: the presence of lateral, axial dislocation; shape and size of the ventricles of the brain.

With diffuse axonal damage, CT (MRI) examination reveals multiple foci (axoplasmic sources), which are mainly located paraventricularly, as well as in trunk structures.

To diagnose the possible formation of delayed focal ZGM-TS, to assess the dynamics of structural changes in the brain, repeated CT examinations are performed (on the 3rd-7th day of the

patient's stay in the hospital, in the future - as indicated).

Conditions under which medical assistance should be provided

Patients with ZGM-TS are subject to inpatient treatment in the intensive care unit under the supervision of a neurosurgeon.

Principles of treatment

Patients with ZGM-TS require treatment in intensive care units under the supervision of a neurosurgeon in accordance with intensive care algorithms.

Depending on the patient's condition, the CT (MRI) data, the study identifies the following variants of the clinical course in ZGM-TS, which require differentiated treatment.

1. Progressive – with an increase in the volumetric impact of the focus of slaughter. Absolute indications for surgical treatment.

2. Regressive – with normalization of the patient's condition, regression of intracranial injuries. Conservative therapy is justified.

3. Wave-like - periods of deterioration of the patient's condition are replaced by positive dynamics with partial regression of symptoms, stabilization of the patient at the level of subcompensation. Such patients are treated conservatively, and in the case of negative neurological dynamics, with the formation of chronic hematomas, hydroma, they are operated on according to the appropriate indications.

Surgical treatment

Indications for surgical intervention are:

Focal ZGM-TS with crushing of the brain substance, accompanied by dislocation of the midline structures > 5 mm, especially with the development of contralateral hydrocephalus, with compression of the basal cisterns, are subject to surgical intervention for the purpose of internal and/or external decompression - brain detritus is removed, decompressive trepanation, ventricular drainage is performed etc. (according to indications).

In the case of ZGM-TS combined with membrane hematomas, surgical intervention is indicated (see relevant protocols). The operation is carried out within 3 hours from the moment of establishing the indications until it is carried out.

Contraindications to operative treatment: 1) atonic coma with gross vital disturbances; 2) verified brain death according to the criteria defined by the regulatory documents of the Ministry of Health of Ukraine; 3) critical disorders of the blood coagulation system (thrombocytopenia - the number of platelets is 50·103 μl and below).

Types of operative interventions for ZGM-TS

Radical surgical interventions

Bone-plastic or decompressive trepanation is performed for patients with focal ZGM-TS. It is necessary to avoid resection of the focus of slaughter, which is significant in volume. Preference is given to methods of aspiration and washing of detritus with physiological solution. If there is no prolapse of the brain substance into the operating hole, the operation is completed by applying an inflow-outflow drainage system or installing passive subdural drains in the cavity of the removed focus of slaughter or hematoma (for 2-3 days).

Palliative operations

Ventriculopuncture with the installation of long-term external ventricular drainage is performed in patients with acute occlusive symmetric hydrocephalus.

With open acute hydrocephalus, it is advisable to establish a long-term external lumbar drainage.

In open normotensive or hypertensive hydrocephalus, accompanied by gross mental defects, pelvic organ dysfunction, long-term external lumbar drainage is justified, in the case of positive neurological dynamics, fluid shunting surgery (lumboperitoneal shunting with shunt installation at the appropriate optimal pressure).

The advisability of performing one- or two-sided wide (with a diameter of the trepanation

window more than 8 cm) decompression in patients with ZGM-TS is debatable, there is no reliable evidence of the effectiveness of such surgical intervention.

Efficacy criteria and expected results of treatment

Lethality with ZGM-TS should not exceed 40%.

The estimated duration of treatment in the neurosurgical department and the intensive care unit is up to 8 weeks. The quality criteria of the treatment are preservation of the patient's life with improvement of his general condition, partial regression of brain, focal and trunk symptoms.

Further treatment is carried out in the department of neurology or rehabilitation.

In the presence of bone defects of the skull, plastic surgery is performed after 3-12 months, with secondary wound healing - after 1.5-2 years.

Medical care protocol
patients with nasal liquorrhoea Code IKH-10: G 96

Signs and criteria for diagnosis of the disease

The main symptom of nasal liquorrhoea is the outflow of liquid from one or two nasal passages or its flow down the back wall of the pharynx.

Fluid leakage can be constant or remitting, depending on the amount of fluid - significant, moderate or insignificant.

Headache occurs in most patients and has a different character - against the background of fluid leakage, it indicates hypotension, during remission, it indicates hypertensive fluid-dynamic disorders.

Nasal liquefaction occurs as a result of craniocerebral injury (traumatic), surgical intervention (iatrogenic), congenital anomalies of development, neoplasms of the base of the skull (symptomatic) and due to enlarged anatomical openings of the ethmoid bone due to liquefaction disorders (spontaneous).

Clinical forms of liquefaction are classified depending on the time of its occurrence, duration, course of the disease and associated complications, localization of the liquefaction fistula. At the same time, the main factors that decide the further tactics of treatment (non-surgical or surgical) are:

1. Time of occurrence: early liquefaction (up to 3 weeks), late liquefaction (after 3 weeks).

2. Duration of liquefaction: short-term traumatic (iatrogenic) liquefaction (up to 3 months), long-term traumatic iatrogenic liquefaction, short-term spontaneous liquefaction (up to 1 year), long-term spontaneous liquefaction (more than 1 year).

3. Presence of meningitis.

4. Localization of the CSF fistula: in the area of the frontal sinus, in the area of the ethmoid bone, in the area of the sphenoid sinus.

5. Presence of secondary hypertensive manifestations.

Conditions in which medical assistance should be provided

Patients with nasal discharge are subject to inpatient treatment in the neurosurgical department.

Diagnosis of nasal liquorrhoea

The presence of glucose (> 0.3 mg per ml) in the fluid that flows out reliably distinguishes it from nasal secretions (< 0.05 mg per ml) and indicates the presence of liquorrhoea.

List of diagnostic measures:

1. Collection of anamnestic data and complaints of the patient.

2. Examination of the ENT specialist.

3. Qualitative ("Glucotest" test) or quantitative determination of glucose in the fluid that flows out.

4. Instrumental research methods:

- inspection craniography - visualizes fractures of the vault and base of the skull;

- computer tomography (CT), magnetic resonance imaging (MRI) - used to determine the possible cause of liquefaction;

- computer-tomographic cisternography is the most informative method of determining the localization of a CSF fistula.

Treatment

Treatment tactics depend on the type of nasal liquorrhoea and the clinical form.

Treatment of symptomatic liquefaction primarily requires elimination of the cause that led to the occurrence of liquefaction with simultaneous plastic surgery of the CSF fistula if necessary.

In case of traumatic and iatrogenic licorrhoea, treatment tactics do not differ.

Non-surgical treatment of patients with nasal discharge

In cases of early short-term liquefaction, conservative (drug) therapy is used, aimed at reducing liquefaction production and CSF pressure (includes the use of a complex: strophantin 0.5 ml 2 times a day intravenously, diacarb 1 t. 2 times a day in combination with potassium preparations).

In case of ineffectiveness of conservative treatment, external long-term lumbar drainage is used, aimed at creating artificial CSF hypotension.

Surgical treatment of patients with nasal discharge

Indications for surgical intervention are established in the case of ineffectiveness of the above-mentioned treatment methods, the presence of tense pneumocephalus, porencephaly, a significant leakage of fluid with pronounced hypotensive manifestations, or if liquor continues. The ways of outflow of cerebrospinal fluid are important - ventricular or subarachnoid (diagnosed with Pusep's test). Ventricular liquefaction (with Pusep's test, fluid leakage increases) is also an indication for surgical intervention.

The method of surgical intervention is determined by the localization of the CSF fistula, the course of the disease. When the fistula is localized in the area of the frontal sinus, the use of the transcranial extradural method of intervention is indicated, in the area of the lattice labyrinth - the transcranial intradural method. Localization of the CSF fistula in the area of the sphenoid sinus requires the use of the endonasal method of intervention. In cases where during the long course of liquefaction, especially in patients with meningitis, secondary hypertensive liquefaction disorders were formed, the use of liquid shunting or a combined method of surgical treatment is justified (transcranial or endonasal intervention is supplemented with liquid shunting). In the case of relapses and minor liquefaction, when the location of the fistula is not determined, liquefaction shunting interventions may also be the method of choice.

Spontaneous liquefaction is better amenable to conservative treatment due to the mechanisms of its occurrence, therefore the use of drug treatment and long-term lumbar drainage is justified.

Due to the fact that the localization of fistulas with spontaneous liquefaction is limited by the lattice plate, the method of surgical intervention is transcranial intradural. In cases accompanied by hypertensive phenomena, the methods described above are used.

Operative interventions are performed under general anesthesia. Endonasal interventions require the presence of an X-ray operating room equipped with an EOP and an operating microscope.

Transcranial interventions are performed from a bifrontal approach. The intradural method is supplemented by ligation of the upper sagittal sinus in its anterior third. Obligatory revision of the front cranial fossa on both sides.

Lumbo-peritoneal modification is mainly used during liquid shunting operations. Previous long-term lumbar drainage is a test-control of the effectiveness of the future shunting intervention (negative reaction of the "Glucotest" sample against the background of functional drainage).

In the postoperative period, during transcranial and endonasal interventions, it is also necessary to use long-term drainage in order to prevent compensatory CSF hypertension.

Diakarb is prescribed for 1-3 months in the postoperative period in order to reduce fluid production according to the scheme (1 t. 2 times a day at 9 am and 2 pm - 5 days with the addition

of potassium preparations, with a break of two weeks).

Efficacy criteria and expected results of treatment

The average duration of treatment in a neurosurgical hospital is up to 30 days.

The criteria for the quality of treatment are the improvement of the general condition of the patient and the absence of liquefaction upon discharge from the hospital.

Outpatient supervision of patients is carried out for 3 years with periodic examination by an otolaryngologist, control sample "Glucotest", since the largest number of relapses occurs in the first 3 months, and then in the period between 1-3 years.

Protocol
providing medical care to patients with traumatic epidural hematomas
IKH-10 code: S06.4

Traumatic epidural hematoma (TEH) is a traumatic accumulation of blood between the inner surface of the bones of the skull and the dura mater, which causes compression of the brain.

The source of bleeding in the formation of epidural hematomas is the meningeal arteries damaged as a result of a head injury, less often the meningeal veins, dural sinuses and diploe vessels. TEG in most cases are formed at the place of application of traumatic force, unilateral.

Signs and criteria for diagnosis of the disease

The clinical picture of TEG depends on the source of bleeding, the localization and size of the hemorrhage, the rate of development of brain compression, the severity of concomitant damage to the skull and brain, the age and individual characteristics of the patient.

The state of consciousness in patients with TEG can range from clear consciousness to coma (from 15 to 3 points on the SHKG). For TEG, the most typical is a three-phase change in consciousness: loss of consciousness during the injury, then recovery of consciousness, the so-called "light interval", and after some time, its re-exclusion. The duration of the light interval can be from a few minutes to three or more days.

Headache with TEG is constant with periodic crisis-like exacerbation, in many cases it has a membranous character. It is often accompanied by nausea and vomiting.

Bradycardia is observed in half of patients with TEG. An increase in arterial pressure is observed in ¼ of patients with TEG.

The vast majority of patients with TEG have focal symptoms that depend on the localization of TEG. Focal symptoms may have the character of shedding or irritation.

Among the craniobasal symptoms observed in TEG, the most important is the dilation of one pupil with a decrease or loss of response to light. In the vast majority of cases, unilateral mydriasis is observed on the side of the TEG, but in 11-15% of cases, mydriasis is contralateral.

A triad of symptoms is typical for TEG - light gap, homolateral mydriasis, contralateral hemiparesis. Another triad of symptoms is also used - light interval, mydriasis and bradycardia.

However, there are no pathognomonic clinical tests and symptoms to recognize TEG.

With TEG, fractures of the bones of the skull on radiographs are found in 75-90% of observations. The presence of a fracture of the skull, especially of the temporal bone, hemiparesis contralateral to it, homolateral mydriasis with high probability indicate the presence of TEG.

The phasic nature of the clinical course in TEG usually ends with a progressive deterioration of the patient's condition with an increase in general cerebral focal dislocation symptoms and subsequent vital disturbances.

The algorithm for providing medical care to a patient with TBI in the acute period is given in the appendix.

Conditions in which medical assistance should be provided

Patients with a head injury and suspicion of TEG should be immediately hospitalized for examination and treatment in the neurosurgery department.

Diagnostics

Diagnostic measures include (in the first 3 hours from the time of admission to the reception department):

1. General somatic examination with determination of indicators of the main vital functions (breathing, pulse, blood pressure).

2. Neurological examination.

3. CT scan (MRI) of the brain in the first 60 minutes (the main method of TEG diagnosis).

4. X-ray of the skull in 2 projections.

5. EchoES (in the absence of CT).

6. Determination of blood group and Rh factor.

7. General analysis of blood and urine.

8. Blood analysis for sugar and alcohol content. Rappoport's test.

9. Biochemical study of blood (electrolytes, total protein), blood plasma osmolality and hematocrit. Blood coagulation control.

EchoES is of particular importance in the absence of CT and MRI. For unilateral TEG of a typical localization, the displacement of the median structures in the opposite direction is characteristic.

CT makes it possible to visualize TEG of any localization by direct and indirect signs. CT makes it possible to assess the degree of compression and displacement of brain structures, including in the case of iso-dense hematomas, to conduct observations in dynamics, and detects concomitant TEG damage to the brain.

The diagnostic capabilities of MRI are superior to CT when detecting isodense TEGs. MRI makes it possible to examine structural changes in the brain stem in detail.

If it is not possible to perform a CT scan (MRI), and according to the clinical examination, supplemented, if available, by EchoES, there is a possibility of TEG, a diagnostic operation is indicated - the imposition of milling holes in the temporal, parietal and frontal areas - necessarily on both sides, starting from the mydriasis side.

Treatment

The scope and sequence of medical care depends on the patient's condition, clinical phase, clinical form and size of TEG.

In the phase of clinical decompensation, treatment begins from the time the patient is admitted to the receiving department according to intensive therapy algorithms.

The main method of treatment of patients with TEG is surgical removal of the hematoma.

Indications for surgical removal of TEG:

1. Clinical signs of compression of the brain according to at least one of the criteria: focal, general brain, dislocation.

2. Repeated disturbance or deterioration of consciousness in the presence of a light interval.

3. TEG volume (according to CT, MRI) >50 ml for supratentorial and >20 ml for subtentorial or thickness more than 1.5 cm regardless of clinical phase, including asymptomatic TEG.

4. The presence of at least one CT (MRI) sign: lateral displacement of midline structures >5 mm, deformation of the basal cisterns, gross compression of the homolateral lateral ventricle with dislocation contralateral hydrocephalus, regardless of the size and location of the TEG.

5. TEG of the posterior cranial fossa of small volume (<20 ml), if they lead to occlusive hydrocephalus.

The presence of at least one of the above criteria is an indication for urgent intervention. Diagnosis, determination of indications for surgical removal of TEG and referral of the patient to the operating room should be carried out within the first 3 hours from the moment of hospitalization.

Bone-plastic trepanation of the skull is optimal. If there are large bone fragments in the area of trepanation, they are then fastened together. If this is not possible, they are removed (resection trepanation). Bleeding is stopped. Revision of the subdural space is carried out based on

the presence of CT (MRI) signs of other factors of brain compression. With increased bleeding of soft tissues, the dura mater is sewn along the edges of the trepanation hole and behind the central part through the holes in the bone flap to prevent recurrence of TEG.

Treatment in the postoperative period includes, depending on the patient's condition, measures of intensive therapy (according to indications). Dynamic (clinical, laboratory, X-ray, etc.) monitoring of the general somatic and neurological status, CT (MRI)-control (for 1-3 days after surgery or when the patient's condition worsens) is carried out.

Contraindications to operative treatment:

1. Atonic coma with gross vital disturbances.

2. Verified brain death according to the criteria defined by the regulatory documents of the Ministry of Health of Ukraine.

3. Critical disorders of the blood coagulation system (thrombocytopenia - the number of platelets is 50·103 μl and below, the concentration of fibrinogen in the blood is less than 0.5 g/l).

Non-surgical treatment of patients with TEG

A mandatory prerequisite is hospitalization in a neurosurgical department, where a neurosurgeon is on call 24 hours a day, conditions for CT (MRI) are available 24 hours a day, and neurosurgical intervention is available at any time.

Indications for non-surgical treatment in TEG:

Stable, relatively satisfactory condition of the patient (SHKG 15-13 points) in the absence or minimal, non-increasing focal and general cerebral symptoms (phases of clinical compensation and subcompensation), in the absence of clinical signs of brain dislocation (permissible displacement of the middle structures of the brain up to 5 mm according to CT scan, MRI without signs of dislocation hydrocephalus, deformation of the basal cisterns).

Dynamic (clinical, laboratory, X-ray, etc.) monitoring of the general somatic and neurological status is carried out, CT control is carried out for 3-14 days and before discharge from the hospital or when the patient's condition worsens, when the displacement of the median echo signal increases. Echocardiography with non-surgical treatment is performed daily. Medical treatment includes hemostatic (1-3 days), dehydration, anti-inflammatory, analgesic and symptomatic therapy, which promotes hematoma resorption.

The criteria of effectiveness and expected results of treatment are regression of compression-dislocation signs according to CT (MRI), improvement of the patient's general condition, partial regression of general brain and focal neurological symptoms.

Approximate duration of treatment in inpatient conditions - up to 15 days.

When the patient's condition is stabilized, continuation of treatment in the rehabilitation or neurology department is indicated.

Protocol
providing medical care to patients with traumatic subdural hematomas
IKH-10 code: S06.5

Traumatic subdural hematoma (TSH) is a trauma-induced accumulation of blood between the inner surface of the dura mater and the arachnoid membrane on the outer surface of the brain, which leads to compression of the brain.

TSH is the most common type of intracranial hematoma. Isolated TSH accounts for 40-60% of cases of brain compression by hemorrhages.

Unlike epidural hematomas, TSGs occur both on the side of the application of the traumatic force and on the opposite side. In 10-15% of cases, bilateral TSGs are observed.

The source of bleeding during the formation of TSH is the veins damaged as a result of a head injury, which flow into the sinuses of the brain, damaged surface vessels of the hemispheres (veins and arteries), damage to the venous sinuses.

Signs and criteria for diagnosis of the disease

The clinical picture of TSH depends on many factors - the source of bleeding, the location

and size of the hemorrhage, the rate of development of brain compression, the severity of concomitant damage to the skull and brain, as well as the age and individual characteristics of the patient. Acute, subacute and chronic clinical courses are characteristic of TSH.

The state of consciousness in patients with TSH can range from clear consciousness to coma (from 15 to 3 points on the SHKG). For acute TSH, the variant without a light gap is most typical due to the frequency of concomitant brain damage. For subacute TSH, a three-phase change in consciousness is most typical: loss of consciousness at the time of injury, then recovery of consciousness, and after some time, re-exclusion. Quite often there are cases with an erased light gap.

Focal symptoms in TSG are less pronounced than in epidural hematomas and more scattered. Symptoms of shedding and irritation are observed.

Disturbances of consciousness, ipsilateral mydriasis, and contralateral hemiparesis are typical.

There are no pathognomonic clinical tests and symptoms to recognize TSH.

Echo-EC is of particular importance for the diagnosis of unilateral TSGs in the absence of CT or MRI. Shifting of the middle structures in the opposite direction is typical for such TSGs.

CT makes it possible to detect TSG of any localization by direct and indirect signs and to assess the degree of compression and displacement of brain structures, including in the case of iso-dense hematomas, to carry out dynamic monitoring, reveals concomitant TSG damage to the brain.

MRI is more informative than CT when detecting subacute and chronic, isodense TSH, makes it possible to differentiate TSH from epidural hematoma, as well as to examine the condition of the brain stem and basal cisterns in detail.

In a situation where CT scan (MRI) is not possible, and according to the clinical examination it is impossible to exclude TSG, a diagnostic operation is indicated - the imposition of milling holes in the temporal, parietal and frontal areas, necessarily on both sides.

The algorithm for providing medical care to a patient with TBI in the acute period is given in the appendix.

Conditions in which medical assistance should be provided

Patients with TSH are subject to inpatient treatment in the neurosurgical (if not available, in the traumatology) department.

Diagnostics

Patients with a head injury and suspicion of TSG should be immediately sent for examination and treatment to the neurosurgery department.

Examination of the patient should include (in the first 3 hours from the time of admission to the reception department):

1. General somatic examination with the definition of the main vital functions (breathing, pulse, blood pressure).
2. Neurological examination.
3. CT scan of the brain in the first 60 minutes (the main method of diagnosing TSH).
4. X-ray of the skull in 2 projections.
5. EchoES (in the absence of CT).
6. Determination of blood group and Rh factor.
7. General analysis of blood and urine.
8. Blood analysis for sugar and alcohol content. Rappoport's test.
9. Biochemical study of blood (electrolytes, total protein), blood plasma osmolality and hematocrit. Blood coagulation control.

Treatment

The scope and sequence of medical care depends on the clinical phase, clinical form and size of TSG.

In the decompensation phase (typical for acute TSH with brain lesions), treatment begins from the time the patient is admitted to the receiving department according to intensive care algorithms.

The main method of treatment of patients with TSH is surgical removal of the hematoma.

Indications for surgical removal of TSG:

1. Clinical signs of compression of the brain according to at least one of the criteria: focal, general brain, dislocation.

2. Repeated disturbance or deterioration of consciousness in the presence of a light interval.

3. The presence of at least one CT (MRI) sign: displacement of median structures >5 mm, deformation of the basal cisterns, gross compression of the homolateral lateral ventricle with dislocation contralateral hydrocephalus, regardless of the size and localization of the TSH.

The presence of one of the above criteria requires urgent surgical intervention. Diagnosis, determination of indications for surgical removal of TSG and referral of the patient to the operating room should be carried out within the first 3 hours from the time of hospitalization.

Acute TSGs have a fairly wide spread over the cerebral hemisphere, often combine with other types of intracranial hemorrhages, therefore preference is given to wide bone-plastic trepanation of the skull, which makes it possible to completely remove TSGs, find the source of bleeding and, if necessary, complete the operation with "external" decompression with removal of a bone flap. After dissection of the dura mater (TMO), the hematoma is removed (blood clots are washed off the surface of the brain with saline, and then removed with an aspirator or window tweezers). Hemostasis is carried out. Drainage tubes are installed subdurally, which are removed through counter-apertures. With cerebral edema, the TMO is not sutured or is hermetically sutured due to plastic surgery with additional tissues (TMO layering, fascia of the temporal muscle, superficial fascia of the thigh, artificial TMO). In the presence of cerebral edema, the bone flap is removed. Soft fabrics are sewn up in layers. During the day after the operation, washing of the subdural space is carried out through closed circuit drains. The drains are removed after a day.

Alternative methods of surgical intervention in subacute TSH are endoscopic removal of TSH, as well as removal of TSH through two expanded milling holes with active drainage of the hematoma cavity for 2-3 days.

Treatment in the postoperative period includes, depending on the patient's condition, measures of intensive therapy (according to indications). Conduct dynamic (clinical, laboratory, X-ray, etc.) observation of the general somatic and neurological status, CT control (for 1-3 days after surgery or when the patient's condition worsens). In the absence of a CT scan, an EchoEG is performed in dynamics.

Contraindications to operative treatment: 1) atonic coma with gross vital disturbances; 2) verified brain death according to the criteria defined by the regulatory documents of the Ministry of Health of Ukraine; 3) critical disorders of the blood coagulation system (thrombocytopenia - the number of platelets is 50·103 μl and below).

Non-surgical treatment of patients with TSG

A mandatory prerequisite for non-surgical treatment of patients with TSG is hospitalization in a neurosurgical department, where a neurosurgeon is on call 24 hours a day, conditions for CT (MRI) round the clock, and opportunities for neurosurgical intervention at any time are provided.

Indications for non-surgical treatment of TSH:

Stable, relatively satisfactory condition of the patient (SHKG 15-13 points) in the absence or with minimal, non-increasing focal and general brain symptoms, in the absence of clinical signs of brain dislocation (permissible displacement of the middle structures of the brain according to CT, MRI data up to 5 mm without signs of dislocation hydrocephalus , without deformation of the basal cisterns).

When choosing a non-surgical method of treatment of TSH, dynamic (clinical, laboratory, X-ray, etc.) observation of the general somatic and neurological status is carried out, CT control is performed for 3-14 days and before discharge from the hospital or when the patient's condition worsens, when the median echo displacement increases. EchoEG is performed daily. Medicinal treatment of TSH in such cases includes hemostatic (1-3 days), dehydration, anti-inflammatory, analgesic, resorptive and symptomatic therapy (according to indications).

The effectiveness criteria and expected results of treatment of patients with TSH are

improvement of the patient's general condition, regression of neurological symptoms and compression-dislocation symptoms according to CT (MRI) data.

Approximate duration of treatment in inpatient conditions - up to 15 days.

When the patient's condition is stabilized, continuation of treatment in the rehabilitation or neurology department is indicated.

Protocol
treatment of patients with chronic subdural hematomas Code IX-10: T 90.5

Chronic subdural hematomas (CSH) are hemorrhages separated by a capsule between the dura mater and the arachnoid membranes, which cause compression of the brain and are clinically manifested several weeks after the injury.

Signs and criteria for diagnosis of the disease

The clinical picture of HCG depends on lateralization (left-sided, right-sided, bilateral), localization (supratentorial, subtentorial), volume (small - up to 50 cm3, medium - from 50 to 100 cm3, large - more than 100 cm3), age, individual characteristics the patient and the phases of the course of hCG:

1. Phase of clinical compensation (manifested by the clinic of astheno-neurotic syndrome, episodes of cephalgia).

2. Phase of clinical subcompensation (disorders of consciousness to the level of mild stupor, mental disorders that may be the main symptom in the clinic, focal symptoms in the form of mild hemiparesis, aphatic disorders).

3. The phase of moderate clinical decompensation (the general condition is of medium severity, consciousness is disturbed to the point of deep stupor, mental disturbances, focal symptoms, individual dislocation trunk signs at the tentorial level).

4. Phase of gross clinical decompensation (the general condition is severe, consciousness is impaired to the degree of sopor - coma I-II, gross subcortical and trunk symptoms with vital disturbances).

5. Terminal phase (consciousness impaired to the level of terminal coma with gross violations of vital functions).

According to the clinical development, it is divided into pseudotumorous, stroke-like and remitting course of HCG.

The algorithm for providing medical care to a patient with TBI in the acute period is given in the appendix.

Conditions in which medical assistance should be provided

Patients with HCG are subject to inpatient treatment in the neurosurgical department.

Diagnostics

Diagnostic measures include:

1. Clarification of the presence of a head injury in the anamnesis (often without loss of consciousness).

2. Evaluation of complaints and neurological symptoms.

3. Instrumental examination methods:

a) X-ray:

- inspection craniography (hypertensive signs may be observed, symptoms of volume influence - displacement to the opposite side of the calcified pineal gland).

b) neuroimaging:

- computer tomography (CT) - direct (hypo- and heterodense hematomas) and indirect (dislocation) signs (isodense hematomas);

- magnetic resonance imaging (MRI) - (the leading diagnostic method for chronic hematomas of all types of density - the "golden" diagnostic standard);

cerebral angiography (vascular zone, dislocations of main vessels).

B) ultrasound examination (in the absence of CT, MRI):

- echoencephalography – displacement of M-echo by 5 mm or more. With bilateral hematomas, M-echo displacement may not be present.

Treatment

Absolute indications for surgical treatment are compressive-dislocation changes according to CT or MRI data.

Relative indications for surgical treatment are a small volume of hematoma with clinical signs of mental disorders without dislocation changes according to CT or MRI.

Only decompensated somatic pathology can be a contraindication to surgery. In the decompensation phase, treatment is carried out from the time of admission of patients to the reception department according to the algorithms of intensive therapy.

Before surgery, a complex examination of the somatic status of patients is carried out with the involvement of a therapist. The possible risk of surgical intervention and anesthetic support is assessed.

Methods of surgical intervention

1. Mini-invasive – evacuation of HCG through a milling hole (holes), drainage of the hematoma cavity; "Twist-drill" craniotomy; endoscopic evacuation of the hematoma.

2. Bone-plastic craniotomy.

The minimally invasive method of surgical intervention is indicated for:

1) single-chamber, rarely two-chamber hematoma structure;

2) hematoma in the form of a liquid or in the form of a liquid-clot.

Bone-plastic craniotomy is indicated for:

1) multi-chamber structure of a hematoma with many membranes occupying a significant part of the volume of the hematoma;

2) hematoma in the form of a dense convolution or with its calcification;

3) relapses of hematoma.

Interventions are performed under general anesthesia, bone-plastic trepanation requires the use of mechanical ventilation.

Possible postoperative complications:

1. Recurrence of hematoma.

2. Formation of acute subdural, epidural and intracerebral hemorrhages.

3. Pneumocephalus.

4. Purulent-inflammatory complications.

5. Thromboembolism.

In the postoperative period, antibiotics (5-7 days), painkillers, nootropics, vascular drugs are prescribed, thromboembolism prevention is carried out.

Non-surgical treatment of patients with HSG

Non-surgical treatment of patients with HCG is allowed if the HCG is small in volume (up to 50 cm3), there is no neurological deficit, compression-dislocation changes, and the possibility of periodic CT or MRI control. Non-surgical treatment includes moderate dehydration, nootropic, vascular, anticonvulsant drugs (as indicated).

The efficiency criteria and expected results of treatment of patients with HCG are improvement of the general condition of the patient with partial regression of general brain and focal symptoms, regression of compression-dislocation changes (according to CT or MRI data) from 3 weeks to 6 months after surgery.

The estimated duration of treatment in the neurosurgical department is up to 15 days.

Further inpatient treatment is indicated in the rehabilitation or neurology department

Appendix to the order of the Ministry of Health No. 317 dated 06-13-2008

Topic, document description: Clinical protocol

Type of care: outpatient, inpatient, target group: not specified

Field of medicine: Neurosurgery

Clinical condition, pathologies: Trauma of the cervical spine and spinal cord

Clinical protocol for providing medical care to patients with injuries of the cervical spine and spinal cord

Code according to IKH-10: S14

Signs and diagnostic criteria

Signs of an injury to the cervical spine and spinal cord are local soreness and deformation, a decrease or absence of sensitivity below the level of damage, movement disorders in the upper and lower limbs (in case of cervical region injury), in the lower limbs (in case of thoracic and lumbar region injury), impaired function pelvic organs.

The clinical picture of damage to the cervical spine and spinal cord depends on the localization, type of damage, rate of development of compression of the spinal cord, age and individual characteristics of the patient.

According to the etiological factor, industrial, road and transport, sports, domestic, and other types of spinal injuries are distinguished.

The following spinal fractures with damage to the spinal cord (CM) and (or) its roots are distinguished:

A. 1. Direct (caused by the direct impact of mechanical force, falling from a height on the legs or head). 2. Indirect (with excessive bending or extension of the spine).

B. 1. Open injuries of the spine and SM (with violation of the integrity of the skin). 2. Closed (without violating the integrity of the skin). 3. Open penetrating injuries (in case of trauma to the dura mater)

B. 1. Complicated. 2. Uncomplicated

According to the stability of the spinal cord injury: stable (most often occur with wedge-shaped compression fractures of the bodies and with fractures of the brackets proximal to the 4th lumbar vertebra, as well as with fractures of the transverse and spinous processes) unstable (all displacements (dislocations) of the vertebrae, fractures and dislocations of the articular processes, tears intervertebral discs and their connection with damage to the vertebral bodies. All patients with spinal instability require medical stabilization with the help of corsets, splints, ties, surgical intervention).

Clinical forms of traumatic lesions of the spinal cord:

Concussion of the spinal cord (synonym - spinal shock). It is possible to establish a concussion of the spinal cord in the presence of pronounced neurological symptoms after the injury, followed by its complete regression.

Spinal cord injury. Spinal cord injury can be diagnosed in the case of persistent neurological manifestations that do not regress over time or regress partially.

Hematomyelia.

Compression of the spinal cord by bony structures.

Crushing with partial violation of anatomical integrity or with interruption of the spinal cord.

Tension of the spinal cord with displaced vertebrae. This type of damage should be diagnosed with kyphotic spinal deformities, vertebral fracture dislocations with displacement.

If there is a concomitant fracture of the arches during fracture dislocations, spontaneous decompression of the spinal cord occurs and isolated tension of the spinal cord occurs on the displaced vertebrae. In the absence of a fracture of the arches, there is a combination of tension of the spinal cord and compression of its bone structures.

Epidural, subdural and subarachnoid hemorrhage.

Traumatic sciatica.

Acute period (2-3 days) - clinical manifestations of spinal cord injury of varying degrees of severity may be similar due to the fact that the clinical picture is caused by spinal shock (syndrome of complete disruption of spinal cord conduction, which is caused by spinal shock, disorders of blood and fluid dynamics, edema and swelling of the spinal cord).

Early period (from 4 days to 2-3 weeks) – with spinal cord injuries of varying degrees of

severity, a syndrome of complete disruption of spinal cord conduction may be observed, which is caused by spinal shock, hemodynamic and fluid dynamics disorders, swelling and swelling of the spinal cord.

Intermediate period (up to 2-3 months). At the beginning of this period (5-6 weeks after the injury), the phenomena of spinal shock, swelling of the spinal cord disappear, and the actual nature and extent of spinal cord damage is revealed - spinal cord injury, partial or complete impairment of neurological functions below the level of damage.

The late period (from 3-4 months to 2-3 years after the injury) - restoration of spinal cord functions, expressed to varying degrees depending on the severity of its damage (restoration of spinal cord functions can occur 5-10 years after the injury). The development of remote aggravation of neurological symptoms is possible, which is due to the development of the scarring process, cyst formation, the development of post-traumatic syringomyelia, the progression of kyphotic deformation of the spine, phenomena of instability with late compression of the spinal cord. For a unified assessment of neurological disorders, the Frankel, ASIA scale is used.

Conditions under which medical assistance should be provided

Patients with injuries of the cervical spine and spinal cord should be immediately sent for examination and treatment to the neurosurgical department in a fixed rigid cell.

Diagnostics

Examination of the patient should include (in the first 3 hours from the time of admission to the admission department of the hospital):

1. General somatic examination with determination of the main vital functions (breathing, pulse, blood pressure).
2. Neurological examination.
3. Ro-graph of the cervical spine in 2 projections (if fractures of the transverse processes are suspected - 1/2 and 3/4 projections on both sides).
4. Lumbar puncture, ascending or descending myelography (in the absence of CT, MRI).
5. CT of the cervical spine in the first 60 minutes after hospitalization (the main method of diagnosing spinal cord damage), MRI of the cervical spine (the main method of diagnosing spinal cord damage).
6. Determination of blood group and Rh factor.
7. General analysis of blood and urine.
8. Blood analysis for sugar and alcohol content. Rappoport's test.

Biochemical study of blood (electrolytes, total protein), indicators of blood plasma osmolality and hematocrit. Control of blood coagulation (from the 3rd day).

Treatment of injuries of the cervical spine and spinal cord

Immobilization of the head and neck (with the help of Schantz collars, Philadelphia collar, CITO splints, other types of collars, two sandbags, on a hard stretcher, head movements and sitting are prohibited) is carried out in all cases and is maintained until the diagnosis of HSMT is completely removed (x-rays are performed in two projections). Treatment of patients with HSMT begins at the pre-hospital stage, assistance includes preservation or normalization of vital functions (breathing, hemodynamics), fixation of the spine, preferably introduction of neuroprotectors (methylprednisolone). Patients with HSMT are recommended to be transported directly to specialized neurosurgical departments or trauma departments of the Central Hospital.

Providing assistance to patients in the acute period of the disease involves, first of all, normalization of breathing and hemodynamics, catheterization of the urinary bladder and central vein. In the case of spinal shock, the lower limbs are bandaged, atropine, hypertonic (3-7%) NaCI solution are administered, symptomatic treatment is carried out (according to the ABC algorithm (airway, breath, circulation), the victim is immediately hospitalized in the neurosurgery department.

In the first 8 hours prescribe methylpred (solumedrol) at a dose of 30 mg/kg/body weight

once, after 6 hours. the patient takes 15 mg/kg of the drug, in the future - 5 mg/kg every 4 hours. within 2 days Administer vitamin E 5 ml intramuscularly, difenin - 500 mg, broad-spectrum antibiotics, analgesics, neuroprotectors, magnesium sulfate, turn the patient over every 30-40 minutes, symptomatic treatment. After 8-12 weeks. the patient is transferred to rehabilitation in the neurological department, rehabilitation center, prevention of thromboembolic complications is carried out (bandaging of the lower extremities, massage, activation of movements, administration of heparin, fraxiparin (0.3 ml 2 times a day) for 7 days, then from the 7th day, the patient is transferred to Plavex (75 mg/day), Ticlid (1 tablet 2 times a day) or aspirin (0.325 times a day, during meals) for 2-3 months.

In the case of an injury to the cervical spine with vertebral dislocations without gross compression of the spinal cord, a skeletal extraction with a load corresponding to 10% of the victim's body weight or the application of a halo apparatus is indicated. If the general somatic condition of the patient allows, urgent surgical treatment is carried out within the first 6-48 hours after the injury. From the 3rd to the 12th day after the injury of the cervical spine, surgical intervention is associated with a high risk of ascending swelling of the spinal cord, high mortality.

Fistulas and places of leakage of cerebrospinal fluid must be sutured. Contraindications to the operation are shock, combined injuries that must be quickly eliminated.

Indications for surgical intervention: 1. Deformation of the vertebral-spinal canal, detected during Ro-graphy, CT or MRI examination data, which indicate compression of the spinal cord or narrowing of the spinal canal by 30% or more. 2. The presence of bone or soft tissue fragments in the spinal canal. 3. Partial or complete blockage of the CSF channels. 4. Progression of SM dysfunction. 5. Instability of the vertebral-motor segment, which creates a danger of increasing neurological symptoms.

Algorithm of indications for the selection of methods of treatment of spine and spinal cord injuries depending on the stability of the fracture and the presence of a neurological deficit:

I. Without neurological deficit:

A. Stable fracture - surgical intervention is not indicated. Immobilization with a craniothoracocervical bandage or Shantz collar is indicated.

B. Unstable fracture. Skeletal extraction or posterior open stabilization is indicated: a) vertebral dislocations - simultaneous closed reduction and external stabilization or skeletal extraction are necessary; b) in the case of impossibility of reduction - surgical intervention aimed at eliminating the deformation of the vertebral-spinal canal and stabilizing the spine (with a fragment of auto bone, posterior fixing transarticular plates with bicortical screws, titanium wire with frames, polyaxial transarticular fixing systems).

II. With partial neurological deficit:

A. Stable fracture. Shown is bone extraction, application of halo devices, external fixation; if it is impossible to eliminate compression of the spinal cord or its roots - surgery. Elimination of compression of the CM, its roots.

B. Vertebral dislocations. Closed reduction is recommended - bone traction, external stabilization; if closed reduction is impossible - open reduction and surgical stabilization (immovable disk prostheses - cages, fragments of autobone with front plates with bicortical screws, titanium wire with frames, polyaxial transarticular fixing systems, etc.).

B. Unstable fracture. Bone extraction and anterior or posterior stabilization are indicated (telescopic titanium prostheses of the vertebral bodies, mesh systems, ceramic prostheses, fragments of autobone with plates with bicortical screws, titanium wire with frames, polyaxial transarticular fixation systems).

III. With gross neurological deficit:

A. Stable fracture. In the absence of signs of compression of the spinal cord, bone traction and external stabilization are indicated.

B. Vertebral dislocations. Closed reduction (one-moment bone extraction) or surgical open reduction with stabilization (fixed disk prostheses - cages, autobone fragments with front plates

with bicortical screws, titanium wire with frames, polyaxial transarticular fixation systems, telescopic titanium vertebral body prostheses, mesh systems, ceramic prostheses).

B. Unstable fracture. If there are signs of spinal cord compression, bone extraction and early surgical intervention aimed at decompressing the spinal cord and stabilizing the spine are indicated.

Surgical approaches are determined by the nature of damage to the spine and spinal cord. In case of damage to the bodies, front, front-lateral accesses are used, in case of damage to the arches, articular processes, rear, back-side accesses are used, in the presence of hematomas (sub-, epidural and intramedullary), rear or combined accesses are used, depending on the localization of bone damage. The operation always ends with stabilization of the spine (internal and external).

Relative contraindications to anterior access surgery are associated dislocations or fracture dislocations, especially bilateral associated dislocation in the remote period. Contraindications are also isolated fractures of arches, articular processes and obvious posterior compression of the spinal cord. In these cases, rear access is necessary.

The criterion of the quality of treatment is the improvement of the patient's condition, the regression of neurological symptoms, the stability of the cervical spine according to control examinations: functional Ro-graphy and CT.

The average duration of treatment is 15-30 days.

After stabilization of the condition, transfer of the patient to the rehabilitation or neurology department is indicated.

Materials for self-control of training quality

Tests

1. Localization of functions in the cerebral cortex?
2. What are the membranes of the brain and spinal cord?
3. What is the composition of cerebrospinal fluid?

Answers:

1. Frontal fate: motor zone, motor center of Broca's speech, center of turning the head and eyes in the opposite direction, mental disorders. Occipital fate - sight. Parietal fate - sensitivity, centers of praxis and gnosis. Temporal fate - hearing, taste, smell, Wernicke's sensory center of speech.
2. Soft, spidery, hard.
3. Protein - 0.33‰, cytosis - 2-8 in cubic mm, sugar - 2.1 - 3.2 mg%, chlorides - 110 mg%.

1. Retrograde amnesia is:
a) amnesia for what happened in childhood
b) amnesia for current events
c) amnesia for the period before the injury
d) amnesia for professional skills
e) amnesia for the period after the injury Answer: c

2. The middle meningeal artery is located:
a) in the vertebra - basilar basin
b) in the system of arteries of the circle of Willis
c) in the duplication of the dura mater
d) in the area of the orbital fissure Answer: c

3. Where is the motor center of Broca's speech located?
a) in the upper temporal gyrus

b) in the front central gyrus
c) in the posterior central gyrus
d) in the lower frontal gyrus
e) in the middle temporal gyrus Answer: d

1. A 20-year-old patient fell during an ice storm. He does not remember the circumstances of the injury, complains of a headache, one-time vomiting. During the examination after a day - insufficient convergence from 2 sides, moderately pronounced vegetative stigmas.

Your diagnosis.
a) Head injury
b) Brain concussion
c) Mild cerebral contusion
d) Cerebral contusion of medium degree
e) Compression of the brain. *Answer: b*

2. As a result of the stab wound, the victim developed spastic paralysis of the right leg and anesthesia on the left below the navel.

What is the name of this syndrome?
a) Weber
b) Benedict
c) Brown - Sekara
d) Claude - Bernard - Horner
d) Wallenberg - Zakharchenko. *Answer: in*

2. Patient V., 18 years old. Complaints of headache, nausea, vomiting, decreased visual acuity. Complaints appeared after a TBI received a year ago.
On the fundus - phenomena of stagnation.
In the neurological status: ptosis, strabismus on the right. Hemiparesis, increase in muscle tone according to the pyramidal type on the right.
On the craniogram, there is an increase in finger depressions, a widened entrance to the Turkish saddle, deepening of the vascular grooves.

What is the most likely diagnosis?
a) Post-traumatic hydrocephalus
b) Post-traumatic encephalopathy
c) Post-traumatic parkinsonism
d) Post-traumatic hypothalamic syndrome.
e) Post-traumatic arachnoiditis. *Answer: d*

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H.

Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 21

Topic: Meningitis. Arachnoidites.

Purpose: to create an idea of the classification of meningitis, clinical forms of meningitis and arachnoiditis, pathomorphological changes in the meninges and brain substance in meningitis and arachnoiditis, general brain symptoms, general infectious symptoms, basic treatment schemes for patients with meningitis and arachnoiditis.

Basic concepts: Neuroinfections are the most common group of organic diseases of the nervous system. Their specific weight in the general pathology of the nervous system is 35-37%.

The action of the disease-causing factor extends, as a rule, to the entire nervous system as a whole. But depending on the conditions of general and local blood and lymph circulation, the aggressiveness of individual agents of neuroinfection, there is a predominant localization of the pathological process in the substance of the brain or in the meninges.

Meningitis is an inflammation of the membranes of the brain and spinal cord.

Inflammation of the soft and arachnoid membrane of the brain is called leptomeningitis, inflammation of the hard meninges is called pachymeningitis. In the clinic, the term "meningitis" usually means inflammation of the soft meninges. The causative agents of meningitis can be various pathogenic microorganisms, viruses, bacteria, protozoa. Therefore, the study of this topic is necessary for every doctor.

Arachnoiditis are chronic productive diseases of the arachnoid membrane of the brain with parallel involvement of the soft membrane, ependyma, subependymal layer of the ventricles, vascular plexuses. Occur as a complication of infectious and infectious-allergic diseases (60%), craniocerebral injuries (30%). A large polymorphism of neurological symptoms and a long, unbearable headache require correct medical measures that ensure the reduction of disability in the population.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Meningitis is a pathological process characterized by inflammation of the membranes of the brain.

The causative agents of meningitis can be bacteria (meningococcus, staphylococcus, pneumococcus, streptococcus, Escherichia coli, tuberculosis mycobacteria, etc.), viruses (herpes, measles, rubella, Coxsackie, ECHO, HIV, etc.), fungi (Candida genus, cryptococci, etc. .). Much less often, meningitis is caused by infection with protozoa or helminths.

The development of aseptic meningitis is possible, the cause of which can be neuroleukosis, carcinomatosis, sarcoidosis, connective tissue diseases or allergic reactions to the administration of vaccines, insect bites.

Pathogenesis.

Paths of penetration of the causative agent into the meninges can be different, but most often the entrance gate and the primary inflammatory site are localized in the nasopharynx. From the primary focus, the infection enters the brain membranes together with the bloodstream. In streptococcal and pneumococcal infections, the primary focus of inflammation is usually localized in the mucous membrane of the trachea and bronchi. The spread of infection with the bloodstream is also characteristic of the presence of chronic foci of infection in the body (otitis, bronchiectasis, abscess, sinusitis, furunculosis, cholecystitis, pneumonia, etc.).

With otitis, rhinitis, sinusitis, osteomyelitis of the bones of the skull, with inflammation of the orbit and eyeball, defects of the skin and soft tissues of the head, after lumbar punctures, with congenital malformations of the central nervous system, skin fistulas and sinuses, in addition to the spread of infection with the bloodstream, contact is possible the path of infection as a result of a violation of the integrity of the bones of the skull or the breakthrough of pus into the cavity of the skull. A rarer way of infection spreading to the membranes of the brain is spreading through the lymphatic vessels of the nasal cavity.

Meningitis affects people of all ages, but children are more often affected, which is due to the insufficient development of immunity and the imperfection of the blood-brain barrier (a mechanism that prevents the penetration of foreign substances into the central nervous system). Favorable factors play a significant role in the development of meningitis: skull injuries, vaccination, intrauterine pathology in children, various infectious diseases. From the primary

focus, the infection enters the bloodstream. Microorganisms, bypassing the liver, enter the central nervous system, where insemination occurs in the soft meninges of the brain and spinal cord. The inflammatory process usually affects the soft and arachnoid membranes, but it can spread to the dura mater, the roots of the spinal and cranial nerves, and to the superficial parts of the brain. The liquid is usually located in the furrows of the brain, but with a significant amount of it, it permeates the membranes completely and accumulates at the base of the brain. As a result of edema, the mass and volume of the brain increases, which can lead to wedging of the cerebellar tonsils into the large occipital foramen, compression of the medulla oblongata, and death of the patient from paralysis of the respiratory center. Subsequently, the inflammatory fluid is subjected to phagocytosis - the process of absorption by special cells - phagocytes. The process is often accompanied by a violation of the patency of the cerebrospinal fluid, which is complicated by the development of occlusive hydrocephalus. The effect of the causative agent and its toxins on the patient's body leads to a violation of the functions of many organs and systems with the development of acute adrenal, renal, cardiovascular and respiratory failure.

According to the nature of development, meningitis is divided into primary (inflammation of the meninges develops independently, without a previous general infection or local infectious damage to any organ) and secondary (inflammation of the meninges develops against the background of an already existing general or local infectious disease).

Bacterial, viral, fungal, parasitic, mixed, nonspecific meningitis are distinguished by origin; according to the nature of inflammation - purulent and serous; according to the localization of the pathological process - diffuse, convexity, basal and local; according to the nature of the course, acute (including fulminant), subacute, chronic and recurrent meningitis are distinguished.

Clinical picture.

Regardless of the causative factor, the clinical picture of meningitis has a number of common features: general infectious symptoms, meningeal syndrome and characteristic inflammatory changes in the cerebrospinal fluid.

Among the general infectious symptoms, intoxication takes the first place, which creates a background in the clinical picture of a serious disease and often leads to circulatory, water-salt and hormonal disorders. Against the background of significant intoxication, patients have pallor of the skin, blueness of the nasolabial triangle, shortness of breath with the involvement of auxiliary muscles. The pulse is frequent, and later relative bradycardia develops. Heart tones are muffled. Blood pressure during heavy running is low. Appetite is reduced, but patients drink a lot. Refusal to take fluids is a prognostically unfavorable sign. Against the background of intoxication, changes in the patient's urine appear: slight appearance of albumins, cylinders and other signs.

Meningeal syndrome is caused by irritation and inflammation of the meninges and is clinically manifested by a complex of general brain symptoms and meningeal symptoms. General brain symptoms include headache, dizziness, sound and photophobia.

Headache occurs as a result of irritation of the meninges, exposure to toxic substances, increased intracranial pressure. Headache is noted in all patients with meningitis. It is intense, distending, felt all over the head and may be accompanied by vomiting, intensifies when moving, against the background of strong sound and light stimuli. The use of analgesics is usually ineffective.

Vomiting occurs suddenly at the height of the headache, is not associated with eating, does not bring relief, differs in intensity ("fountain" vomiting). Most patients have a general increased sensitivity - visual, sound, tactile. Increased sensitivity develops as a result of irritation of the posterior roots, cells of the spinal cord nodes, receptors of the meninges, which lowers the threshold of sensitivity to various stimuli. Even a light touch to the patient causes increased pain. Patients usually lie with their back to the wall and cover their head with a blanket. Infants are restless, often scream, and are suddenly excited by any touch.

Seizures are often one of the first symptoms of meningitis, especially in young children. They tend to repeat themselves. The onset of the disease in adults and older children with convulsive twitching is considered an unfavorable sign.

Meningeal symptoms appear most often on the 2nd-3rd day after the onset of the disease, but can be noted from the first day: stiffness of the occipital muscles, symptoms of Kernig, Brudzinsky, Lesage, etc. Rigidity of the occipital muscles - limitation or impossibility of passive bending of the head. This symptom is the earliest and most persistent in meningitis. Kernig's symptom is the impossibility of extending the leg, previously bent in the knee and hip joints. Upper Brudzinsky symptom - when the head is passively brought to the chest in the position of the patient lying on his back, involuntary bending of the legs in the knee and hip joints occurs. Brudzinsky's symptom is average - when pressing on the area of the pubic joint, there is involuntary bending of the legs in the knee and hip joints. Lower Brudzinsky's symptom - when checking Kernig's symptom, there is an involuntary bending of the other leg in the same joints.

A separate group consists of symptoms based on increased pain sensations. Patients feel pain:

when pressing on the eyeballs through closed eyelids (Lobzyn's symptom);

when pressing on the front wall of the external auditory canal from the inside (Mendel's symptom);

when pressed at the exit points of various nerves;

when tapping on the zygomatic arch, which leads to a contraction of facial muscles (Bekhterev's symptom);

when tapping the skull (Pulatov's symptom).

In young children, the above-mentioned meningeal symptoms are often weakly expressed, therefore, during the examination, attention is paid to the bulging, tension and pulsation of the large fontanelle, the "hanging" symptom of Lesage (when holding the child in the armpit area, he pulls his legs to the abdomen and keeps them in this position), the child's characteristic pose - the head is thrown back, the legs are bent at the knees and pulled up to the stomach (the "lying dog" pose).

When the pathological process is localized on the basis of the brain, damage to the cranial nerves can be noted, which is clinically manifested by a decrease in vision, nystagmus, diplopia, ptosis, strabismus, hearing loss, facial muscle paresis, etc. When brain matter is involved in the process, symptoms of focal neurological symptoms in the form of paresis and paralysis appear in the clinical picture. In most cases, patients have a change in consciousness. In the first days from the onset of the disease, the processes of excitement predominate, which can increase and are accompanied by motor restlessness and hallucinations or change to lethargy up to the development of a comatose state.

Meningitis is characterized by changes in the cerebrospinal fluid. The presence of such changes in the cerebrospinal fluid makes it possible to diagnose meningitis regardless of the severity of meningeal symptoms. Inflammatory changes are characterized by cell-protein dissociation. With purulent meningitis, the cerebrospinal fluid is cloudy, the protein content increases, the pressure of the cerebrospinal fluid is increased, and the Pandey reaction is positive. With serous meningitis, the cerebrospinal fluid is transparent or slightly opalescent, lymphocytes predominate, the protein content is slightly increased, the cerebrospinal fluid flows out under increased pressure, there may be a positive Pandey reaction. The concentration of glucose in viral meningitis usually does not change, but in bacterial meningitis it depends on the origin of the disease and the severity of the pathological process in the meninges.

Meningococcal meningitis.

Meningococcal meningitis is typical of purulent meningitis. The causative agent of the disease is diplococcus, which is sensitive to environmental conditions and is transmitted by airborne droplets. This disease is characterized by winter-spring seasonality and low incidence rates. The epidemic process among the population takes place mainly in the form of bacterial carriage or acute nasopharyngitis. The onset of the disease is acute. Often, the patient or those around him can accurately indicate not only the day, but also the hour of the onset of the disease. The body temperature rises to 39-40 ° C, chills, a sharp headache, vomiting, symptoms of increased

sensitivity, meningeal symptoms appear. Symptoms of intoxication are pronounced. When examining patients, herpetic rashes on the skin, lips, mucous membranes of the oral cavity, and red dermographism are often detected. When meningococcal meningitis is combined with meningococcemia, in addition to the above symptoms, the patient develops a characteristic hemorrhagic rash.

In the general blood analysis, an increase in the level of leukocytes up to 15-30 x 10^9, eosinophils, a rod-nuclear shift up to 14-45%, an increase in ESR up to 45-70 mm/h are noted. Changes in the cerebrospinal fluid are typical for purulent meningitis, but on the first day of the disease, the cerebrospinal fluid may be clear, with some predominance of neutrophils. From the second day after the onset of the disease, the cerebrospinal fluid becomes cloudy, white or yellowish-green in color, the pressure is significantly increased (up to 300-500 mm H2O), the number of neutrophils is significantly increased, the amount of protein reaches 1-4.5 g/ l, the content of sugar and chlorides is reduced.

With timely treatment, meningococcal meningitis proceeds favorably and ends with recovery by the 8-12th day after the start of treatment. Lightning forms are dangerous, when death can occur within the first day from acute cardiovascular, respiratory or adrenal insufficiency.

Pneumococcal meningitis

Pneumococcal meningitis ranks second after meningococcal meningitis. The causative agents are pneumococci of various types. This form of meningitis usually affects young children. The development of the disease is preceded by the presence of a purulent focus: otitis, sinusitis, pneumonia. Acute respiratory diseases, other infections of the upper respiratory tract against the background of craniocerebral trauma also lead to the development of meningitis. Pneumococcal meningitis is severe and has a high mortality rate.

The body temperature rises to 40 ° C. From the first hours, symptoms of intoxication and meningeal symptoms are expressed, loss of consciousness quickly occurs, convulsions appear. At the end of 1-beginning of 2 days from the onset of the disease, patients have symptoms of damage to the cranial nerves, paresis and paralysis, a clinical picture of meningoencephalitis. The skin is pale, blueness of the limbs and shortness of breath are observed. The heart sounds are deaf, the pulse is arrhythmic, with weak filling and tension. Hepatolienal syndrome is noted, the development of toxic hepatitis is possible. Patients often die in the first days after the onset of the disease. The composition of cerebrospinal fluid is purulent: cloudy, yellowish-green in color, with a high protein content (up to 30-160 g/l), the level of neutrophils is high, the amount of sugar has been reduced since the first days of the disease.

Starting treatment later can lead to a protracted and relapsing course, since the causative agent, being in compacted areas of pus, is less accessible to the action of antibiotics. Recurrent cases of pneumococcal meningitis are accompanied by complications and persistent residual effects.

Meningitis caused by Haemophilus bacillus

The disease is caused by the Afanasyev-Pfeiffer bacillus, which often lives on the mucous membrane of the upper respiratory tract and under appropriate conditions can cause various diseases (rhinitis, otitis, pneumonia, sepsis, purulent meningitis). This form of meningitis most often affects weakened young children. The development of the disease is often preceded by pneumonia, rhinitis, otitis, sinusitis. Meningitis caused by Haemophilus bacillus begins gradually. The course of the disease is sluggish, wave-like. Periods of deterioration alternate with periods of improvement, which can occur even in the absence of appropriate treatment. In the acute course of the disease, already in the first hours of the onset of the disease, reduced pressure in the blood vessels of the brain (cerebral hypotension) can be observed. Intracranial pressure in such patients is sharply reduced (cerebral collapse). Cerebrospinal fluid during its puncture flows out in rare drops or it can be obtained only by suction with a syringe. The disease in this condition is very severe, with severe toxicosis and dehydration. Symptoms of cerebral hypotension develop

violently within several hours. The general condition of the patient worsens in the eyes. The patient is severely dehydrated, facial features are sharpened, the skin has a jaundiced color, the head of the head is inflamed, there are no meningeal symptoms, the muscle tone is reduced, tendon reflexes are not determined. The cause of the development of cerebral hypotension on the 3rd-4th day after the start of treatment can be the use of massive doses of benzylpenicillin and intensive dehydrating therapy.

The composition of cerebrospinal fluid is purulent: cloudy, milky-white or yellow-green in color, cytosis up to 400-600 in 1 μl, a large number of pathogens. With the development of cerebral hypotension, cerebrospinal fluid flows out in rare drops. The prognosis is favorable with timely treatment.

Staphylococcal meningitis

The development of staphylococcal meningitis is preceded by chronic pneumonia, abscesses, osteomyelitis of the bones of the skull and spine, sepsis. This form of meningitis has an unfavorable prognosis, as the percentage of deaths is high. Against the background of the main disease, the patient's body temperature suddenly rises, chills and meningeal symptoms appear, consciousness is disturbed up to the point of coma. Symptoms of focal damage to the nervous system often occur. A feature of staphylococcal meningitis is the propensity to abscess, which is due to the insensitivity of staphylococcus to antibiotics, the rapid development of resistance by the causative agent in the course of etiotropic therapy, and the difficulty of eliminating the primary focus of infection.

Meningitis

In progress	Lightning, acute, subacute, chronic
According to the degree of expression	Light, medium, heavy and extra heavy forms

According to the nature of the inflammatory process in the membranes and changes in the cerebrospinal fluid:

Purulent meningitis		Serous meningitis	
Primary	Secondary	Primary	Secondary
Meningococcal	Otogenic	Lymphocytic choriomeningitis	Tuberculous
	Odontogenic		Influenza
	Post-traumatic		Syphilitic

Clinical symptoms of meningitis

1. General infectious
   - Numbness
   - Increase in body temperature
   - Leukocytosis with a shift of the formula to the left
   - Tachycardia
   - Skin hyperemia
   - Increase of SSE.
2. Cerebral symptoms
   1. Headache
   2. Dizziness
   3. Nausea, vomiting
   4. Disturbance of consciousness
3. Meningeal
   1. stiffness of the muscles of the back of the head
   2. Brudzinsky's symptom (upper, middle, lower)
   3. meningeal pose
   4. Lesage symptom (in children)
   5. general hyperesthesia
   6. bradycardia
4. Changes in cerebrospinal fluid
   1. increase in CSF pressure
   2. violation of the transparency of the liquor
   3. change in the color of the liquor
   4. neutrophilic leukocytosis in purulent meningitis
   5. lymphocytic pleocytosis in serous meningitis
   6. change in the concentration of sugar and lipids in the cerebrospinal fluid
   7. manifestation of the causative agent

Meningococcal	Pneumococcal	Serous lymphocytic	Tuberculous
Exciter			
meningococcus	pneumococcus	ESNO and Coxsackie viruses	Tuberculosis bacillus Koch
Path of infection			
Air-drop and contact	hematogenously	enterovirus	hematogenously
Pathomorphology			

The inflammatory process in the membranes of the brain, incoma passes to the parenchyma of the brain and to the ependyma of the ventricles of the brain.	The inflammatory process in the membranes of the brain sometimes spreads to the brain parenchyma and cranial nerves.	Inflammatory process in the soft and arachnoid membranes	Inflammatory process in the soft meninges mainly on the basal surface of the brain with the formation of miliary nodules and the formation of serous-fibrous exudate.
Clinic			
The incubation period is 1-5 days. Pronounced general infectious and general brain symptoms. Meningeal symptoms increase by 2 - 3rd day of illness. It is possible to damage III and IV couples. On the 2-5th day, herpetic rashes appear on the lips, or hemorrhagic rash on the skin	It occurs as a complication of otitis, mastoiditis, sinusitis, pneumonia. General infectious and general brain symptoms increase gradually. Often comatose. When the triad is detected - pneumococcal meningitis, pneumonia, bacterial endocarditis - high mortality	The disease is benign, lasts 10-15 days. Characteristic epidemic outbreaks occur most often in the summer-autumn period. Severe general brain symptoms. Meningeal symptoms are moderately expressed. The face is hyperemic, the nasolabial folds are pale	The primary focus in the bronchial nodes, less often in the lungs and other organs begins slowly. In the first 2-3 weeks, moderate general brain symptoms appear. Then – meningeal damage to the oculomotor nerves. Seizures appear,

Indicators of cerebrospinal fluid in normal conditions and with meningitis of various etiologies

stand up	Liquor pressure, mm Hg.	Cytosis in 1 mm ^{3}	Protein content, g/l	Glucose content
Norm	100-200	Less than 5 lymphocytes	0.2-0.45	From 60% to 70% of the glucose content in the blood
Bacterial meningitis	Increased	100-60000, mostly neutrophils	0.5-5	Less than 40% of blood glucose
Tuberculous meningitis	Increased	10-500, lymphocytes	0.5-5	Much less than 40% of blood glucose
Fungal meningitis	Increased	25-500, mainly lymphocytes	0.5-5	Less than 70% of blood glucose
Viral meningitis	Normal or elevated	An increase in the number of lymphocytes	0.5-2	Norm

Treatment of meningitis

In case of suspicion of meningitis, hospitalization of the patient is mandatory. With a severe course of the pre-hospital stage (depression of consciousness, fever), the patient is administered 50 mg of prednisone and 3 million units of penicillin. Performing a lumbar puncture at the pre-hospital stage is contraindicated!

The basis of purulent meningitis treatment is the early appointment of sulfonamides (etazol, norsulfazole) in an average daily dose of 5-6 g or antibiotics (penicillin) in an average daily dose of 12-24 million units. Allows intralumbar administration of benzylpenicillin (in extremely severe cases). If such treatment of meningitis during the first 3 days is ineffective, therapy with semi-synthetic antibiotics (ampiox, carbenicillin) in combination with monomycin, gentamicin, nitrofuran should be continued. The effectiveness of such a combination of antibiotics in the isolation of a pathogenic organism and the detection of its sensitivity to antibiotics has been proven. The maximum duration of such combination therapy is 2 weeks, after which it is necessary to switch to monotherapy. The criteria for cancellation are also a decrease in body temperature, normalization of cytosis (up to 100 cells), regression of brain and meningeal symptoms.

The basis of the complex treatment of tuberculous meningitis is the continuous administration of bacteriostatic doses of two or three antibiotics (tubazid 0.6 g/day + streptomycin 1 g/day). In the event of possible side effects (vestibular disorders, hearing impairment, nausea), there is no need to cancel this treatment, it is indicated to reduce the dose of antibiotics and temporarily add to the treatment desensitizing drugs (diphenhydramine, pipolfen), as well as a second antituberculosis drug (rifampicin, PASK, ftivazid). Indications for discharge of the patient: absence of symptoms of tuberculous meningitis, sanation of cerebrospinal fluid (after 6 months from the onset of the disease) and improvement of the patient's general condition.

Treatment of viral meningitis can be limited to the use of symptomatic and tonic agents

(glucose, analgin, vitamins, methyluracil). In severe cases (pronounced general brain symptoms), corticosteroids and diuretics are prescribed, less often – repeated spinal puncture. Antibiotics may be prescribed in case of bacterial infection.

Prognosis in meningitis

In the future, the form of meningitis, the timeliness and adequacy of treatment measures play an important role in the prognosis. Headaches, cerebrospinal fluid hypertension, epileptic seizures, vision and hearing disorders often remain as residual symptoms after tuberculous and purulent meningitis. Due to late diagnosis and resistance of the causative agent to antibiotics, the mortality rate from purulent meningitis (meningococcal infection) is high.

Prevention of meningitis

As preventive measures to prevent meningitis, regular hardening (water procedures, sports), timely therapy of chronic and acute infectious diseases, as well as short courses of immunostimulating drugs (Eleutherococcus, ginseng) in foci of meningococcal meningitis (kindergarten, school, etc.)

Excerpt from the order of the Ministry of Health of Ukraine dated 07/09/2004 No. 354

3. PROTOCOL

DIAGNOSIS AND TREATMENT OF PURULOUS MENINGITIS IN CHILDREN

CODE MKH-10 - GOO-G09

Purulent meningitis is a group of diseases of the central nervous system of bacterial etiology, the basis of which is purulent inflammation of the membranes of the brain.

All meningitis is classified

by etiology: meningococcal, pneumococcal, staphylococcal, hemophilic, streptococcal, enterococcal, salmonellosis, etc.;

by pathogenesis: primary and secondary;

by severity: moderate, severe;

according to the course: acute (up to 3 months), prolonged or subacute (over 3 months); with and without complications.

DIAGNOSTIC CRITERIA:

CLINICAL:

- general infectious syndrome;
- meningeal syndrome.

Meningeal syndrome includes:

- hydrocephalus-hypertensive symptoms: sharp diffuse headache, repeated vomiting, general hyperesthesia;
- meningeal symptoms: meningeal posture, tonic tension of the back muscles, stiffness of the back muscles, positive symptoms of Kernig and Brudzinsky (upper, middle, lower);
- in young children - bulging (rarely retraction) and straining of the large fontanel, positive Lesage symptom, head tossing.

PARACLINICAL RESEARCH:

1. General blood analysis - neutrophilic leukocytosis with a shift to the left, increased ESR;
2. Analysis of cerebrospinal fluid - neutrophilic pleocytosis, increased protein level, decreased sugar and chlorides;
3. Bacterioscopic examination of cerebrospinal fluid sediment and blood smears - "thick drop";
4. Bacteriological cultures on selective nutrient media - cerebrospinal fluid, blood, mucus from the nasopharynx to isolate the pathogen;
5. Serological methods (latex agglutination reaction (RAL), counter-

immunoelectrophoresis (CIEF) to determine the antigen of the pathogen.

TREATMENT

1. Strict bed rest until stable normalization of body temperature, disappearance of meningeal syndrome and normalization of blood and cerebrospinal fluid indicators, on average within 10-14 days;

2. Antibacterial therapy.

For meningitis of minor severity or associated with meningococcal infection, penicillin 300-500 thousand units/kg per day can be the starting antibiotic. If a meningitis patient shows signs of ITS, the starting antibiotic should be chloramphenicol 100 mg/kg per day (until the patient is discharged from ITS).

In severe forms of meningitis at the first stage of therapy (before the causative agent is identified), the drug of choice is ceftriaxone 100 mg/kg/day or cefotaxime 200 mg/kg/day. In children up to 1 month. life: ampicillin 150-200 mg/kg per day in combination with cephalosporins of the III generation or aminoglycosides (amikacin 15-30 mg/kg per day, netilmicin 6-9 mg/kg per day). After 24 - 48 hours from the start of the therapy, a control lumbar puncture is performed to control the effectiveness of the started therapy. The criterion of effectiveness is a decrease in pleocytosis by at least 1/3.

When the etiological cause of the disease is identified, the starting antibiotics can be replaced with others, according to the sensitivity of the pathogen. However, in the presence of pronounced positive dynamics, namely reduction of intoxication syndrome, normalization of body temperature, disappearance of meningeal symptoms, significant reduction of pleocytosis, improvement of general blood analysis), it is advisable to continue the initial therapy.

Reserve drugs in the absence of positive dynamics from the initial therapy for 48-72 hours are meropenem 120 mg/kg/day, cefepime 100 mg/kg/day, vancomycin 60 mg/kg/day.

The duration of antibacterial therapy should be on average: for meningococcal and influenzae meningitis - 7-10 days; with pneumococcal - 10-14 days; with streptococcal and listeriosis - 14-21 days; with meningitis caused by gram-negative bacilli - 21 days; with staphylococcal, enterococcal - 28 days.

Sanitation of the cerebrospinal fluid is a criterion for canceling antibiotic therapy. A control lumbar puncture is performed after stable normalization of temperature, disappearance of clinical signs of meningeal syndrome, normalization of general blood analysis. Therapy is stopped if the number of cells in 1 μl of cerebrospinal fluid does not exceed 50 due to lymphocytes.

In case of recurrence of purulent meningitis, a second course of reserve antibiotics (meropenem, ceftazidime, vancomycin) is prescribed.

3. Detoxification therapy is carried out with a 5% glucose solution in combination with a 7.5% potassium chloride solution, saline solutions (isotonic sodium chloride solution, Ringer's solution).

The total daily volume is no more than 2/3 of the physiological need (with normal diuresis and the absence of initial dehydration). From it, the infusion volume should not exceed 1/2 of the physiological need. From the second day, fluid deficit is maintained in the mode of zero water balance. The volume of infusion is 1/3 - 1/2 of the physiological need.

If oliguria or anuria occurs, the introduction of liquid is contraindicated until diuresis is restored.

4. For the purpose of dehydration, furosemide, mannitol is used.

5. Dexamethasone is prescribed to prevent sensorineural deafness in a daily dose of 0.15 mg/kg/weight every 4 hours in the first 2 days. The first dose of dexamethasone should be administered 10-30 minutes before the administration of the antibiotic.

COMPLICATION:

(treatment is carried out according to the relevant protocols)

- infectious-toxic shock (ITS);
- acute edema-swelling of the brain;
- acute intracranial hypertension;

- ependymatitis;
- meningoencephalitis;
- brain abscess;
- CSF hypotension syndrome;
- subdural effusion

Etiology of cerebral arachnoiditis (CA):

Infectious (flu) – 49%
Infectious and allergic (chronic tonsillitis, rheumatism, sinusitis, measles) – 26%
Traumatic (TBI) – 17%
Unexplained etiology - 12%
In case of specific infections (syphilis, brucellosis, tuberculosis)
Autogenic.

Infection with arachnoiditis can spread by the general hematogenous route in different directions or by regional vascular routes, when infectious-toxic-allergic substances from the corresponding blood and lymphatic vessels of the primary focus spread to the brain membranes of the same side at different distances. At the same time, connective tissue grows in the vascular membrane, cellular infiltration appears with further deposition of calcium salts.

Adhesions form between the membranes and the substance of the brain, then cystic cavities develop. In the future, all this leads to a violation of fluid dynamics. There are 3 types of histopathomorphological picture:

General changes - in the acute period.
Fibrous changes - in the remote period.
Hyperplastic plates - with repeated exacerbations of the process.

Additional risk factors for the development of CA:

Increased pressure of the cerebrospinal fluid in the acute period of the disease.
Increased content of protein in the cerebrospinal fluid during the acute period of the disease.
Severe TBI with subarachnoid hemorrhage.
Chronic recurrent course of infectious diseases (at least 3-4 months).

Classification of CA:

Adrift:
Acute - 7%
Subacute - 10%
Chronic - 85%

(the pia mater, arachnoid, periventricular zone (periventricular encephalitis), the walls of the ventricles of the brain and vascular plexuses (chorionependymatitis) are affected.

By nature, after zonal changes in the subarachnoid space, arachnoids are divided into:
Sticky
Cystic
mixed

Forms of CA by localization of the process:
Convexital - when the process is localized in the frontal, occipital, temporal, parietal lobes of the brain, and in the central gyri.

Basal - opticochiasmal, interpeduncular zone and transverse cistern are affected.

Posterior cranial fossa - mesocerebellar angle, lateral cistern of the brain, large cistern of the brain, cranio-spinal localization.

Diffuse 47%

Clinical course:

Most often, the disease begins gradually, subacutely with a subfebrile temperature. Headache, dizziness, nausea, vomiting gradually increase, unconsciousness may be lost (depending on the state of intracranial hypertension), convulsive syndrome and various psychopathological syndromes may develop. Blood pressure rises, changes in the fundus (phenomena of stagnation), asthenia and autonomic dysfunction increase.

The neurological status depends on the localization of the process.

Arachnoiditis of the posterior cranial fossa: (PSA) is a predominant lesion of the soft meninges in the area of the lateral or large cistern, as well as in the craniospinal area with a possible violation of the circulation of cerebrospinal fluid (CSF) in the posterior cranial fossa.

Predominantly general brain phenomena are noted - a headache in the back of the head (with irradiation to the eyeballs) is first constant, and then paroxysmal (related to a violation of the cerebral circulation), often accompanied by a forced position of the head. Changes in the fundus appear (from mild dilatation of retinal veins to congestive phenomena or secondary atrophy of the optic nerve). There is a violation of the mental state.

The main focal signs are cerebellar symptoms, lesions of CN - V, VI, VII, VIII, pairs and non-gross pyramidal insufficiency (asymmetry of normal and pathological reflexes).

In patients with occlusive forms of arachnoiditis, there is a gross violation of statics and gait. A characteristic dissociation between significant static disorders and minor coordination disorders.

Most often, occlusion occurs in a large cistern of the brain (arachnoid cysts) with the subsequent development of hypertensive-hydrocephalic syndrome. It is necessary to differentiate with brain tumors.

Basal arachnoiditis.

When the opticochiasmal zone is affected, there is stagnation of optic nerve discs, reduced visual acuity, concentric narrowing of visual fields, hemianopsia, tendinous hyperreflexia, central paralysis of the VII CN, certain pathological reflexes, retrobulbar neuritis, increased intracranial pressure.

When the interpeduncular cistern is affected, headache, total paralysis of the III CN and alternating paralysis are observed.

With arachnoiditis of the transverse cistern, the main symptom is damage to the auditory nerve with vestibular and auditory disturbances, headache, minor damage to the V and VII PN, mainly on one side.

Convexital arachnoiditis.

The leading clinical symptoms are signs of dysfunction of the frontal, parietal, temporal lobes and central gyrus zone.

Headache of a hypertensive nature, pain intensification is often associated with mental overload and physical fatigue. With percussion - pain in the bones of the skull.

Characteristic epileptic seizures. General weakness, dizziness, blood pressure instability, signs of autonomic dysfunction. There is damage to the pyramidal system in the form of anisoreflexia, some pathological reflexes, and a decrease in abdominal reflexes. Short-term visual disturbances, supranuclear lesions of the VII and XII pairs of CNS. At the bottom of the eye there is an expansion of the veins of the retina.

With CA, the protein fractions of the blood change (an indicator of the body's allergy).

With TSA normal
Total protein 5.8-7.25% 7.4%
Albumins 41.3-54% 54.6%
Globulins:
α1 4.4 % 4.4%
α2 10-14% 8%
β 11.9 % 11.9 %
γ 25-27.5 % 19.8 %
A/T becomes lower than normal.

Autoantibodies to brain antigens are detected in blood serum, which emphasizes the immunological genesis of the disease.

When prescribing therapy, you should take into account:
Morphological changes in the membranes of the brain.
The state of hemo- and lycodynamics.

Treatment should be comprehensive.

When prescribing treatment, first of all, it is necessary to establish the primary source of infection in the patient's body.

In the first week of the disease, the following are prescribed: antibiotics, sulfonamides, corticosteroids, desensitizing, antispasmodic drugs, vitamins of group B and C, calcium and nicotinic acid drugs, brain metabolites. From the second week, anticholinesterase and resorbing drugs are added. In the third week, you can add iodine preparations. Pathogenetic therapy consists in the appointment of long-term courses of absorbent drugs, drugs that contribute to the normalization of intracranial pressure and drugs that improve cerebral blood circulation and metabolism in brain tissues.

Treatment should be carried out systematically, over several years, until a stable remission is achieved.

Materials for self-control of training quality

Tests

	Standard
1. Is a recurrent course of meningococcal meningitis possible?	Yes
2. Are tuberculous nodules located at the base of the brain?	Yes
3. Does the level of sugar in the cerebrospinal fluid decrease with the primary serous meningitis?	No
4. Is a fibrinous film in the cerebrospinal fluid characteristic of syphilitic meningitis?	No
5. Is the rapid development of tuberculous meningitis characteristic?	No
6. Is a decrease in chlorides in the cerebrospinal fluid characteristic of tuberculous meningitis?	Yes

1. Indicate in which meningitis is observed a decrease in sugar in the cerebrospinal fluid? A) meningococcal, b) secondary purulent, B) tuberculous, d) primary serous.	**Standard IN)**

Which of **the listed symptoms** : a) a long prodromal period B) acute onset, c) a sharp increase in temperature at the end, D) minor meningeal symptoms, d) opalescent cerebrospinal fluid, e) positive protein reactions, g) lymphocytic pleocytosis, h) fibrin film, i) small lymphocytic leukocytosis in the blood - **characteristic of** : 1) tuberculous and 2) acute serous lymphocytic meningitis?	1-a, c, d, d, e, z, z 2-b, f, i.

Tasks for self-control :
A 21-year-old patient came in with complaints of severe headache, vomiting, and double vision. It is known from the anamnesis that he got meningitis 10 days ago, when he felt general weakness, frailty, moderate
headache. Subfebrile temperature was noted. Over the course of 6 days, the headache gradually increased sharply to unbearable, there was double vision, vomiting. Objectively: stiffness of the muscles of the back of the head, symptoms of Kernig-Brudzinsky, anisocoria, left pupil wider, ptosis of the jaw, diverging strabismus (absent movement of the left eyeball towards the nose). In cerebrospinal fluid lymphocytic pleocytosis, reduced sugar content, a film formed when cerebrospinal fluid settled.
1. Establish a diagnosis
2. Prescribe treatment.

Answer
1. Tuberculous meningitis.
2.a) Streptomycin,
b) PASK.
c) Vitamins of group B
b) Corticosteroids
d) Dehydrating preparations

Materials for classroom self-training:
List of educational practical tasks:
1. To study meningeal signs.
2. Investigate the composition of liquor.
3. Determine the presence of photo and phonophobia.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 22

Topic: Encephalitis

Purpose: to create an idea among the applicants about the classification of encephalitis, clinical forms of encephalitis, pathomorphological changes in the mucous membranes and brain substance in encephalitis, general brain symptoms, general infectious symptoms, basic treatment schemes for patients with encephalitis.

Basic concepts: neuroinfections make up about a third of all pathology of the nervous system. In recent decades, the epidemic situation in the world has changed. The prevalence of some neuroinfections decreased, but new ones accompanied by severe damage to the nervous system appeared. New types of pathogens appear in the surrounding nature as a result of transformation and modification of old ones. Some types of pathogens are transferred to regions geographically quite distant from the former place of their distribution. There is a change in the types of carriers of a number of infections. Therefore, the study of this topic is necessary for every doctor.

Equipment: classroom, furniture, equipment.

Plan:

3. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
4. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Encephalitis - inflammation of the brain. This name unites a group of diseases caused by various pathogens. Changes in the immunological reactivity of the body play an important role in the development of these diseases.

Encephalitis is divided into primary and secondary.

Primary encephalitis is caused by neurotropic viruses that penetrate directly into the cells of the nervous system and destroy them. Such encephalitis includes epidemic, tick-borne, mosquito-borne encephalitis, and in addition, encephalitis caused by poliomyelitis-like viruses, herpes simplex virus. However, it is not always possible to detect the virus that caused encephalitis. These forms of pathology are most often found in young children.

Secondary encephalitis, as a rule, is a complication of such infectious diseases as measles, chicken pox, toxoplasmosis. Rarely, secondary encephalitis develops after preventive vaccinations.

In all forms of encephalitis, inflammatory changes in the brain occur in the acute period. These changes are expressed in edema, increased blood vessel filling, destruction of nerve cells and their processes. In the future, persistent disorders may form in the changed brain tissue in the form of growth of connective tissue elements, scars, cysts (a cyst is an inflammatory cavity bounded by a membrane and filled with fluid), adhesions. Depending on the predominant damage to brain cells or conductors, encephalitis is divided into polioencephalitis ("polio" - gray, i.e. damage to brain cells), leukoencephalitis ("leuko" - white, i.e. damage to white matter). Examples of polioencephalitis are acute epidemic (lethargic) encephalitis and the encephalitic form of poliomyelitis. Most forms of encephalitis occur with simultaneous damage to both cells and the leading pathways of the brain, so they are called panencephalitis. Primary viral panencephalitis includes tick-borne, mosquito-borne and other forms.

Encephalitis is a serious disease and, along with meningitis, constitutes the main group of infectious diseases of the nervous system. Encephalitis affects people of all ages.

The disease is especially severe in children. It usually starts acutely. In full health or against the background of the main disease (influenza, measles, chicken pox, etc.), the patient's condition

deteriorates sharply, the body temperature rises, general brain and focal symptoms of brain damage develop.

General cerebral symptoms include headache, dizziness, vomiting, convulsions, impaired consciousness - from its light blackout to deep coma. The comatose state is characterized by a severe disorder of brain functions: the patient is unconscious, does not react to the environment, the regulation of vital functions (breathing, blood circulation) is disturbed, the reaction of the pupils to light is sluggish or absent, tendon reflexes are suppressed. In some cases, psychomotor agitation, delirium, and hallucinations are observed. Focal symptoms of brain damage can be detected in the acute stage of encephalitis. They largely depend on the form of the disease and the prevalence of inflammatory and degenerative changes.

Epidemic encephalitis mainly affects formations located near the ventricles of the brain (reticular formation, nuclei of oculomotor nerves, autonomic centers). Clinically, this is expressed in increased drowsiness or insomnia, strabismus, double vision in the eyes, the difference between the pupils (size and shape). Vegetative disorders are characterized by disorders of the frequency and rhythm of breathing, palpitations, fluctuations in blood pressure, persistent increase in body temperature, increased facial fat, thirst and other symptoms.

Focal symptoms in tick-borne encephalitis arise as a result of selective damage to nerve cells of the Varolian pons, medulla oblongata and midbrain, as well as motor cells of the anterior horns of the spinal cord. Clinically, it is manifested by hanging of the head, lethargic paralysis of the arms and upper shoulder girdle. When the medulla oblongata is affected, a symptom complex of bulbar paralysis is observed: swallowing disorders, hoarse tone of the voice or its complete absence, paralysis of the tongue muscles, breathing and heart activity disorders. These changes are the main causes of death in the truncal form of tick-borne encephalitis.

In encephalitis, which develops as a complication after chicken pox, there are characteristic cerebellar and vestibular disorders, which are relatively rarely observed in other encephalitax. Due to impaired coordination, children cannot eat, dress, walk and sit on their own. However, with most encephalitis, there is no pronounced selectivity of damage to individual brain structures. Paresis and paralysis, coordination disorders, sensitivity disorders, hyperkinesis, vegetative disorders, speech disorders and other higher cortical functions, etc. are observed.

Depending on the predominance of certain focal symptoms of brain damage in the clinical picture, the following forms of encephalitis are distinguished: cortical, subcortical, mesodiencephalic, trunk, trunk-spinal, and others. Damage to the brain substance can be accompanied by inflammatory changes in the membranes. In these cases, the disease is considered as meningoencephalitis.

The diagnosis of encephalitis is made in the case of an acute development of the disease, as well as on the basis of the data of a neurological examination, which reveals general brain and focal symptoms. Information about the epidemic situation is important for making a diagnosis. The presence of enterovirus infections in the children's team and the fact of the patient's stay in an endemic area, that is, for example, in an area where there are ixodid ticks and rodents, are taken into account. The diagnosis is confirmed if inflammatory changes are detected in the blood and cerebrospinal fluid. With the help of special virological and immunological studies, the virus that caused encephalitis can be isolated and determined.

In the course of encephalitis, several stages are distinguished: acute, recovery and pesidyalny, that is, the period of persistent consequences. The duration of the acute and recovery stages, the severity of encephalitis depend on the pathogenic properties of the pathogen, the body's defenses and the age of the patient. The duration of the acute period is very variable: from 10-15 days to several months. With some encephalitis, a chronic period of the disease is observed. Its development is caused by a change in immunoreactive processes in brain tissues, as a result of which the dead cells become foreign - an autoimmune ("auto" - self, own) process develops. Clinical symptoms of the chronic stage of encephalitis appear either at the end of the acute period or after months or years. With tick-borne encephalitis, the symptoms of the chronic period are

called Kozhevnikov epilepsy. It is characterized by constant involuntary twitching of the muscles of the face, neck, and shoulder girdle. Periodically, these twitches spread to other muscles, leading to the development of a large convulsive attack with loss of consciousness. With epidemic encephalitis, the chronic period is characterized by the gradual development of parkinsonism syndrome.

The consequences of transferred encephalitis are largely determined by the age of the patient. If the disease was transmitted in early childhood, the process of development of the central nervous system may be disturbed. Disorders of growth and differentiation of cerebral cortex cells lead to the formation of secondary mycocephaly. The death of nerve cells can be the cause of gross delays in mental and motor development. In children, interest in the environment is reduced, and often absent. The development of other higher cortical functions - memory, attention, thinking - is also disturbed. If the nervous system was already formed by the time of the disease, the consequences of the transferred encephalitis largely depend on which brain structures and how deeply they were affected during the acute period of the disease. The most frequent consequences include paresis and paralysis of the limbs, as well as focal symptoms of brain stem damage in the form of impaired cranial nerve function. Endocrine-metabolic disorders can be manifested by growth retardation, obesity, trophic disorders of nails, hair, etc.

One of the frequent consequences of encephalitis is a convulsive syndrome. Repeated convulsions, as a rule, are the cause of intellectual impairment. The patients' memory, attention, ability to study decrease. They become irritable, petty, aggressive, sticky. This often complicates the patient's contact with others.

Isolated vision and hearing loss is relatively rare after encephalitis. Violations of higher cortical functions are associated not only with focal lesions of the brain, but also with a disorder of the integral analytical and synthetic activity of the cortex, which leads to disorders of speech, writing, reading, calculation, severe emotional and volitional disorders. The latter are manifested by a tendency to emotional outbursts, unstable mood. Patients are disinhibited, inadequate, aggressive, which is often the cause of conflict situations in the family and school. Decline in intelligence can vary from mild to severe degrees. An uneven, disharmonious decline in intelligence is described. In the acute period, urgent measures aimed at combating cerebral edema, stabilization of breathing, cardiac activity, and normalization of metabolism should be carried out. In the presidial stage, treatment measures are carried out with the aim of compensating for impaired functions and social adaptation of patients.

The herpes simplex virus is classified as a pantropic virus capable of affecting various organs and systems (skin, mucous membranes, nervous system, liver). The virus enters the central nervous system by hematogenous and perineural routes. Long-term persistence in the body and the ability to periodically activate under the influence of non-specific factors are characteristic.

Clinical picture: the disease begins acutely. from a rise in body temperature. Meningeal symptoms appear quickly. General epileptic seizures often occur. Focal symptoms are manifested by central mono- and hemiparesis, hyperkinesis. In the cerebrospinal fluid, pleocytosis with a predominance of lymphocytes (up to several hundred cells in 1 μl), an increase in protein content (up to 2-3 g / l), light xanthochromia or a small admixture of erythrocytes are detected.

The diagnosis is confirmed by various serological reactions and the method of immunofluorescent antibodies. With CT scans, zones of pathologically reduced density in the brain substance are revealed in the early stages. The course is usually difficult. Mortality is much higher. than in other viral diseases of the nervous system. In rare cases, complete recovery is possible without consequences. Mostly, patients with herpetic encephalitis have focal symptoms, "giant" slow waves on the EEG.

The essential role of cytomegalovirus infection among viral diseases of the central nervous system. For the diagnosis of cytomegalovirus meningoencephalitis, it is necessary: analysis of cerebrospinal fluid for the permeability of the blood-brain barrier and intrathecal synthesis of IgG, data of instrumental studies (magnetic resonance imaging of the brain). Difficult differential

diagnosis, taking into account the onset and further progression of the disease, between systemic vasculitis and the debut of demyelination. For treatment, etiotropic drugs for cytomegalovirus, ganciclovir and panavir, are used.

Materials for self-control of training quality

Tests

1.	The patient, a 35-year-old lumberjack, developed toothache, high body temperature, and headache at the end of May. On the 6th day, hiccups, weakness of the hands joined, the head stopped holding and fell on the chest. The neurological status shows paresis of the hands with muscle atony, areflexia. The head hangs. What is the previous diagnosis? What is the form of the disease?	Tick-borne encephalitis, poliomyelitis form
2.	A patient with a high temperature, pathological sleepiness was found to have divergent squint, facial fat. What is the previous diagnosis?	Acute stage of lethargic encephalitis

1.	Specify the main clinical differences between encephalitis and meningitis? A) presence of meningeal syndrome; B) loss of consciousness; C) the presence of general brain symptoms; D) the presence of inflammatory neurological symptoms	D)
2.	List the main types of prevention of tick-borne encephalitis	1. Vaccination 2. Wearing protective clothing 3. Reduction of ticks and rodents
3.	Name the clinical varieties of the acute period of epidemic encephalitis	1. Hyperkinetic 2. Vestibular 3. Diencephalic 4. Oculo-cephalic 5. Mental disorders 6. Abortive

Tasks for self-control :

The patient, 30 years old, has a low-grade fever, diplopia, ptosis on the left side, hyperhidrosis, hypersalivation, drowsiness, increased muscle tone of the plastic type. In the cerebrospinal fluid: lymphocytic pleocytosis; in the blood - leukocytosis, increased ZE; EEG - desynchronization of cortical neutron activity.

1. Make a diagnosis.
2. Prescribe treatment.

Answer

1. Epidemic encephalitis
2. a) anti-inflammatory drugs
   b) vitamin therapy
   c) desensitizing therapy

A 32-year-old patient gradually developed tremors, slowness of movements, monotonous quiet speech, a mask-like face, and hypersalivation. The gait is shuffling, with small steps. In addition to the "flexors", 2 years ago he suffered a disease with a high temperature (in the words of the patient, "the flu").

1. What syndrome is observed in the patient?
2. Specify topical and clinical diagnoses?
3. What treatment will you prescribe for the patient?

Answer

1. Parkinsonism syndrome.
2. Damage to the pallidone-nigral system of the brain. Chronic stage of lethargic encephalitis.
3. Antiparkinsonian drugs (cyclodol, midantan, selegelin, levodopa, nacom, steelo, parkopan).

Materials for classroom self-training:

List of educational practical tasks:

1. To study the functions of the brain.
2. To study the functions of the spinal cord.
3. Check the state of intracranial pressure.
4. To check the presence of dislocation of the middle structures of the brain with the help of Echo-EG.
5. Make a topical and clinical diagnosis.
6. Prescribe adequate treatment.

Instructional material for mastering professional skills

1. Carefully collect anamnesis.
2. To detect the presence of a focal lesion of the n.s.
3. To check the neurological status of the patient, determining the presence of meningeal signs.
4. If necessary, prescribe an Echo-EG, cranio- or spondylogram, CT scan, consultation of an ophthalmologist.
5. Make a diagnosis.

Practical lesson No. 23

Topic: Poliomyelitis. Acute myelitis. Amyotrophic lateral sclerosis.

Purpose: to create an idea of the etiology, pathogenesis, epidemiology of poliomyelitis, clinical classification of the disease, diagnosis and differential diagnosis of poliomyelitis, treatment in the acute and convalescent period, polio-like diseases in children, to teach the

recipients to diagnose and carry out differential diagnosis of poliomyelitis and poliomyelitis-like diseases, justify clinical research methods for the diagnosis of poliomyelitis, prescribe treatment in the acute and convalescent periods of the disease, predict the consequences of the disease and provide preventive measures.

Basic concepts: poliomyelitis is an acute infectious disease of viral origin, which mainly affects the spinal cord, has the character of an epidemic and is especially widespread in children's groups. This disease leads to severe consequences and is often the cause of death or permanent disability.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Materials for self-control of training quality

Tests

1) Symptoms of damage to the anterior horns of the spinal cord:
a) central paralysis;
b) peripheral paralysis; Answer: b)
c) sensitive ataxia;
d) root-type anesthesia;
e) anesthesia by conductor type.
2) Symptoms of damage to the tracts of Hall and Burdach:
a) sensitive ataxia;
b) root-type anesthesia;
c) central paralysis; Answer: a)
d) peripheral paralysis;
e) atrophy.

1) Where is the I neuron of the motor pathway located?
2) Where is the II motor neuron located?
a) bark;
b) undercut; Answer:
c) cerebellum; 1)- a
d) anterior horns of the spinal cord; 2) - Mr
e) posterior horns of the spinal cord.

3) What happens when the anterior horns of the spinal cord are damaged ?
4) What happens when the posterior horns of the spinal cord are damaged?

a) violation of sensitivity;
b) flaccid paralysis; Answer:
c) violation of coordination; 3)-b
d) central paralysis; 4)-a
e) dysfunction of the pelvic organs.

4. What is the etiology of ALS?

A. Infectious
B. Spadkava
B. Traumatic
G. Sudynna
D. Tumor Answer: A

5. What is the clinical course of ALS?
A. Sharp
B. Remitting
V. Chronic Answer: V

6. Where is the focus of damage in ALS localized?
A. Fibers of the pyramidal tracts
B. Muscles
B. Intervertebral discs
D. Peripheral nerves
D. Extrapyramidal system. Answer: A.

Tasks for self-control :

A 6-year-old child complains of feeling unwell, a sore throat, loose stools, and an increase in temperature. The pediatrician prescribed treatment against GDV I , the temperature normalized. The next morning, the child stopped moving his left leg. Objectively: cranial nerves without pathology, active movements in the left leg are absent. Muscle tone is reduced. Knee and Achilles reflexes are not triggered. There are no pathological reflexes. Meningeal symptoms of Kernig (++), Brudzinsky (++), stiffness of the occipital muscles are caused. .

Analysis of cerebrospinal fluid: transparent, colorless, flows out under pressure. Protein - 0.45‰, cytosis - 60/3, (neutrophils). Sugar - 60mg%. Analysis of blood and urine without pathology. After 5-6 weeks, the movements began to recover, he began to walk, while walking - steppage.

Question:
1. Name the diagnosis of the disease?
2. How to explain sore throat and unstable stool?
3. Where is the lesion in this disease?
4. Why did the meningeal symptoms occur?

Answers:
1 **.** Poliomyelitis **.**
2. Inflammatory reaction in the place where the virus meets the body.
3. Motor cells of the front horns.
4. From irritation of membranes by viral infection and secondary CSF hypertension.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

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- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

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Practical lesson No. 2 4

Topic: Neurosyphilis. Tuberculosis of the nervous system.

The goal: to create in the students an idea of the etiology and pathogenesis of neurosyphilis, diagnosis of neurosyphilis, the clinic of early and late neurosyphilis, the basic principles of treatment and prevention of neurosyphilis, to create in the students an idea of the etiology , pathogenesis, the main clinical manifestations of neurotuberculosis, damage to the nervous system, etc. associated with infections that develop against the background of immunodeficiency, damage to the nervous system in tuberculosis, ways of tuberculosis transmission, clinical manifestations of tuberculous meningitis, local brain lesions: causes, symptoms, ways to prevent tuberculosis.

Basic concepts: syphilis is a chronic infectious venereal disease that affects all organs and is characterized by a progressive course. A person cured of syphilis does not develop persistent immunity, so re-infection (reinfection) is possible. This disease leads to extremely complex

damage to the nervous system and to persistent, progressive disability of the population. Given that there is currently an outbreak of this disease, doctors of all specialties must be well aware of it in order to carry out work both in terms of identifying patients and in order to prevent the spread of the disease. In connection with different periods of occurrence of damage to the nervous system, different pathogenesis and pathomorphological changes, as well as localization of the pathological process and different clinical manifestations, it is important to study the main clinical syndromes of tuberculosis. In order to develop measures to prevent the further spread of this infection.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Logical structure graph

Neurosyphilis

Mesenchymal forms (damage of membranes, blood vessels inflammatory)			Ectodermal forms (damage to neurons, glia degenerative)
early (primary period of syphilis 3-8 weeks)	Later ones (secondary period 2-3 years)	late (tertiary period 3-6 years)	(quaternary 10-15 years)
meningeal, meningoradicle. syndromes, Liquor syphilis	Acute serous Meningitis Meningomyelitis Neuritis, neuralgia Plexites	Chr. meningitis and meningomyelitis Gumma Pachymeningitis Endarteritis	Spinal tuberculosis Progressive paralysis Amyotrophic spinal syphilis Erb's spastic paralysis

Materials for self-control of training quality

Tests

1. List the clinical forms of early mesenchymal neurosyphilis:

a. *A) meningomyelitis
b. *B) rubber
c. C) progressive paralysis
d. D) spinal tuberculosis
e. *D) endarteritis
f. E) Strümpel's paraplegia
g. F) Friedreich's ataxia

2. Name the clinical forms of late, parenchymal neurosyphilis:

a. A) meningitis
b. B) meningomyelitis
c. C) endarteritis
d. *D) spinal tuberculosis
e. *D) progressive paralysis

3. State the signs of basal syphilitic meningitis:

a. A) paresis of limbs
b. *B) oculomotor disorders
c. C) violation of superficial types of sensitivity
d. *D) neuritis of the optic nerves
e. *D) Argyle-Robertson syndrome
f. E) violation of urinary excretion

4. List the main clinical signs of spinal tuberculosis:

a. A) bulbar syndrome
b. *B) shooting pain, paresthesias
c. *B) sensitive ataxia
d. D) cerebellar ataxia
e. *D) absence of knee and Achilles reflexes
f. *E) primary atrophy of optic nerve discs
g. *E) Argyle-Robertson syndrome
h. G) Bernard-Horner syndrome

5. Name the diseases with which the gum of the brain is differentiated:

a. A) Huntington's chorea
b. *B) brain abscess
c. C) amyotrophic lateral sclerosis
d. * D) parasitic diseases of the brain
e. *D) membrane hematomas
f. E) polyneuropathy

6. Specify the signs of the neurological stage of spinal tuberculosis:

a. *A) radicular pain, paresthesias
b. B) congestive discs of the optic nerves
c. B) hyperreflexia of knee and Achilles reflexes
d. *D) Argyle-Robertson syndrome
e. D) reverse Argyle-Robertson syndrome

7. Name the signs of the ataxic stage of spinal tuberculosis:

    a. A) cerebellar ataxia
    b. *B) sensitive ataxia
    c. *B) Argyle-Robertson syndrome
    d. D) hyperreflexia of knee and Achilles reflexes
    e. *D) absence of knee and Achilles reflexes

8. Specify the signs of the paralytic stage of spinal tuberculosis:

    a. A) cerebellar ataxia
    b. *B) pronounced sensitive ataxia
    c. *B) trophic disorders
    d. *D) paresis of limbs
    e. *D) Argyle-Robertson syndrome

9. Specify the drugs used to treat neurosyphilis:
    a. A) sulfonamides
    b. B) coagulants
    c. *B) penicillin
    d. *D) potassium iodide
    e. D) unitiol, cuprenil
    f. *E) bioquinol

10. Name the main stages of ankylosing spondylitis:

    a. A) preparalytic
    b. B) residual
    c. *B) neuralgic
    d. D) stage of Brown-Sécar paralysis
    e. *D) atactic
    f. *E) paralytic
        1. The most common causes of decreased hearing acuity in patients with tuberculosis are:
    g. A. meningitis
    h. B. encephalitis
    i. B. retinitis
    j. G. myelopathy
    k. D. neuropathy Answer: A.

11. A symptom of vacuolar myelopathy is:
    a. A. hemianaesthesia
    b. B. paresthesias
    c. B. weakness in the legs
    d. G. ataxia
    e. D. hemiparesis Answer: V,S.

12. Which of the following is not characteristic of tuberculous meningitis?
    a. A) gradual onset of the disease
    b. *B) sudden onset of the disease
    c. *B) high, hectic temperature

d. D) subfebrile temperature
e. D) lesions of the CNS

13. What is characteristic of the changes in the cerebrospinal fluid with tuberculosis of the NA?
    a. A) neutrophilic pleocytosis
    b. *B) lymphocytic pleocytosis
    c. *B) decrease in the amount of glucose
    d. D) increasing the amount of glucose
    e. D) significant increase in protein content

14. Which of the drugs should not be prescribed for tuberculosis?
    a. A) streptomycin
    b. B) rifampicin
    c. B) isoniazid
    d. D) ethambutol
    e. *D) benzohexonium

Recommended literature:

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Practical lesson No. 25

Topic: Demyelinating diseases of the nervous system.

Purpose: to acquaint applicants with the epidemiology of multiple sclerosis, its pathogenesis, features of the clinical course of the disease.

Basic concepts: multiple sclerosis is a severe organic disease of the central nervous system, which belongs to the demyelinating pathology, and affects the young, able-bodied part of the population. Steady progressiveness, the course with and without remissions and exacerbations leads to permanent disability due to paralysis, ataxia and premature death, if intercurrent diseases join, deep general trophic disorders and damage to vital centers of the brain stem.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Demyelinating diseases are a group of diseases of the nervous system in which the myelin sheath of nerve conductors is damaged.

Myelin diseases are divided into two main groups: myelinopathy and myelinoclasty. Myelinopathy associated with a biochemical defect in the structure of myelin, as a rule, is genetically determined. The basis of myelinoclastic (or demyelinating) diseases is the destruction of normally synthesized myelin under the influence of various actions, both external and internal. The most common disease from the entire group of myelin diseases is multiple sclerosis.

Multiple sclerosis.

This disease usually manifests itself in the form of repeated exacerbations, accompanied by symptoms of neurological dysfunction caused by focal or multifocal lesions of the central nervous system. Exacerbations alternate with remissions and occur again at different intervals over many years. As a rule, the disease begins at a young age. The frequency of exacerbations is highest in the first 3-4 years of the disease. The first episode of the disease can be so inconspicuous that it is not a reason to consult a doctor, and later the patient hardly remembers it; in addition, the next exacerbation may occur only after 10-20 years. During typical episodes, symptoms increase over a period of several days to 2-3 weeks and then regress. Recovery is usually quick, within a few

weeks, but sometimes the process can take months. The degree of recovery varies significantly in different patients and with different exacerbations in the same patient. Remission can be complete, especially after the first attacks; however, remissions are often incomplete, and as one exacerbation replaces another, there is a gradual progression of the disease with increasing permanent neurological deficits. Approximately 30% of patients have a slow but steadily progressive course of the disease. This is most typical for cases with the onset of the disease after the age of 40. Although some patients die within the first few years after the onset of multiple sclerosis (PC), the average survival in PC is more than 30 years from the onset of the disease. PC differs in polymorphism of clinical manifestations. The clinical picture is determined by the localization of the focus of demyelination in the central nervous system. Among the classic signs are visual disturbances, nystagmus, dysarthria, decreased vibrational sensitivity and sense of position, ataxia and intentional tremor, weakness or paralysis in one or more limbs, muscle spasticity, urinary disorders.

Reliable criteria for the clinical diagnosis of PC are a clear history with indications of at least two episodes of neurological deficit and objective clinical symptoms that indicate the presence of more than one lesion in the CNS. Detection of the second lesion using laboratory methods (for example, evoked potentials, CT, NMR, and urological studies) in combination with objective data on the first lesion serves as an additional diagnostic criterion. The presence of an elevated level of immunoglobulins in the CSF when tested with oligoclonal bands confirms the diagnosis, but does not replace the above criteria. Clinically probable PC is diagnosed after two exacerbations with clinical symptoms of one lesion or after one attack with clinical signs of two lesions (or in the presence of one focus that manifests itself clinically and the second - instrumental). Observations in the dynamics of patients with probable PC testify to the significant diagnostic accuracy of these criteria. If the patient has symptoms of simultaneous involvement of the optic nerves, brain stem, and spinal cord and indicates in the history of more than one exacerbation episode, the diagnosis of PC can be established with a probability exceeding 95%. In the first years of the disease, after there have been only a few exacerbations and persistent neurological symptoms are slightly pronounced, making a diagnosis can be difficult, and it is necessary to exclude single or multiple lesions due to other causes. Pathological changes. A distinctive pathological feature of PC is numerous scattered discrete foci of demyelination, called plaques. Macroscopically, the plaques look like areas with clear outlines of gray-pink color, protruding against the background of the surrounding white matter of the central nervous system. Lesions may spread to the gray matter, but microscopic examination shows preservation of nerve cell bodies. The sizes of plaques vary from several millimeters to several centimeters; larger plaques are formed when small plaques merge or due to the spread of their boundaries. Plaques are found throughout the white matter, but are more often localized in the paraventricular and subpial regions of the brain, as well as inside the brainstem and in the spinal cord. Their topography is consistent with the venous drainage system of the brain and spinal cord, while their correspondence to certain anatomical structures was not found. The peripheral nervous system remains intact. The number of plaques detected at autopsy invariably exceeds the number of plaques expected based on the existing symptoms. Consequently, many plaques remain mute; this indicates a significant conduction of impulses through areas of demyelination. Indeed, according to pathological studies, 20% of patients with PC remain clinically silent throughout their lives. Microscopic features of PC plaques depend on their age. Lesions of various ages and signs of fresh activity are usually found at the edges of old lesions. Fresh lesions in PC are characterized by accumulations of T-lymphocytes and monocytes with macrophages near the venules and along the borders of the plaque, where the process of myelin destruction is taking place. Inflammatory cells penetrating the white matter are responsible for the destruction of myelin. It is believed that macrophages (microglia) serve as vectors of myelin breakdown. They also perform the function of disposal of myelin fragments; fat-laden macrophages can persist for months and possibly years after the acute inflammatory response has subsided. Plasma cells accumulate inside the plaques and are usually found in their central parts or near the centers. Within the foci of demyelination or

near their borders, a characteristic reaction of astroglia is observed. In the formed, inactive plaques, dense fibrillar-glious deposits are usually found in areas of demyelination and only a small number of residual perivascular macrophages. It was reported that the number of oligodendrocytes at the edges of the plaque was normal or increased. Meanwhile, inside the plaques, the number of oligodendrocytes is reduced; this indicates that cells of this type in PC eventually die. Thus, the defeat of oligodendrocytes can be a primary event.

In PC, only limited myelin regeneration occurs. The reason for this is unclear, but may be related to the death of oligodendrocytes. From the pial edges of plaques in the spinal cord, remyelination can occur at the expense of Schwann cells of peripheral nerves. Despite careful searches, no viral inclusions were detected in the pathological foci of PC. Mechanisms of recovery after PC exacerbations are apparently diverse. The resolution of edema, confirmed by CT scan, as well as inflammation ensures restoration of saltatory conduction along partially demyelinated axons (plaque shadow). The recovery of conduction may also be partially associated with the inclusion of K + channels along the length of exposed axon segments, and not exclusively with Ranvier intercepts, as in the case of myelinated nerves.

In most cases, axons inside the plaques are preserved, although in acute lesions, pronounced necrosis is sometimes observed, accompanied by the death of axons. Significant loss of axons is found in at least 10% of PC plaques. As shown by ultrastructural studies, the death of axons appears to be more widespread than it can be established by conventional histological analysis. There are pathological anatomical changes of all degrees of severity.

Correspondence of the pathoanatomical features of PC with the growth characteristic of this disease and the reduction of neurological symptoms, which occurs in a matter of hours or days, cannot be traced. Disturbed conduction of impulses along demyelinated nerves changes, and these changes are associated with transient fluctuations in the body's internal environment, such as changes in temperature or electrolyte balance, stress. Fever and even a slight increase in body temperature, for example, when taking a hot bath or during physical exertion, can cause decompensation of conduction through areas of demyelination and lead to the disappearance of symptoms. The mechanism of such axonal fatigue is unknown, but the presence of some type of conduction block is assumed. It is important to distinguish the transient fluctuations of symptoms considered here from exacerbations of the disease.

Etiology. The cause or causes of PC remain unknown. Immunological or infectious factors have been suggested to play a role, but evidence supporting these concepts is fragmentary and circumstantial.

Epidemiology. Epidemiological studies have established several facts that must necessarily be included in any coherent theory of the disease. The average age of the first clinical episode of PC is in the 3rd-4th decade of life. Among the patients, 60% are women. It is extremely rare that the disease begins in childhood and at the age of over 60, but such cases are known.

In general, the frequency of the disease in climatic zones with a temperate climate exceeds that in the tropics, but there are differences in frequency between regions with similar climates. Therefore, the prevalence of the disease does not depend only on geographical latitude and temperature. The frequency of PC in the North of Europe, in Canada and the northern states of the USA reaches about 10 new cases annually per 100,000 inhabitants aged 20 to 50 years. The frequency in Australia, New Zealand and the southern states of the USA is from 1/3 to 1/2 of this indicator; in Japan, all countries of the East and Africa, PC is rarely observed. Some epidemiological data also suggest that persons migrating in childhood from areas of high risk of PC to areas of low risk may be partially protected from PC. These data are consistent with the existence of an environmental factor, probably viral in nature and possibly geographically limited, that influences the development of PC.

Genetic factors. The frequency of PC among American Indian and Negroid populations is lower than among the Caucasian population living in these areas. This suggests that genetic factors also affect susceptibility to the disease. Close relatives of PC patients (parents, brothers, sisters) have an 8-fold increased risk of PC disease. This may reflect the interaction of several genetic

factors along with the influence of environmental conditions or the combined effect of those and other factors. When studying PC in monozygotic twins, it was found that they have a higher concordance in PC than in dizygotic twins; concordance among identical twins exceeded 50%. In family studies, it was not possible to identify any prognostic genetic features, but their results strongly suggest a genetically determined predisposition to the disease.

Among patients with PC, certain histocompatibility antigens (HLA) are found with increased frequency. HLA-B7 and DW2 alleles are found with a high frequency in Caucasians suffering from PC. Most diseases for which an association with HLA has been established are autoimmune in nature; it is from these positions that the etiology of PC is currently considered. The DW2 allele is found in the majority of Americans of Negroid origin who suffer from PC; this allele is rare in representatives of the Negroid population in Africa, among whom cases of PC are practically unknown. Therefore, there must be an HLA-linked genetic factor that predisposes to PC, but since the vast majority of individuals with B7 or DW2 do not develop the disease, additional genetic or environmental factors appear to play a role. It seems paradoxical that in sibs, PC concordance, HLA haplotype concordance indicators slightly exceed possible expected values. The HLA-B12 allele is less common in PC patients than in the general population. These data suggest that genetically determined protective factors may act in PC.

Autoimmune factors. Lesions characteristic of PC are imitated by pathological changes in experimental allergic encephalomyelitis (EAE) - an autoimmune disease induced in animals by immunization with myelin. Lesions in EAE are demyelinating, periventricular, reminiscent of plaques, present in chronic and relapsing forms and characterized by an inflammatory infiltrate consisting of lymphocytes, macrophages, and plasma cells. In the CSF during the active stage of the disease, obvious signs of T-cell activation are observed. Excess production of IgG in the CNS is characteristic of all stages of PC; whether this reflects the presence of some B-cell stimulator in the PC brain or the result of a defect in immune regulation is unknown. Viral infection of the brain continues to be considered a possible cause of PC, despite the fact that all attempts to isolate the virus from the brains of PC patients or to visualize the virus in brain tissue have failed.

Provocative factors. It is believed that the factors provoking the first exacerbation of PC can be various infections, injuries and even emotional disorders. However, there is no convincing evidence of this. The probability of exacerbation of PC during the first 6 months of pregnancy is higher than random, but it has been established that during the II and III trimesters of pregnancy the risk of exacerbation decreases. It was also not possible to show the connection of trauma, including lumbar puncture, myelography and surgical intervention with exacerbations or increasing disability, as well as the influence of emotional disruptions on the pace of the course of the disease. As practice shows, vaccinations also do not provoke an exacerbation of PC.

Clinical manifestations. A first episode of PC may present with only a single symptom (45% of cases) or more than one symptom (55%). Approximately 40% of PC patients have optic neuritis at the time of disease onset or at some point during its course.

Optic neuritis is manifested by partial or complete loss of vision, usually in one and rarely in both eyes, and is often accompanied by pain when moving the eyeball. Macular vision (central scotoma) is most affected, but the observed visual field defects are very diverse. Sometimes a symptom of the disease in its initial stage is color perception disorders. Less than 50% of patients with optic neuritis show signs of optic disc inflammation (papillitis); in the rest, at the beginning of the disease, no changes on the part of the optic nerve disc are detected, which indicates the development of a focus of demyelination at some distance behind the disc (retrobulbar neuritis). Subsequently, atrophy of the optic nerve occurs, which is determined by discoloration of the optic nerve disc. It is important to remember that in most cases optic neuritis occurs as an isolated manifestation. 40% of patients with optic neuritis develop PC in the future; unfortunately, it is very difficult to predict which of these patients will develop PC and which will not, but the presence of oligoclonal bands in the CSF seems to be an unfavorable sign. Whether the optic neuritis occurs in isolation or for unknown reasons is an obliterated form of PC with a single exacerbation is unknown. Approximately 33% of patients with optic neuritis recover completely, 33% - partially,

the rest do not recover or it is insignificant. When studying evoked visual potentials, in more than 80% of confirmed cases of PC, an increase in the latency of the evoked potential in the occipital cortex is observed; in less than half of these cases, this change can be considered as a predictor of optic neuritis. Clear subclinical involvement of the visual pathways is very common.

With PC, neurological symptoms due to damage to the brain stem, cerebellum, and spinal cord are common. Diplopia is observed when the fibers of the III, IV or VI cranial nerves are involved in their course in the CNS or in connection with nuclear ophthalmoplegia. Nuclear ophthalmoplegia develops when the posterior longitudinal bundle is damaged and is characterized by the inability to adduct one eye when attempting to make a combined turn of the eyeballs to the side, but at the same time, complete abduction of the other eye and its horizontal nystagmus are observed. Bilateral nuclear ophthalmoplegia in a young adult patient is almost pathognomonic for PC, although there are several case reports of bilateral nuclear ophthalmoplegia in systemic lupus erythematosus. Another clinical sign of brainstem involvement is hypoesthesia in the face and trigeminal neuralgia (V cranial nerve). If trigeminal neuralgia is noted in a young adult, then with all seriousness the question should arise that PC may be at its basis. Bell's palsy and facial hemispasm (VII cranial nerve), dizziness, vomiting and nystagmus (vestibular part of VIII cranial nerve) are also often observed; complaints of deafness are rare. Damage to the connections of the cerebellum or spinocerebellar pathways leads to ataxia, which is accompanied by slurred speech, head swaying, unsteady gait, intentional tremor of the limbs, disorders that occur in the patient while standing and walking. Cerebellar ataxia may be accompanied by sensory ataxia due to involvement of the spinal cord.

Damage to the spinal cord leads to various motor and sensory disorders. Interruption of the fibers of the corticospinal pathways causes the development of classic signs of central motoneuron dysfunction (paresis, increased muscle tone of the spastic type, hyperreflexia, clonus, Babinsky's reflex, loss of abdominal skin reflexes). Damage to the posterior columns of the spinal cord causes a loss or decrease in muscle-joint and vibration sensitivity, as well as frequent complaints of a tingling sensation, tightness in the limbs and a "hoop" around the body. Less often, pain and temperature sensitivity is lost or reduced, which reflects the involvement of spinothalamic pathways. Partial lesions of sensitive pathways and posterior roots at the places of their entry into the spinal cord provoke painful dysesthesias, as well as interruption of reflex arcs. Sometimes spinal foci give paroxysmal symptoms, including tonic spasms, often painful. Common signs of spinal cord involvement include symptoms of bladder dysfunction, including delayed voiding, urgency, urgency, and incontinence. Patients often suffer from intestinal dysfunction, especially constipation. With a targeted survey of men with PC, complaints about decreased sexual function are often found; there are methods of determining its physical and psychogenic causes. Some PC patients experience a shock-like sensation of electrical discharge when the neck is bent, called Lhermitte's symptom. Severe lesions of the spinal cord can lead to loss of functions, sometimes complete, below the level of the lesion; partial lesions may be accompanied by Brown-Sécard syndrome of a half-transverse lesion of the spinal cord. Any of these manifestations is referred to as transverse myelitis. A single episode of transverse myelitis without further progression of the disease can, like an episode of isolated optic neuritis, represent an erased form of PC, although later PC develops in less than 10% of patients with acute transverse myelitis. As with optic neuritis, in approximately 33% of patients with transverse myelitis, complete recovery occurs, in 33% - partial recovery, and in the rest there is no improvement. It should be emphasized that the symptoms of spinal cord injury prevail in most patients with advanced clinical PC. Cerebral symptoms in patients with PC may be due to extensive involvement of subcortical and central white matter. With widespread brain damage, intelligence often suffers. The most common emotional disorder in PC is depression. Euphoria, if it occurs, indicates diffuse brain damage and is often combined with dementia and pseudobulbar palsy. 3-5% of patients (2 times higher than the expected level) have one or more epileptic seizures, presumably due to the spread of plaques to the gray matter. Focal neurological symptoms of cerebral origin, such as hemiparesis, homonymous hemianopsia, and dysphasia, are observed in PC only in rare cases.

Neuromyelitis of the optic nerve and PC.

A pathological symptom complex known as Dewick's syndrome or neuromyelitis optica is considered a condition that should be differentiated from PC. This syndrome is characterized by acute neuritis of the optic nerve, usually bilateral, followed (or less often preceded) by a time interval of several hours to several weeks transverse myelitis. In the CSF, pleocytosis with polymorphonuclear cells is detected, the protein level is slightly higher than is usually the case with PC. Pathological examination in cases with a fatal outcome reveals more pronounced tissue destruction and cavitation than would be expected in PC, but this is probably due to the intensity of the process.

Course of the disease and prognosis. The clinical course of PC is unpredictable. In general, in the case of a disease manifested by acute symptoms of damage to sensitive pathways and cranial nerves, the prognosis is more favorable than in the case when the symptoms develop gradually and are caused by disorders of motor and especially cerebellar functions. According to McAlpine, during the course of the disease with clear exacerbations and remissions, 80% of patients 10 years after the onset of PC do not have functional limitations. In the same cases, when a progressive course is superimposed on exacerbations and remissions, after 10 years 50% of patients become disabled. In cases with a purely progressive course since the onset of the disease (while the spinal cord is mainly affected), the prognosis from the point of view of the ability to move independently is unfavorable. Sometimes PC occurs rapidly (acute PC), and the patient dies a few weeks or months after the onset of the disease. In such cases, an inflammatory reaction is detected in the plaques. The onset of the disease can be accompanied by headache, vomiting, delirium, convulsions and even coma, as well as symptoms that indicate severe disorders of the cortex of the large hemispheres, brain stem, optic nerves and spinal cord. Lifetime differentiation from acute diffuse encephalomyelitis is difficult. Differential diagnosis. The diagnosis of PC becomes certain only when the symptoms of the disease indicate the presence of multiple foci of damage to the white matter of the CNS, remitting at different times. Very often, especially in the early stages of the disease, the existing neurological symptoms can serve as the basis for a diagnostic error, in connection with which it is necessary to exclude other causes of focal damage. A good clinical rule is that PC should not be diagnosed if all symptoms point to a single focus. There is also a common aphorism: PC is manifested by subjective symptoms in one leg, while objective symptoms in both.

The list of conditions that must be excluded during differential diagnosis depends on the localization of the lesions. Sudden blindness in one eye can occur due to a violation of the blood supply in the optic nerve, with embolic or thrombotic occlusion of the carotid, orbital, and central retinal arteries, or as a concomitant symptom of migraine. If the decrease in vision in one eye occurs gradually, then the presence of compression lesions affecting the optic nerve and optic nerve glioma should be suspected.

In patients with acute or progressive lesions of the spinal cord, it is necessary to exclude focal spinal processes and degenerative-metabolic diseases with selective involvement of the leading pathways of the spinal cord. Patients with progressive spastic paraplegia should be examined for possible submucosal or extradural neoplasms and cervical spondylosis, which often requires CT, MRI, and myelography. Hereditary ataxias can manifest as degenerations of many leading pathways of the CNS with or without involvement of the peripheral nervous system. In these conditions, degeneration of the posterior columns, cortico-spinal and spinocerebellar tracts is often observed. Hereditary ataxias are characterized by a slowly progressive course with a stereotypical symmetrical distribution of neurological symptoms; the disease is inherited in an autosomal dominant or autosomal recessive type. Amyotrophic lateral sclerosis (ALS) is usually manifested by severe symptoms of peripheral motor neuron damage (atrophy, weakness, and fasciculations) in the presence of pyramidal symptoms (spasticity, hyperreflexia), and sensitivity disorders are not observed. Subacute combined degenerations of the spinal cord can be excluded on the basis of symmetrical severity of symptoms; the content of vitamin B12 in the blood serum, the picture of the bone marrow and the results of the Schilling test in this case are normal. With

increasing trunk symptoms, it is necessary to exclude tumors of the posterior cranial fossa and trunk encephalitis. Isolated cranial nerve palsies, especially Bell's palsy, sensory neuropathy of the trigeminal nerve or its neuralgia can be included in the clinical picture of PC, but to be sure of this, it is first necessary to establish that the lesion is multifocal. If the patient complains of dizziness and nystagmus is detected in him, then one should assume a disease of the inner ear and a possible side effect of taking barbiturates and phenytoin.

There are several multifocal and relapsing CNS diseases that can mimic PC. Thus, with systemic lupus erythematosus and other vasculitis, diffuse and recurrent focal lesions of the brain, trunk, and spinal cord also develop. Behcet's disease is characterized by repeated episodes of focal brain damage, pleocytosis, uveitis, ulcers on the mucous membrane of the oral cavity and genitals. Diseases such as meningovascular syphilis, cryptococcosis, toxoplasmosis, other chronic infections of the nervous system, and sarcoidosis should also be considered in the differential diagnosis. In cases where the complaints are vague and the objective examination gives minimal results, the thought of conversion reactions (hysteria) arises. This diagnosis should always be based on positive criteria for hysteria, but it cannot be made by the exclusion method. In the early stages of PC, hysteria is misdiagnosed with a depressingly high frequency. At the same time, hysterical phenomena can be superimposed on the picture of PC, thereby increasing the complexity of interpreting the clinical syndrome. In some patients, the leading symptom is pain. A physician must know the specifics of PC pain, and a thorough, careful examination usually clarifies the diagnosis. A reliable diagnosis of PC can only be made if there are clear signs of the disease. In addition to the distress caused by this diagnosis, it can serve as a basis for explaining any subsequent changes in neurological status and divert attention from other possible diseases that are amenable to treatment.

Results of laboratory studies. Although the diagnosis of PC is based on clinical signs, laboratory tests are becoming increasingly important to confirm the diagnosis. In the vast majority of patients with PC, at least one parameter of laboratory studies has changed. However, the diagnosis of PC cannot be excluded when normal results are obtained. In the study of CMP, slight pleocytosis is sometimes detected. The cells are mainly represented by T-lymphocytes, but sometimes plasma cells are also detected. There is a certain correlation between the expressiveness of pleocytosis and the activity of the disease. Increased cytosis is also more characteristic of the early stages of the disease. Data were obtained that lymphocytes in the CSF are activated not only during exacerbations, but also during periods of apparent remissions, which indicates the constant "smoldering" activity of the disease, even when both the doctor and the patient are unable to detect any changes . PC is characterized by an increase in the content of IgG in CMP, which contrasts with relatively normal concentrations of total protein and albumin. IgG levels are increased in 80% of patients with PC, their increase is maximally expressed in people with a long history of the disease and a severe neurological defect. In the early stages, while the diagnosis remains doubtful, the IgG content may be within the normal range. During electrophoresis, IgG CMP of PC patients is divided into fractions in the form of a limited number of bands (called oligoclonal bands). During periods of PC exacerbations, myelin breakdown products and myelin basic protein appear in the CSF.

Conduction of nerve impulses along axons that have lost their myelin sheaths slows down. It is possible to determine the slowing down of the conduction of visual, auditory and somatosensory impulses using the method of evoked potentials. In the course of these tests, repeated sensory stimulation is carried out and the technique of computer averaging is used in order to register the electrical reactions caused in the process of carrying out these stimuli along the visual, auditory and somatosensory afferent pathways. The study of evoked potentials makes it possible to confirm the presence of additional lesions in suspected cases of PC in which there is only one clinically visible lesion.

At CT of the brain in the white matter in 10-50% of patients, foci of low density are found, localized paraventricularly or subcortically and corresponding to both active and chronic lesions (according to pathological criteria). Similar lesions are found in the optic nerves and brain stem.

The image can be enhanced by the introduction of iodine-containing contrast material, especially when using high doses of it and slow scanning. This indicates the presence of acute lesions and disruption of the blood-brain barrier. Enhancement may disappear with regression of clinical symptoms. Some patients show cortical atrophy with expansion of the ventricles.

MRI is an even more sensitive method for detecting lesions. MRI visualizes more pathological areas than CT.

An increase in the content of IgG in CMP, studies of evoked potentials, visualization of pathological foci at CT and NMR serve as important additional information obtained during the examination of patients with suspected PC; however, the primary importance in making a diagnosis belongs to clinical data.

Treatment for PC. There is no effective treatment for PC. Therapy is aimed at reducing the severity of exacerbations, preventing exacerbations and reducing the severity of symptoms of the disease.

During acute attacks of the disease, glucocorticoids contribute to reducing the severity of symptoms and speeding up recovery; this, however, does not lead to recovery and does not affect the degree of disability. Glucocorticoids improve conduction along demyelinated nerves, reduce swelling and inflammation inside plaques. Long-term courses of steroids taken daily or every other day are not recommended.

The results of several clinical trials suggest that immunosuppressants such as azathioprine help reduce the number of exacerbations, but there is no consensus on the effectiveness of this drug. The results of clinical studies of plasmapheresis and interferon are ambiguous. Symptomatic treatment should satisfy both physical and psychological needs of patients. Patients should avoid excessive fatigue, extreme temperature effects, and follow a balanced diet. Diets low in saturated fat are recommended. In case of bladder dysfunction, it is advisable to use belladonna alkaloids. Periodically, it is necessary to carry out tests to rule out urinary tract infection. Drugs used for spasticity include diazepam, baclofen, and sodium dantrolene. Carbamazepine and gabapentin are recommended for painful dysesthesias, twitching of facial muscles, neuralgia of the trigeminal nerve and tonic spasms.

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 occurs 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 micronecrosis and foci of demyelination 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. 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 pronounced movement and other disorders remain. Remissions are not observed.

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

Subacute sclerosing encephalitis (demyelinating leuko-and panencephalitis) - Peculiar forms of chronic and subacute encephalitis with a progressive, severe course (encephalitis with Dawson's inclusions, subacute sclerosing leukoencephalitis of Van-Bogart, nodular Pete-Dering panencephalitis, subacute sclerosing panencephalitis of Tarishka). Since the differences between

them in the clinical picture and morphology are relative and insignificant, they are currently treated as one disease, most often under the name "subacute sclerosing panencephalitis". This group of diseases also includes Schilder's periaxial encephalitis (diffuse periaxial sclerosis), which, however, has some well-defined clinical and pathomorphological features. Subacute sclerosing panencephalitis is more common in children who had measles before the age of two. Subacute sclerosing panencephalitis develops several years after measles and usually leads to dementia within a few months. It is characterized by a very high titer of anticortical antibodies in serum and cerebrospinal fluid. Apparently, this disease is the result of the macroorganism's reaction to the impaired replication of the measles virus in the brain. In the United States, less than 10 cases of this rare disease are registered annually. Morbidity decreased dramatically after the introduction of vaccination. The anamnesis in most cases is typical: measles in early childhood (up to 2 years), latent period for 6-8 years, then increasing neurological disorders; in 85% of cases, the diagnosis is made at the age of 5-15 years.

In the etiology of subacute sclerosing encephalitis, a persistent viral infection, probably a cow, plays a major role. In patients with subacute sclerosing panencephalitis, very high titers of cortical antibodies are found in the blood and cerebrospinal fluid. A specific measles immunoglobulin characteristic of the current measles infection was also detected. The measles virus in the brain is in a suppressed state. Some researchers managed to isolate the measles virus from the brain tissue of deceased patients. Autoimmune mechanisms play a role in the pathogenesis of the disease, as well as an acquired or congenital defect of the immune system.

Pathogenesis.

In the pathogenesis of subacute sclerosing panencephalitis, two main factors are important - persistence of the virus in the CNS and impaired immunological reactivity. Microscopically, pronounced diffuse demyelination and gliosis of the white matter of the cerebral hemispheres are revealed. In some cases, there are many glial nodules. In other cases, oxyphilic inclusions are found in the nuclei of neurons of the cortex, subcortex, and brainstem against the background of their 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 the growth of glia with foci of sclerosis.

Symptoms of subacute sclerosing panencephalitis:

The disease mainly affects children and adolescents aged 2 to 15 years, but sometimes the disease also occurs in adulthood. The onset of the disease is subacute, imperceptible. Symptoms that are considered neurasthenic appear: inattention, irritability, fatigue, tearfulness. Then there are signs of personality change, deviations in behavior. Patients become indifferent, lose a sense of distance, friendship, duty, correctness of relationships, discipline. Primitive drives begin to dominate: greed, selfishness, cruelty. At the same time, slowly increasing disorders of higher brain functions (agraphia, aphasia, alexia, apraxia), spatial orientation, and disorders of the body scheme appear. After 2-3 months. from the onset of the disease, hyperkinesias in the form of myoclonia, torsional spasm, and hemibalism are detected in the neurological status. At this time, convulsive epileptic seizures, small epileptic seizures, permanent partial convulsive seizures of the Kozhevnikov epilepsy type occur. In the future, as the disease progresses, hyperkinesis weakens, but the phenomena of parkinsonism and dystonic disorders begin to increase, up to decerebration rigidity. Extrapyramidal disorders are usually combined with pronounced vegetative disorders - facial oiliness, drooling, hyperhidrosis, vasomotor lability, tachycardia, tachypnea. Involuntary laughter and crying, sudden screams ("cry of a seagull") are often observed. A frequent symptom is static locomotor ataxia of frontal origin (the patient does not keep the body in a vertical position). In the late stage of the disease, mono-, hemi-, and tetraparesis of a spastic character appear, which are superimposed on extrapyramidal and frontocerebellar motor disorders. Sensory and motor aphasia, auditory and visual agnosia are revealed. Cachexia progresses.

Course and forecast:

Four stages are distinguished during subacute sclerosing encephalitis.

- In the 1st stage, the leading symptoms are personality changes, deviations in behavior, increasing defects of higher brain functions, various hyperkinesis, convulsive and non-convulsive seizures. This I stage (psychotic) lasts from 1 to 12 months.

- During the 2nd stage, extrapyramidal disturbances of tone and disorders of autonomic central regulation increase; Violations of muscle tone often have a mixed spasticity-rigid character, hyperkinesis (athetosis, myoclonus, tremors) is often noted. Subcortical disorders in the initial period develop according to mono- or hemitype, but later spread to both arms and legs. Symptoms of damage to the pyramidal pathways in panencephalitis appear against the background of an already developed picture of the disease. Frequent symptoms also include static and locomotor ataxia (due to damage to the cerebellum or frontal lobe of the cortex). Typical and permanent signs of the disease include epileptic seizures in the form of generalized seizures, simple and complex absences, twilight states and automatisms. This stage usually lasts from 6 months to 1 year.

- the 3rd stage is characterized by a sharp increase in muscle stiffness, the appearance of torsion spasms, myogenic contractures. Long-term tonic convulsions of the decerebrate type are noted in patients, and mental retardation is increasing. Due to increased rigidity, clonic convulsions and hyperkinesis weaken. The duration of the III stage is several months.

- The last (comatose) stage of subacute sclerosing encephalitis is characterized by a complete loss of mental functions, persistent decerebration rigidity, hyperthermic crises with fever of the hectic type, cachexia. The fatal outcome occurs with the development of circulatory collapse or intercurrent disease. The course of sclerosing encephalitis progresses steadily and always ends fatally. The duration of the disease is usually from 6 months. up to 2-3 years. There are forms that occur chronically with periodic remissions.

The clinical picture of Schilder's leukoencephalitis has some features: pyramidal symptoms are more pronounced, dominating extrapyramidal symptoms, large epileptic seizures are more often noted. In the initial stages, mental disorders prevail. A course in the form of a pseudotumorous form with signs of increasing single-focal hemispheric symptoms accompanied by intracranial hypertension is possible. Damage to the cranial nerves, especially the II and VIII pairs, is characteristic. Amblyopia up to amaurosis is possible. Atrophy of the optic nerve discs is found on the fundus. In some cases, with amaurosis, pupillary reactions to light remain preserved, which is due to the central character (due to damage to the occipital lobe) of amaurosis. In the cerebrospinal fluid, moderate pleocytosis, increased protein content and gamma-globulin level are detected. Extremely high titers of cortical antibodies are determined in blood serum and cerebrospinal fluid. Periodic stereotypical regular bilaterally synchronous high-amplitude discharges of electrical activity are recorded on the EEG. Diagnosis of subacute sclerosing panencephalitis: There are some difficulties in the early stage, when neurasthenia, hysteria, schizophrenia are often diagnosed. In the future, a differential diagnosis is made with a brain tumor. The diagnosis should be based on the diffuseness of the lesion, the absence of intracranial hypertension, the displacement of the middle structures of the brain during echoencephalography, MRI, and the pathognomonic pattern of the EEG.

Treatment of subacute sclerosing panencephalitis: pathogenetic methods of therapy include the use of glucocorticoids, but quite often this treatment is ineffective. Barbiturates (phenobarbital, benzonal, hexamidine) are used to stop seizures in combination with carbamazepine (finlepsin, tegretol) or valproic acid derivatives (depakine, convulex) in age-appropriate doses. Midokalm, baclofen (lioresal) are used to reduce muscle tone. Prescribe vasodilators (cavinton, sermion), nootropil, group B vitamins, in some cases anabolic hormones (nerabol, retabolil), tonics. There is evidence that inosine pranobex can sometimes prolong life and improve the condition, but they are contradictory.

Materials for self-control of training quality

Tests

1. What symptoms make up Charcot's triad?

A. Nystagmus
B. Hemiparesis
V. Scanned language
D. Intentional tremor
D. Ataxia Answer: A, B, G.

2. What symptoms make up the Marburg triad?

A. Nystagmus
B. Absence of intestinal reflexes
B. Discoloration of the temporal halves of the discs of the optic nerves
D. Spastic paraparesis
D. Scanned language Answer: B, B, G.

Tasks for self-control :

A 25-year-old patient, at the age of 19 she first felt a decrease in vision in her right eye. After a month, the vision improved. A year later, the patient developed ataxia, a feeling of heaviness in her legs.

Objectively: horizontal nystagmus, tendon and periosteal reflexes are increased, more on the legs. Abdominal reflexes are absent; bilateral pathological foot reflexes of Babinski, Rossolimo, Zhukovsky are caused. Muscle tone in the legs is increased. In the Romberg pose - swaying. When performing PNP - intentional tremor on both sides. The gait is spastic-atactic. During ophthalmoscopy, bilateral bitemporal discoloration of optic nerve discs.

Question:

A. Name the diagnosis of the disease?
B. Where is the lesion located?

Answers:

1. Multiple sclerosis, cerebro-spinal form.
2. The pathological process is localized in the cerebellum and spinal cord.

Materials for classroom self-training:

List of educational practical tasks:

1. Check for nystagmus.
2. Check for paralysis (central and peripheral).
3. Check for cerebellar symptoms.
4. Check for trophic disorders.
5. Check for symptoms of irritation of the anterior horns of the spinal cord.
6. Check the patient's progress.
7. Check for bulbar paralysis.
8. Check the patient's language.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

Electronic information resources

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

Practical lesson No. 26

Topic: Structure and functions of the peripheral nervous system. Symptoms of nerve tension.

The goal: to acquaint students with the structure of the peripheral nervous system, to focus their attention on the main functions of this system, the damage of which leads to long-term incapacity, and sometimes even disability.

Basic concepts: diseases of the peripheral nervous system occupy one of the leading places among neurological diseases and lead to long-term disability of patients. Therefore, timely diagnosis and correctly prescribed therapy are of great importance both for the economy of the country and for the rapid improvement of the health of patients.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

The structure of the peripheral nervous system.

The peripheral nervous system is a part of the nervous system. It is located outside the brain and spinal cord, provides two-way communication between the central parts of the nervous system and the organs and systems of the body.

The peripheral nervous system includes cranial and spinal nerves, sensitive nodes of cranial and spinal nerves, nodes (ganglia) and nerves of the autonomic (autonomic) nervous system and, in addition, a number of elements of the nervous system, with the help of which external and internal stimuli are perceived (receptors and effectors).

Nerves are formed by processes of nerve cells, the bodies of which lie within the boundaries of the brain and spinal cord, as well as in nerve nodes of the peripheral nervous system. Externally, the nerves are covered with a loose connective tissue sheath - the epineurium. In turn, the nerve consists of bundles of nerve fibers covered with a thin membrane - perineurium, and each nerve fiber - endoneurium.

Peripheral nerves can be different in length and thickness. The longest cranial nerve is the vagus nerve. It is known that the peripheral nervous system connects the brain and spinal cord with other systems with the help of two types of nerve fibers - centripetal and centrifugal. The first group of fibers conducts impulses from the periphery to the CNS and is called sensitive (efferent) nerve fibers, the second carries impulses from the CNS to the innervated organ - these are motor (afferent) nerve fibers.

Spinal nerves depart from the spinal cord and branch out in the trunk, neck and limbs. These nerves are mixed: they consist of centripetal and centrifugal fibers. According to the first, excitation from receptors enters the spinal cord, and according to the second, excitation is carried out from the spinal cord to various organs.

Cranial nerves originate from the brain and spread in the area of the head and neck, only one of them branches in the organs of the body cavity.

Nerve nodes are clusters of interconnected neuron bodies. Nodes are located inside or around various organs. Two long chains of such nodes stretch on both sides of the spine. The largest nodes that form the solar plexus lie in the abdominal cavity.

Centrifugal fibers of cranial and spinal nerves are divided into motor and vegetative.

Motor nerve fibers start from the motor centers of the spinal cord and brain and end in skeletal muscles. These fibers conduct excitation, which causes contraction of skeletal muscles.

Autonomic nerve fibers terminate in all organs of the body. On their way, these fibers, unlike motor fibers, pass through nerve nodes that lie in the organs or near them. In nodes, excitation is transmitted from one neuron to another.

Vegetative fibers conduct excitation, which strengthens or weakens the activity of organs. These fibers are also present in skeletal muscle . The excitation that passes through them cannot cause muscle contraction, but, increasing their metabolism, increases their work.

Usually, two autonomic nerves approach the organ, which act on it oppositely. So, for example, vasodilator and vasoconstrictor nerves approach vessels. Irritation of the first increases the nutrition of the organ and strengthens its activity, irritation of the second reduces the nutrition of the organ and weakens its work. Two nerves also branch in the heart. Irritation of one of them increases the number and force of heart contractions, irritation of the second decreases the number of contractions and weakens each of them. Such double innervation of organs ensures very precise

regulation of their activity, because the action of one nerve is under the restraining influence of the other.

The absolute majority of peripheral nerve fibers are myelinated and all fibers are covered with a sheath of Schwann cells. Two mechanisms ensure the function of the fibers - the electrical transmission of impulses due to the myelin sheath and the axonal transport of active substances, including mediators to effector organs (muscles, skin, glands, internal organs). Peripheral nerves are protected by three sheaths: epineurium, perineurium, and endoneurium. It is in the subendoneurial space that nerve fibers come into contact with the capillaries of the vascular system, and in this space most pathological events that damage peripheral nerves take place. There are four main mechanisms of damage – Valerian degeneration (of course, with mechanical nerve damage), segmental demyelination, axonal degeneration and neuronopathy. In Valerian degeneration, fragmentation of the axon and myelin sheath occurs in the segment of the nerve distal to the damage, and later only a strip of Schwann cells remains, into which the regenerating axon grows and thus slowly (1 mm/day) the integrity of the nerve is restored. In segmental demyelination due to an autoimmune conflict, the myelin sheath is lost in certain areas of the nerve fiber and myelin can subsequently be restored with a corresponding restoration of the normal speed of nerve impulse conduction and function. Axonal degeneration occurs as a result of metabolic disturbances in the entire nerve (for example, accumulation of sorbitol and inositol in diabetes, thiamine deficiency in alcoholism, toxic effects of heavy metals, etc.). The speed of conduction almost does not suffer, but the number of impulses reaching the working organ is sharply reduced (clinical manifestations prevail in the distal parts of the limbs). Neuronopathy is a specific type of lesion, when the bodies of the neurons themselves are affected first, and their processes are affected secondarily. An example of a benign neuronopathy is a herpetic lesion of sensitive ganglia (Herpes zoster), and a malignant, irreversible lesion of the cells of the anterior horns as part of a paraneoplastic process, with malignant neoplasms of somatic organs. All types of damage to PNS formations are called neuropathy. In the clinical sense, the terms neuralgia and neuritis are often used. These two types of damage may have the same etiology and mechanisms of damage, but in neuralgia, the pathological process usually does not reach the degree of morphological damage to the myelin sheath or axial cylinder, and therefore the clinical symptoms of irritation prevail - pain (usually throbbing, sharp, short) and hyperesthesia in the area of innervation of the corresponding nerve, its tenderness during palpation. With neuritis, there are signs of damage to the integrity of fibers (axons and/or myelin sheath), which is manifested by existing symptoms of loss of function: flaccid muscle paresis, areas of hypo- or anesthesia, a decrease in the corresponding reflexes. At the same time, pain may be present, as with neuralgia. This is the difference in the essence of two clinical terms. When formulating the diagnosis, the predominant site of the lesion is often indicated: radiculitis (root level), plexitis (nerve plexus level), ganglionitis (in case of damage to the corresponding ganglion) or neuritis. Lesions of the PNS are also classified into mononeuropathy (one nerve), multiple mononeuropathy (several nerves in one or several different places) and polyneuropathy (when the pathological process affects all nerves).

Methods of studying the peripheral nervous system.

When peripheral nerves are damaged, patients present two main complaints - pain or impaired movement. Since almost all peripheral nerves are mostly mixed, when any of them is damaged, the patient feels pain. Irritation of the mixed nerve is called neuralgia. The pain is localized in the innervation zone of the affected nerve. In order to objectify the patient's complaints and differentiate the nerve damage, it is necessary to palpate the points that correspond to the anatomical course of the nerve, as well as to check the symptoms of stretching of those nerves that can be stretched. At the same time, the pain intensifies.

Brachial plexus.

When the brachial plexus is damaged, the patient complains of pain in the arm. To objectify the pain, research begins with palpation of points along the course of its main branches (Yerba points) - in the furrow along the medial surface of the shoulder, on the forearm between the ulna and radius bones, in the first interdigital space, above and subclavian points. Then the

muscle strength in the hand, muscle tone and their trophicity are determined, which may decrease or fall out in the case of an organic lesion of the plexus. Carpo-radial, flexor and extensor elbow reflexes, which may decrease or fall out, are studied.

When examining sensitivity, a decrease or loss of surface sensitivity is revealed on the entire arm and part of the shoulder girdle, and on the hand, of course, it increases more sharply than in the proximal parts. This is the so-called "plexal type" of sensitivity disturbance. When the brachial plexus is damaged, for example, with a fracture of the clavicle or a prolapsed shoulder, a peripheral atrophic flaccid paralysis of the hand develops with all the resulting symptoms. Moreover, a typical plexus-type violation of all types of sensitivity is found on the paralyzed limb.

Lumbar roots and sciatic nerve.

Damage to the roots is called radiculitis, irritation radiculalgia. Damage to any nerve is neuritis, irritation is neuralgia. The main symptom of root and nerve damage is pain in the affected area. To objectify pain complaints, it is necessary to palpate root zones or points along the course of the sciatic nerve. Root zones are detected on 2 - 3 смto the right and to the left of spiny processes. The points of the course of the sciatic nerve (Vale points) correspond to the anatomical course of the nerve: the root zone of 1 and 2 sacral roots, the area of the sacroiliac joint, the middle of the sciatic fold, in the popliteal fossa, lateral to the medial process of the ankle, the medio-plantar point. When pain is detected in these points, one phrase can be written in the medical history: there is pain in Vala points (right or left). Then we begin to study the symptoms of tension.

Laseg's symptom - the examiner raises the patient's leg, which is bent at the knee joint, when the patient is lying on his back. At the same time, the sciatic nerve and lumbosacral roots are stretched. The patient feels increased pain (1st phase), then the doctor bends the leg in the knee joint - the tension is relieved. The pain disappears or is significantly mixed. If the first phase is painful, and the second (relief of tension) is the opposite, it is said to be a positive Laseg symptom.

Brogard's symptom - 1) in a patient lying on his back, the leg, which is bent at the knee joint, should be slightly raised. 2) Then bend the foot to the back. At the same time, there is pain along the course of the sciatic nerve.

Symptom "landing" - a patient lying on his back is asked to sit in bed without lowering his legs. If the patient is unable to sit down without bending the legs in the knee joints, the positive symptom is "sitting".

Bekhterev's symptom - the patient lies on his back. The examiner raises the healthy leg to the mountain as when checking the Laseg symptom. If the lumbosacral roots are affected, there is pain in the lower back, from the side of the affected part.

Neri's symptom - the patient lies on his back. The researcher, holding his hand under the back of the patient's head, bends the patient's head with a sharp movement. When the extremities are damaged, the patient feels pain in the place of damage. This symptom is checked for cervical, thoracic and lumbosacral sciatica.

It should be remembered that tension symptoms will be positive in radiculalgias and neuralgias, serve to objectify the patient's complaints and differentiate peripheral nervous system lesions from muscle, joint, and bone lesions when they are negative.

After establishing a lesion of the lumbosacral roots or the sciatic nerve, they begin the study of muscle trophicity , knee and Achilles reflexes, and superficial sensitivity.

With neuritis of the sciatic nerve, the trophism of the calf muscles first suffers, then the back surface of the thigh and buttock (the muscles become flabby), the reflex from the Achilles tendon is lost, the sensitivity on the outer surface of the thigh, lower leg and back of the foot is reduced. With neuralgia, pain in Vala points does not have positive symptoms of tension - Laseg, Brogard, "landing".

Femoral nerve. When the femoral nerve is damaged, the pain is localized on the front surface of the thigh. They begin to check for symptoms of tension. In order to stretch the femoral

nerve, the patient is asked to lie on his stomach. To c, the recumbent person lifts up the patient's straightened leg, the pain on the front surface of the thigh intensifies - Wasserman's symptom. If the patient lying on his stomach bends his leg at the knee joint, pain also occurs on the front surface of the thigh - Matskevich's symptom.

Then check the trophicity of the quadriceps muscle , knee reflex and sensitivity on the front surface of the thigh. If the patient shows hypotrophy of the quadriceps muscle of the thigh, the knee reflex decreases or disappears, hypoalgesia of the front of the thigh is detected, it is said to be neuritis. If, in addition to pain and positive tension symptoms, there are no other phenomena, it is about femoral neuralgia

Materials for self-control of training quality

1. The peripheral nervous system includes the listed structures, with the exception of:

A. Korintsi
B. Nerves
V. Plexus
G. Anterior horns of the spinal cord
D. Ganglia

Answer: G

2. All the listed nerves are motor, with the exception of:

A. Okorukhoi
B. Face
V. Blokovy
G. Zorovy
D. Additional

Answer: G

1. Radiculitis is a lesion of which part of the nervous system?
2. Plexitis is a lesion of which department of the peripheral nervous system?

A. Korynets
B. Spinal ganglion
V. Plexus
D. Anterior horn of the spinal cord
D. Distal departments of peripheral nerves

Answer: 1-A
2-V

1. How does the muscle tone change when the roots are damaged?
2. How does muscle tone change with nerve damage?

A. hypertension
B. hypotension
B. "gear wheel" symptom
G. the symptom of a "folding knife"

Answers: 1 - B
2 – B

1. What reflexes disappear when the sciatic nerve is damaged?
2. What reflexes disappear when the lumbosacral plexus is damaged?

A. knee
B. Achilles
V. plantar
G. cremasterny
D. stomach

Answers: 1 - B
2 - A, B, C.

Tasks for self-control :
Patient D., 18 years old, motorcyclist. Six months ago, he suffered a fracture of the right clavicle. Severe pain developed and decreased movement in the right arm. The hand began to lose weight. Objectively: cranial nerves without pathology. Active movements in the right hand are limited; passive in full. Muscle strength is reduced to 2 points. Muscle tone is low. Tendon and periosteal reflexes on the hands on the left are lively, on the right they are not evoked. The volume of the right shoulder is 33 см, the left shoulder is 37 см. Sensitivity on the right hand, forearm and shoulder is reduced.

1. What type of sensitivity disorder does the patient have?
2. What are the rules of sensitivity research?
3. What is the diagnosis?

Answers:

1. Plexiglas type.
2. From top to bottom, in symmetrical parts of the body and face, on the limbs in a circle.
3. Right-sided traumatic shoulder plexitis.

Materials for classroom self-training:
List of educational practical tasks:
1. Investigate the Laseg symptom.
2. Investigate Neri's symptom.
3. Investigate the "landing" symptom.
4. Investigate Brogard's symptom.
5. Investigate Bekhterev's symptom.

Instructional material for mastering professional skills
1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

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Practical lesson No. 27

Topic: Diseases of the peripheral nervous system

Purpose: to create an idea among the applicants about the symptoms of damage to the lumbar sacral roots, sciatic and femoral nerves; symptoms of damage to the ulnar, radial and median nerves; etiology and clinical course of polyneuritis; paraclinical research, treatment and prevention of diseases of the peripheral nervous system.

Basic concepts: Among the diseases of the nervous system, damage to its peripheral department occurs most often. Unlike the nerves of the central N.S. , peripheral nerves do not have strong bone protection and a blood-brain barrier. They are damaged over a considerable length and show increased sensitivity to the action of various exogenous and endogenous factors.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

Graph of the logical structure of the lesson

Diseases of the peripheral nervous system

I. Nosological forms — Mononeuritis (neuralgia); Polyneuritis (polyneuropathy); Plexites (plexopathy)

II. Etiology of Trauma

Common infectious diseases are acute and chronic
Intoxications - exogenous and endogenous
Avitaminosis

Allergy

Hypothermia

III. Clinical Pain

signs of Paresthesia

Violation of peripheral sensitivity

type (hypesthesia, hyperesthesia, etc.)
Vegetative and vascular disorders

IV. Principles of medical treatment

Orthopedic treatment

Surgical

Physiotherapy

Sanatorium-resort

Materials for self-control of training quality

Tests

A. Duchenne-Erb upper plexitis shows the following symptoms:
a) flexion-elbow reflex is lost.
b) the elbow flexion reflex increases.
c) there is an elbow extensor reflex
d) the elbow extensor reflex increases.
e) reflexes do not change. The answer is a)

B. With neuritis of the femoral nerve, tension symptoms appear:
a) Lasega
b) Neri
c) Brogard
d) Wasserman
d) Sokolyanskyi Answer - d)

A) Name the type of sensitivity disorder in plexitis.
B) Name the type of sensitivity disorder in polyneuritis:
a) root
b) conductive
c) plexal
d) mononeural
e) by type of stockings and gloves

Answers

A. - v
B. - b

When the peripheral nervous system is damaged, symptoms are observed:
a) muscle hypertrophy
b) hyperreflexia
c) muscle hypotonia
d) pathological reflexes
e) hypotrophy
Answer: v.d.

Tasks for self-control :
Patient N., 24 years old, drank almost 300 ml in the evening. of vodka, fell asleep with his right hand at his side. Waking up in the morning, he noticed that his hand was not moving. Objectively: the hand in the elbow joint does not extend, the fingers of the bone do not extend, supination of the bone is not possible, the bone "hangs", the thumb cannot be opposed to others.
1. Name the diagnosis of the disease.
2. Prescribe treatment.

Answer:
1. Intoxication-traumatic neuritis of the right radial nerve.
2. Proserin, nicotinic acid, dibazole, vitamins B1, B12, furosemide, massage, HRT, myoton.

Materials for classroom self-training:
List of educational practical tasks:

1 Be able to research:
a) brachial plexus functions;
b) symptoms of sciatic and femoral nerve tension;
c) symptoms of sciatic nerve damage;
d) symptoms of femoral nerve damage;
e) symptoms of damage to the ulnar, radial and median nerves;
2. Assign the necessary examinations and treatment to the thematic patients.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

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Practical lesson No. 28

Topic: Somatoneurological syndromes.

The goal: to create in the students an idea about the pathomorphology of damage to the nervous system in diseases of internal organs and systems, the main somatoneurological syndromes, diagnosis, treatment and prevention of asthenic and polyneuropathic syndromes, neuromuscular disorders, vegetative dystonia, etc.

Basic concepts: the nervous system is a connecting link of the entire organism as a whole. The basis of research on the study of somatoneurological and neurosomatic disorders is based on the works of I.I. Sechenova, I.P. Pavlova, S.P. Botkin and other scientists who proved the existence of cortico-visceral and viscero-cortical connections. In the clinic, both neuropsychiatric disorders in somatic diseases and somatic disorders in diseases of the brain and spinal cord are observed. Therefore, the ability to diagnose such disorders is of great importance both for the proper treatment of patients and for the prevention of further long-term disability of the population.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Lesson content

The pathogenesis of damage to the nervous system in somatic diseases is caused mainly by metabolic, toxic, vascular and reflex disorders. A shift in homeostasis resulting from a violation of protein, carbohydrate, fat metabolism, hypoxemia and tissue hypoxia, accumulation of various waste products, has a toxic effect on nervous tissue (neurons and glial cells, synapses, axons). The first signs indicating the involvement of the nervous system in somatic diseases are increased fatigue, irritability, headache, sleep disturbances, paresthesias and dysesthesias in the Zakharyin-Ged zones. All these symptoms are initially expressed weakly and not constantly. In the future, if the dysfunction of this or that organ or endocrine gland grows, then organic lesions may gradually develop - nystagmus, symptoms of oral automatism, changes in reflexes, motor and sensory disorders. Sometimes an acute disease of an internal organ (thromboembolism of the pulmonary artery, pancreatitis, etc.) debuts with neuropsychological disorders: excitement, motor activity, hallucinations, meningeal manifestations, etc.

Diseases of the heart and main vessels (congenital and acquired defects, myocardial infarction, heart rhythm disorders, septic endocarditis, etc.).

The pathogenesis of these diseases is due to changes in blood and fluid circulation, vessel occlusion, embolisms, and sometimes the spread of the inflammatory process to the vessels of the brain (with vasculitis, septic endocarditis). The initial period of the disease of these organs is most often characterized by asthenic vegetative-vascular disorders - general weakness, instability of pulse and blood pressure, cephalic syndrome. The symptomatology of congenital heart defects manifests itself in childhood, while there is a lag in physical and mental development, syncopal

states and epileptiform seizures, paresis. An extensive myocardial infarction can be complicated by various disorders of cerebral circulation (drowsiness, excitability, headache, meningeal symptoms), which can turn into a cardiocerebral syndrome (dizziness, loss of consciousness, motor and sensory disorders, pathological reflexes) and a cardiospinal syndrome (weakness in the limbs, changes tendon and periosteal reflexes, conductive and segmental disorders of sensitivity). Changes in heart rhythm (paroxysmal tachycardia, atrial fibrillation, bradycardia) are a frequent cause of fainting. Syncopal states occur most often with atrioventricular blockade (Morganhi-Adams-Stokes syndrome) on the background of slowing down the pulse to 30-10 beats/min. The cerebral form of obliterating thromboangiitis is characterized by simultaneous damage to the vessels of the brain, limbs and internal organs, which is manifested by symptoms of dyscirculatory encephalopathy and repeated transient ischemic attacks. With septic endocarditis, as a result of infected emboli entering the vessels of the meninges, the development of purulent meningitis and brain abscess is possible. Chronic obliteration of the abdominal aorta, bifurcation of the aorta and main vessels of the lower extremities is manifested by the gradual development of symptoms of dyscirculatory myelopathy. At the same time, vegetative-trophic, sensitive and motor disorders are especially pronounced in the distal parts of the limbs.

Lung disease.

Neurological disorders can develop against the background of both acute lung diseases (thromboembolism of the pulmonary artery, infarct and severe bilateral pneumonia), and chronic non-specific lung diseases (emphysema, chronic bronchitis, bronchial asthma, pneumosclerosis). In the pathogenesis, the leading role is played by the influence of hypercapnia, hypoxemia, a drop in blood pressure in the large blood circulation, vascular spasm, and disruption of homeostasis resulting from disorders of ventilation and gas exchange of the lungs. All this leads to a higher frequency and variety of focal lesions of the brain than with other types of hypoxia. In uncomplicated pneumonia, mild headache, photophobia, and general hyperesthesia are observed. With severe pneumonias, neuropsychological complications are added, accompanied by general brain, membrane, focal and autonomic symptoms. Extremely polymorphic clinical picture of pulmonary embolism. It can be manifested by the following neurological syndromes – psychomotor agitation (most often), meningeal, focal, epileptiform. Chronic lung lesions are more often manifested by symptoms of chronic encephalopathy, which is manifested by headache, increased fatigue, irritability, minor focal symptoms.

Liver disease (cholecystitis, gallstone disease, mechanical jaundice, liver cirrhosis).

Asthenic manifestations and diffuse damage to the brain and spinal cord, and sometimes to individual peripheral nerves, that is, syndromes of encephalopathy, encephalomyelopathy and polyradiculoneuropathy, are most often observed. The pathogenesis of the lesion is due to violations of the detoxification function of the liver and various types of metabolism - protein, lipid, carbohydrate, water-electrolyte, vitamin. Changes in the coagulation properties of blood and the development of hemorrhagic syndrome develop. The acid-base state of the blood shifts towards acidosis. With jaundice, the accumulation of bilirubin and bile acids, ammonia in the blood has a toxic effect. Neurasthenic syndrome manifests itself in two variants - hypersthenic (irritability, emotional lability) and asthenic (fatigue, irritability). Hepatogenic encephalopathy is manifested by polymorphic neurological symptoms with frequent psychomotor agitation, as well as hyperkinesis (choreoathetosis). With mechanical jaundice, toxic-dyscirculatory encephalopathy of varying severity can develop, even up to severe damage to the brain and spinal cord. The symptoms of polyradiculoneuropathy have the following features: at the beginning of the disease, one or two nerves are affected, and after a few weeks or months, the process gradually takes over all limbs with the development of the most often sensitive form of polyradiculoneuropathy, which is characterized by pain, paresthesias, and disorders of surface sensitivity.

Diseases of the pancreas (pancreonecrosis, purulent pancreatitis, cyst, insulinoma, diabetes).

Enzymatic dysfunction of the pancreas with the release of a large number of proteolytic enzymes plays a leading role in the pathogenesis of neurological complications in pancreatitis.

This leads to a disturbance of the water-electrolyte balance, general intoxication. Edema, changes in blood vessel walls and circulatory disturbances develop in the brain. Neuropsychiatric disorders are often caused by hypoglycemia or hyperglycemia. Acute encephalopathy syndrome develops against the background of severe pancreatitis and is manifested by sharp psychomotor excitement, meningeal and conduction symptoms. Chronic encephalopathy is formed in patients who suffer from pancreatitis for a long time, in the stage of enzymatic decompensation of the pancreas. In hypoglycemic encephalopathy, delirious, meningeal, polyneuropathic, pseudotumorous, hemiplegic, comatose, and convulsive forms are distinguished. In hyperglycemic conditions due to diabetes, the following syndromes are distinguished: neurasthenic, encephalopathic, polyneuropathic, autonomic polyneuropathy, neuralgia and neuropathy of individual nerves, most often facial, as well as hyperglycemic (diabetic) coma. In the pathogenesis of encephalopathy and strokes in diabetes, an important role belongs to microangiopathies, that is, damage to arterioles, precapillaries that vascularize the cortex, subcortical formations and brain stem. Diagnosing strokes against the background of diabetes is marked by certain difficulties. Neurological symptoms are masked by sensitivity disorders, aniso- and areflexia accompanying diabetes. In many patients, the course of diabetes worsens during the acute period of a stroke. Disorders of cerebral circulation can be complicated by the development of hyperglycemic coma, which complicates differential diagnosis with diabetic (hyperglycemic) coma. With diabetes, polyneuropathic disorders are often observed, which are manifested by the predominance of sensitive, vegetative and motor symptoms. Very often, diabetic polyneuropathies show peripheral autonomic failure, which is manifested by orthostatic hypotension, tachycardia, nocturnal diarrhea, and pelvic disorders.

Diseases of the gastrointestinal tract (ulcer disease, gastritis, colitis) are more often manifested in the form of neurasthenic and polyneuropathic syndromes. Their pathogenesis is caused by disorders of various types of metabolism (absorption processes of proteins, vitamins, fats, carbohydrates, trace elements, etc. are disturbed) and pathological impulses from internal organs, which can lead to functional, and later to small organic changes in the nervous system. For example, vitamin B deficiency is considered as one of the causes of peripheral nerve damage. Premorbid autonomic dysfunction should also be taken into account. Neurosthenic disorders are observed in approximately 13 patients. One of the leading symptoms of the pathology of the gastrointestinal tract is pain in the Zakharyin-Ged zones. Polyneuropathic syndrome is characterized by pain and small disturbances of sensitivity in the distal parts of the limbs, vegetative disorders.

Kidney diseases (glomerulo- and pyelonephritis, poisoning, trauma, urolithiasis) in the stage of subcompensation and especially decompensation can cause polyneuropathy, encephalopathy, dyskalemic paralysis, uremic coma. The pathogenesis of neurological disorders in the stage of subcompensation is caused by a decrease in the colloid-osmotic pressure of the blood, an increase in the permeability of blood vessels with the development of edema, diapedesis hemorrhages, and changes in nerve cells. In the stage of decompensation, the combination of toxic effects of azotemia, metabolic acidosis, hyperkalemia and hypercreatinemia, as well as arterial hypertension, becomes of primary importance. At the beginning of the disease, the neurasthenia-like syndrome is manifested by symptoms of hypersthenia, and then by symptoms of hyposthenia. It should be taken into account that kidney pathology can cause an exacerbation of the lumboischialgic syndrome in patients with deforming spondylosis and osteochondrosis of the spine. Toxic damage or mechanical compression of the vegetative plexus of the kidneys can lead to the appearance of renocardial and renovisceral syndromes. For example, renocardial syndrome is characterized by long-lasting pain in the left half of the chest, which regresses simultaneously with the regression of renal failure. Acute encephalopathic disorders usually occur against the background of a sharp and rapid increase in renal failure. Patients have general brain, meningeal and minor focal symptoms. With a significant increase in hypo- or hyperkalemia, dyskalemic paralysis often develops - weakness of the muscles of the arms, legs, and trunk, which can reach the level of complete immobilization. Hypokalemic paralysis is mainly expressed in the proximal

parts of the limbs, and hyperkalemic paralysis - in the muscles of the face, pharynx, and larynx. Disorders of cerebral blood circulation are more often observed in chronic nephritis complicated by arterial hypertension. Treatment of chronic renal failure with the help of hemodialysis led to the appearance of a new form of neurological pathology - dialysis encephalopathy, the main manifestation of which is dementia. To date, the cause of the appearance of this pathology remains unclear.

Gynecological diseases, pregnancy and menopause are most often manifested by the syndromes of vegetative-vascular dystonia, lumboischialgia, myopathy, encephalopathy and disorders of cerebral circulation. VSD syndrome develops more often against the background of menopause and inflammatory diseases of the small pelvis as a result of concomitant dysfunction of the hypothalamic-pituitary-ovarian system. Radiculalgias are caused by irritation of the roots and nerves as a result of inflammatory diseases of the appendages, ligaments or compression of these formations by the pregnant uterus. Encephalopathy and focal disorders of blood supply develop more often as a result of toxicosis of pregnant women, which is accompanied by changes in kidney function (increased blood pressure, azotemia). Myelopathic disorders with ischemia of the spinal cord are observed with large uterine fibroids and in the II-III trimester of complicated pregnancy. During physical work or walking, there are sudden attacks of leg weakness, paresthesia. During a short rest, these disorders pass. Paroxysmal disorders can later turn into more persistent ones with disorders of urination and defecation (by the type of delay). Changes in water-electrolyte (calcium) metabolism may manifest as tetanic symptoms.

Damage to the connective tissue.

Nodular periarteritis, temporal arteritis, lupus erythematosus, rheumatism are often accompanied by syndromes of encephalopathy, polyneuropathy, myasthenia, myopathy. The pathogenesis of these disorders is due to autoimmune degenerative-inflammatory changes in the membranes of the brain, spinal cord and blood vessels. Rheumatism can be complicated by cerebral disorders with predominant damage to the subcortical nodes - minor chorea. The previously dominant concept of "cerebral rheumovasculitis" as a frequent cause of damage to the nervous system turned out to be wrong. Damage to cerebral vessels in rheumatism is a rarity. Among the causes of cerebral strokes, both primary vasculitis and vasculitis in connective tissue diseases occupy a prominent place. Special attention is drawn to Sneddon's syndrome, which is associated with the antiphospholipid factor - perhaps the most frequent cause of ischemic strokes in young patients. Temporal arteritis (Horton's disease) is characterized by sharp local pain in the temporal area. During palpation, a thickened and painful temporal artery is observed. Nodular periarteritis is pathomorphologically characterized by damage to small arteries with the development of dense nodules along their course. Since vessels of almost all organs and tissues, including the nervous system, can join the process, the clinical manifestations of the disease are extremely diverse.

Blood diseases (malignant anemia - Addison-Birmer disease, leukemia, lymphogranulomatosis, hemorrhagic diatheses).

Addison-Birmer anemia and its neurological disorders - funicular myelosis and polyneuropathy have at their core the same pathogenetic factor - vitamin B12 deficiency. Pathological changes are more pronounced in the posterior and lateral cords of the spinal cord, so the clinical picture is characterized by a combination of sensory and motor disorders mainly in the lower extremities. Leukemias are often accompanied by damage to the nervous system due to the fact that malignant leukemic infiltrates develop in vessels and membranes. Most often, the spinal cord is affected with the development of transverse myelitis. According to the development and course, two types of lesions are distinguished - vascular and pseudotumorous. The basis of the vascular type is thrombosis and hemorrhage, often multiple, characterized by the acute development of paresis, paralysis, and speech disorders. The pseudotumorous type is caused by massive infiltration of cellular elements of the myeloid series at different levels of the nervous system and is manifested by a gradual increase in general brain and focal symptoms, epileptic seizures. Hemorrhagic diatheses (hemophilia, thrombocytopenic purpura (Werlhoff's disease),

hemorrhagic capillarotoxicosis (Schönlein-Henoch's disease) are manifested by one main symptom - a tendency to bleed. Damage to the nervous system is a consequence of hemorrhages in the brain, meninges, and spinal cord.

Endocrine diseases.

They are often accompanied by various neuropsychological and neuromuscular disorders. Sometimes neurological symptoms are the main complaints of the patient. Dysfunction of the thyroid gland. Hyperthyroidism (hyperfunction of the thyroid gland). Myasthenia is quite common, in 5% of patients weakness is the main complaint, in half of patients weakness is one of the symptoms. Weakness is more pronounced in the proximal musculature than in the distal musculature. Damage to the cortico-spinal tract in thyrotoxicosis is manifested by increased tone in the muscles of the legs, hyperreflexia, plantar pathological signs. Hyperthyroidism can cause euphoria, but its more frequent manifestation is hyperactivity. Almost all patients have a tremor, the frequency of which is identical to the physiological one, but the amplitude is usually greater. Ocular symptoms associated with hyperthyroidism are divided into two main types. The first type includes retraction of the eyelids, wide eye slits, rare blinking. Damage of the second type is caused by pathological changes in the orbit and its structures. These include pronounced exophthalmos, diplopia, eye pain, ophthalmoplegia. Hypoparathyroidism is more often due to a decrease in the secretion of parathyroid hormone with a decrease in the level of calcium in the blood serum. Clinically, this is manifested by tetanic convulsions, which occur more often in the distal muscles. Sometimes epileptic seizures can be observed, which clinically do not differ from attacks of idiopathic epilepsy. CT scan reveals intracerebral calcifications. With hyperparathyroidism, neurological symptoms are observed in 50-70% of cases. Myopathic syndrome is most often observed. It is characterized by a predominant lesion of the leg muscles, rapid fatigue, muscle hypotonia, and pain in the limbs. Damage to the hypothalamus and other endocrine glands (pituitary gland, adrenal glands, etc.). Itsenko-Cushing's disease (basophilic adenoma of the pituitary gland with excess secretion of ACTH) is manifested by fatigue, lethargy, sleep disorders, increased blood pressure, impaired fat metabolism, sexual function, bone and skin damage, and steroid myopathy. A clinically similar syndrome can be observed with hypothalamic dysfunction. With acromegaly syndrome (eosinophilic adenoma of the pituitary gland and hypersecretion of THG), there is a disproportionate growth of bones and soft tissues, especially the face, as well as headache and pain along the roots and peripheral nerves, visual disturbances, hyperhidrosis, amenorrhea, impotence. Symonds' syndrome (fading of the function of the anterior lobe of the pituitary gland with a sharp decrease in the production of ACTH, THG, etc.) is manifested by severe somatic and neuropsychological disorders - asthenia, apathy, arterial hypotension, sharp weight loss up to the development of cachexia.

Malignant neoplasms.

Neuropsychiatric disorders are caused by the presence of a tumor in the parenchyma, brain membranes or general metabolic-toxic and vascular disorders that accompany the growth of tumors. Clinical symptoms of brain metastases are similar to primary brain tumors in many respects. In patients without metastases, encephalopathic lesions with acute, subacute or gradual development of mental changes, small subcortical-stem disorders are often noted.

Palliative care is a comprehensive approach, the purpose of which is to ensure the maximum quality of life of a patient with an incurable (fatal) disease and his family members, by preventing and alleviating suffering due to early detection and accurate diagnosis (assessment) of emerging problems, and the implementation of adequate treatment measures (with pain syndrome and other life disorders), as well as providing psychosocial and moral support.

The medical component of palliative care consists in monitoring the patient's physical condition, symptomatic treatment of pathological manifestations of the disease, including pain, counseling and training the patient and his relatives and relatives in patient care skills.

According to existing definitions, palliative medicine is a branch of scientific medicine and health care, the main task of which is to improve the quality of life and alleviate the suffering of patients with various nosological forms of chronic incurable diseases, mainly in the terminal period

of their progression and under conditions when the capabilities of a specialized treatment of the underlying disease is limited or, from the point of view of modern scientific concepts, unpromising.

Palliative care should be started in parallel with other types of treatment (for example, chemotherapy or radiotherapy, surgical or conservative treatment) of a patient with an incurable chronic disease. While specific (pathogenetic or etiotropic) treatment is aimed at the recovery of the patient, palliative care should provide prevention and reduction of suffering that occurs as a result of the relentless progression of the disease. Palliative care and etiotropic therapy should be combined into a single course of patient treatment. It is necessary to start providing palliative care before the symptoms become uncontrollable.

Therefore, palliative care:

Provides relief from pain and other symptoms or disorders of life that cause suffering;

Affirms life and views death as a natural process;

Not intended to hasten or delay the onset of death;

Combines social, psychological and spiritual aspects of patient care;

Offers a support system that helps the patient live as actively as possible until death;

Offers a support system that helps the family survive the difficult times of the patient's illness and bereavement - the death of a loved one;

Uses a complex interdisciplinary (team) approach aimed at the needs of the patient and his family;

Improves the quality of life and has a positive effect on the course of the disease;

It is prescribed regardless of the stage of the disease or the main treatment, in combination with other therapies that provide life extension, for example, such as chemotherapy and radiation therapy, and includes studies that are necessary to better understand the dynamics of the disease, control and prevent clinical complications.

2. Components of palliative care

patient palliative pain analgesic

PD is a holistic approach consisting of medical, social, psychological and spiritual components.

The medical component of PD consists in monitoring the physical condition of the patient, symptomatic treatment of pathological manifestations of the disease, including pain, providing consultations and teaching skills useful in caring for the patient.

The social component aims to provide satisfactory living conditions, support social life, and at the same time help patients with HIV infection/AIDS and their environment to learn how to independently solve social problems.

The psychological component within PD solves the task of alleviating psychological conditions associated with an incurable disease, overcoming stress, anxiety, depression of patients, their environment, and staff involved in providing PD. The task of psychological support within PD is to normalize the internal state of the patient, maintain the psychological balance between the patient and his environment at all stages of the course of the disease, during the preparation of the patient for ART and accompanying treatment, discussion and preparation for death, assistance to the close environment of the patient during and after severe losses

Spiritual support is provided by a representative of a religious denomination, at the patient's choice. Regardless of the organizational form of medical care, it is important to ensure the possibility of performing religious ceremonies if needed.

3. Treatment of pain syndrome

Taking into account the fact that protection from pain in incurable patients is a component of the patient's right to qualified medical assistance, medical care and a dignified death, without a doubt, it is necessary to urgently review the norms limiting the access of palliative patients to opioid analgesics in Ukraine.

After all, "the credit of trust in medicine is preserved as long as the latter is able to effectively deal with pain."

In general, the pain syndrome can be divided into two categories: nociceptive and

neuropathic.

Nociceptive pain occurs as a result of stimulation of an intact pain receptor and, in turn, is divided into:

- somatic pain (due to stimulation of pain receptors of the skin, soft tissues, muscles and bones)

- visceral pain (due to stimulation of pain receptors of internal organs)

Non-narcotic and narcotic analgesics are used to treat nociceptive pain.

Neuropathic pain occurs as a result of damage to the myelin sheath of axons of peripheral nerves and occurs in 40% of HIV-infected patients with an advanced stage of the disease. As a rule, it is a manifestation of peripheral symmetric polyneuropathy (PSP), apparently arising as a result of the direct action of the human immunodeficiency virus and characterized by a decrease in sensitivity, a feeling of numbness, the appearance of ringing in the ears, "tingling" and pain, mainly in the lower limbs and feet.

The treatment of neuropathic pain requires the use of non-narcotic and narcotic analgesics in combination with adjuvant treatment using antidepressants or anticonvulsants.

In addition, some antiretroviral drugs, such as didanosine (ddI) and stavudine (d4T), can cause similar symptoms of neuropathy, which are associated with damage to the axons of peripheral nerves, mainly in the lower extremities. In this case, replacing the indicated antiretroviral drugs in treatment regimens can lead to a reduction in pain syndrome, although this does not always happen.

Treatment of pain syndrome should begin with a thorough and systematic study, in particular, determination of the possible etiology and nature of pain. Important characteristics are the intensity of the pain, its type, the impact of the pain syndrome on the general condition and the possibility of its relief.

Intensity of pain. For its evaluation, a numerical standard scale is used to describe pain from 0 to 10 points, where 0 is described as the absence of a pain syndrome, and 10 as the most pronounced pain. It is especially important to use this scale at certain intervals in a particular patient to monitor changes in condition.

Type of pain. Nociceptive pain can be described as deep, throbbing, dull, or as a stabbing sensation. Neuropathic pain can be described as burning, tingling, tingling, ringing in the ears, numbness, or other pathological sensations. These characteristics can help when choosing pain relievers, especially when neuropathic pain is suspected.

Impact of pain. Assess the impact of pain on the patient's functional and mental state, ability to perform normal daily work, the results of such an assessment should be clearly documented in the medical records.

The possibility of pain relief. Assess and describe conditions or interventions that lead to exacerbation or relief of pain syndrome.

Severe chronic pain is most common in patients with malignant tumors, chronic pancreatitis, joint damage, and severe neuropathy. The rank distribution of analgesic use for mild, moderate, and severe pain, along with the potential use of adjuvant agents at each stage, as well as the characteristics of the main pain relievers are shown below.

4. Opioid analgesics

In the absence of an effect from the use of non-opioid analgesics, opioids are prescribed.

Weak opioids include tramadol, which is a μ-agonist.

Tramadol hydrochloride, daily dose up to 400 mg. Tramadol is usually taken at 50-100 mg every 6 hours. The most common side effects of tramadol are dizziness, nausea, and drowsiness.

Strong opioids include promedol, buprenorphine, omnopon, morphine, and fentanyl.

Promedol (higher daily dose of 240 mg). With parenteral use, the analgesic effect is more pronounced.

Buprenorphine is a partial μ-agonist. Buprenorphine has a higher analgesic potential than morphine and a longer analgesic effect (6 hours). Buprenorphine is characterized by the so-called "ceiling" effect: an increase in the dose above 3-5 mg/day is not accompanied by an increase in

analgesia, which is considered the main drawback of buprenorphine in the treatment of chronic pain (maximum daily dose of 3.6 mg).

Omnopon - contains approximately 50% morphine and approximately 35% other opium alkaloids. In terms of potency, it is approximately 1.5-1.8 times weaker than morphine (the highest daily dose is 160 mg).

Morphine is a classic representative of opioid analgesics - μ-receptor agonists. Morphine is quickly absorbed both when taken internally and when administered subcutaneously. The effect develops after 10-15 minutes after subcutaneous administration and after 20-30 minutes after oral administration. The effect of a single dose lasts 3-5 hours (maximum daily dose is 120 mg).

5. Adjuvant medicines

The following adjuvant drugs can either extend the range of action of analgesics, or have an independent analgesic effect.

Tricyclic antidepressants (amitriptyline, imipramine) are often used to treat neuropathic pain. They have relative contraindications in patients with coronary artery disease and risk of ventricular arrhythmias. Amitriptyline has an analgesic effect, but is poorly tolerated due to a pronounced cholinolytic effect (dry mouth, urinary retention, constipation, hallucinations). A sedative effect and orthostatic hypotension are often noted, which may limit its simultaneous use with narcotic analgesics. ECG monitoring is necessary to rule out heart rhythm disturbances, especially in patients receiving anthracycline anticancer drugs. Administration of the full dose of amitriptyline at night normalizes sleep and reduces daytime side effects, but the patient should be warned about the possibility of orthostatic hypotension at night. The analgesic effect of amitriptyline is manifested in low doses (25-150 mg/day). It is recommended to start with 10-20 mg for patients with body weight more 50 кгand 0.3 mg/kg for patients with body weight less 40 кг. The dose is slowly increased until the desired effect is achieved (maximum 150 mg in adults and 3 mg/kg in children).

Antihistaminic drugs (diphenhydramine) have analgesic, antiemetic and moderately sedative effects. The usual dose is 10-20 mg orally or intramuscularly every 4-6 hours (0.5-1 mg/kg for children).

Benzodiazepines (sibazone, diazepam, relanium) are effective for the treatment of sudden anxiety and muscle spasms accompanied by acute pain. They are indicated for patients who have contraindications to taking antidepressants, as well as for the treatment of terminal dyspnea. With the exception of pain associated with muscle spasms, these agents are not effective analgesics, and their sedative and respiratory depressant effects are enhanced when combined with opioids. Therefore, patients with symptoms of anxiety and pain are first prescribed and adjusted doses of an opioid analgesic, and then benzodiazepines are used.

Caffeine in a single dose of approximately 65 mg quite successfully enhances the analgesic effect of NSAIDs. The optimal daily dose of caffeine has not been established, but 65-200 mg/day is usually well tolerated by most patients. Single doses of 1.0-1.5 mg/kg can be used in children with chronic cancer pain.

Corticosteroids have specific and nonspecific effects in the treatment of chronic pain. They can directly treat some tumors (for example, lymphoma) and relieve pain by reducing swelling in the area where the tumor is compressing soft tissues, nerves, or the spinal cord. Corticosteroids are prescribed as a method of emergency therapy for spinal cord tumor compression (dexone 16-96 mg/day or its equivalent). Corticosteroid treatment (Dexon 16 mg/day or its equivalent) can be effective when pain occurs from damage to the brachial or lumbosacral plexuses in cancer patients in the absence of an effect from large doses of opioids. In patients in the terminal stage of HIV infection, corticosteroids can improve mood and appetite, while reducing pain; side effects should not bother the doctor in this situation. Long-term use of steroids causes weight gain, Cushing's syndrome, proximal myopathy and psychosis (rarely), increases the risk of gastrointestinal bleeding, especially when combined with NSAIDs. Corticosteroid withdrawal syndrome may increase pain.

Anticonvulsants (carbamazepine, baclofen) can relieve attacks of acute tic-like pain in such

peripheral neurological syndromes as trigeminal, postherpetic, glossopharyngeal neuralgia, etc., which occur against the background of nerve damage caused by a viral infection, a tumor, or specific anticancer treatment. Similar to tricyclic antidepressants, gabapentin at a dose of 2.4-3.6 mg/day is effective for diabetic neuropathy and postherpetic neuralgia.

Receptor agonists (clonidine, clofelin) are used in the treatment of opioid-resistant neuropathic pain at 0.075-0.01 mg orally 2-3 times a day. The patient should be warned about the need to take a sufficient amount of liquid (1.5 - 2 лper day) against the background of taking the drug in order to avoid arterial hypotension. Uncontrolled administration of Clofelin to weakened, exhausted patients with hypovolemia is dangerous. Clofelin should not be prescribed to patients with bradycardia due to the agootropic properties of this drug.

Palliative care should be available to the patient around the clock and provided in an amount acceptable to the patient, in accordance with approved standards and clinical protocols. Palliative care can be provided taking into account the needs and consent of a specific patient at home, in a polyclinic (as a consultative service) or in inpatient treatment and prevention facilities of any ownership with the use of various organizational forms of medical care, including, in a day hospital, in specialized inpatients (hospices or palliative care units). For example, if a patient wants to receive palliative care at home, health professionals can teach relatives, loved ones or neighbors who are caring for him, how to properly give him the drugs prescribed by the doctor, as well as provide other types of medical and psychological care. They should also be able to turn, if necessary, to medical professionals for advice.

Therefore, WHO recognizes that appropriate services should be established in each country to meet the medical and social needs of palliative patients.

An institution where incurable (incurable) patients can receive professional, specialized, complex and multidisciplinary palliative care is called a hospice in many countries. The first mention of the word "hospice" (from Latin - shelter, protection) in a close to modern meaning can be found in written records of the Middle Ages. Among the pilgrims going to the Holy Land, there were many people who hoped that the Almighty would save them from a serious illness. On the way, they stopped at monasteries to rest. The premises provided to such people were called hospices.

Materials for self-control of training quality

Tasks for self-control :

Task 1.

The "ambulance" team was called to the patient who suddenly lost consciousness; Convulsions, respiratory arrest, blue lips, pale face, swollen neck veins, dilated pupils are observed. The attack ended with slow urination, retrograde amnesia developed. The attack was observed twice in an hour. After an attack in a neurological state: lethargy of pupillary reflexes, weakness of convergence, asymmetry of the nasolabial folds, decreased muscle strength in the right limbs, increased tendon and periosteal reflexes on the right. In the Romberg pose, he performs rough swinging, PNP and KPP with a miss to the right. It is known from the anamnesis that similar attacks have occurred during the last 5 years, developed after a myocardial infarction. Paroxysmal asystole was recorded on the ECG.

Make a diagnosis and justify it.

Answer: Neurological status data, anamnesis and ECG data make it possible to make a diagnosis: Paroxysmal asystole, Morgana-Adoms-Stokes syndrome, epileptiform form. For the treatment of such patients, complex medical measures are used, aimed at reducing the excitation of the nervous system, stabilizing the heart rhythm, improving blood supply and metabolic processes in the myocardium.

Task 2.

Against the background of emotional stress, patient N., 58 years old, developed pain in the area of the heart, left arm, shoulder, and shoulder blade. On the second day, the patient experienced headache, dizziness, noise in the head, darkening of the eyes, lethargy, drowsiness, feeling of fear, anxiety, boredom. At the end of the second day, the patient developed motor aphasia, hemiparesis of the right half of the body. In the blood, there is an increased level of cholesterol, on REG, there is a slowing down of cerebral blood flow due to heart failure, a drop in blood pressure, venous congestion. On the ECG - a decrease in the voltage of the PQ complex , a negative T wave, bradyarrhythmia.

Make a diagnosis.

Answer: Myocardial infarction in combination with acute ischemic disturbance of cerebral circulation in the left hemisphere.

Task 3.

Patient P., 64 years old, suffers from shortness of breath, hemoptysis for 3 months. In the last year, he was constantly suffering from bronchitis; smokes up to 20 cigarettes a day. He feels a diffuse headache, dizziness. Adynamia, increased excitability, gets tired quickly, poor sleep, blood pressure and pulse fluctuations, profuse sweating, occasional disturbances of consciousness in the form of somnolence. On the fundus - phenomena of stagnation. In the blood - an increase in SZL up to 60 mm/hour, leukocytes $16.8 \cdot 10^{9}$ /l. On REG - reduction of pulse blood filling in all vascular basins, venous stasis.

Make a diagnosis.

Answer: Lung cancer. Vegetative-vascular dystonia.

Task 4.

Patient Z., 36 years old, was brought to the infectious disease hospital with a diagnosis of acute hepatitis. Against the background of the treatment, the patient's condition improved somewhat, but after eating a protein meal and sweets, he suddenly developed a headache, dizziness, and nausea. In a few hours, he lost consciousness, tendon reflexes increased, abdominal and plantar reflexes decreased, there were unstable pathological reflexes, stiffness of the muscles of the back of the head, Kernig's, Bekhterev's symptoms. Within a day, mydriasis, lack of reaction of the pupils to light, decreased corneal, abdominal, tendon, periosteal reflexes, convulsions joined.

Make a diagnosis.

Answer: Acute hepatitis complicated by encephalopathic syndrome, hepatic coma.

Materials for classroom self-training:

List of educational practical tasks:

1. Diagnose somatoneurological pathology.
2. To justify the appointment of laboratory, electrophysiological and other studies.
3. To treat disorders of the nervous system in diseases of internal organs.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

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Practical lesson No. 29

Topic: Hereditary-degenerative diseases of the nervous system: neuromuscular, and with damage to the pyramidal, extrapyramidal and cerebellar systems.

The goal: to create an idea among the applicants about the clinic of systemic degenerations (pyramidal, extrapyramidal, cerebellar systems), the main principles of treatment; issues of prevention, prognosis of the listed diseases.

Basic concepts: Hereditary diseases of the pyramidal, extrapyramidal and cerebellar systems are widespread. Many of these diseases have begun to be well treated. Therefore, their early diagnosis is of great importance. Doctors of all specialties should be aware of the risk and prevention of pathology, for the occurrence of which, along with external factors, hereditary predisposition is of great importance.

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

Materials for self-control of training quality

Tests

1. Specify the diseases in which the extrapyramidal system is affected.
A) Huntington's chorea
B) family paroxysmal paralysis
C) Friedreich's disease
D) Wilson-Konovalov disease
D) Pierre-Marie disease
Answer: a, g

2. Specify the diseases in which the coordination system is damaged.
A) Werdnig-Hoffman disease
B) Huntington's chorea
C) Friedreich's disease
D) Pierre-Marie's disease
D) Parkinson's disease
Answer: in, g

3. Name a disease with a predominantly autosomal recessive type of inheritance.
A) Huntington's chorea
B) Pierre-Marie ataxia
C) Friedreich's disease
D) Strümpel's disease
D) Wilson-Konovalov disease
Answer: in, d

4. Which of the listed drugs are used to treat hepato-cerebral dystrophy?
A) depenicillamine
B) proserin
B) cuprenyl
G) unitiol
D) dexamethasone
Answer: a, c, d

5. Choose one correct answer about the type of inheritance of Duchenne progressive muscular dystrophy:

a) autosomal recessive type; Answer: c)
b) autosomal dominant type;
c) X-linked recessive type;
d) X-linked dominant type;
d) linked to the Y-chromosome.

6. For patients with atrophic myotonia Kurshman-Batten-Steinert, the following symptoms are characteristic, with the exception of one. Specify which one?

a) myotonic syndrome;
b) atrophic syndrome; Answer: d)
c) decrease in the activity of the gonads;
d) point cataracts and early baldness;
d) increase of knee and Achilles reflexes.

7. In a 70-year-old man, the fingers and hand of the right hand began to tremble almost 3 years ago, and this was most evident when the patient was at rest. With active movements, the tremor decreased. After some time, stiffness of the right limbs developed, which gradually moved to the left. The march was broken; it became slow, with small "shuffling" steps. Speech became quiet, monotonous, facial expressions impoverished. Intellectual functions were not affected.

1. What brain structures are affected?
2. What treatment should be prescribed?

Answer.

1. Pallidar department of the extrapyramidal system.
2. Antiparkinsonian treatment.

8. A 15-year-old boy has a shaky gait, his speech has changed, he has become "shock-like". The knee-heel and toe-nose tests are performed with a miss; Romberg's symptom is positive. Achilles reflexes are not triggered. The arch of the foot is high, the big toe on it is hammer-shaped. Kyphosis.

1. What disease should be assumed?
2. Specify the type of inheritance.

Answer:

1. Friedreich's hereditary ataxia.
2. Autosomal recessive type.

Tasks for self-control :

An 18-year-old girl began to notice that she gradually developed weakness and wasting of the muscles of the shoulder blades, she could not raise her arms above the horizontal level. A year later, she noticed the weakness of the facial muscles, impoverished facial expressions. The patient's mother also suffers from a similar disease. Objectively: Mental abilities are preserved. The face is "myopathic". "Wing-shaped" blades. The symptom of "free shoulders" appears. Reflexes in the hands are sharply reduced, legs are normal. Atrophy of the muscles of the upper shoulder girdle.

1. Make a diagnosis.
2. What is the prognosis of the disease?
3. Is it possible to give birth to a child in such a state?

Answer:

1. Scapulofacial myopathy Landuzi-Dezherin.
2. The prognosis for life is satisfactory.

Yes, but half of the children born will be prone to this form of myopathy

Materials for classroom self-training:

List of educational practical tasks:

1. Master research methods:
   e) dermographism;
   f) pilomotor reaction;

g) hydrophilicity of the skin;
h) Danini-Ashner reflex;
i) clino-orthostatic test.

Instructional material for mastering professional skills

1. Investigate the neurological status of the patient and correctly interpret the obtained data.
2. Determine the level of damage to the nervous system.

Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

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Practical lesson No. 30

Topic: Practical skills

Purpose: to generalize the skills acquired in previous classes, identify problem areas and eliminate them.

Basic concepts: no doctor can work without mastering the practical skills of neurological examination of a patient, since all doctors must know the symptoms of life-threatening neurological diseases (meningitis, intracranial hemorrhage, brain tumor, etc.)

Equipment: classroom, furniture, equipment.

Plan:

1. Organizational moment (greetings, checking those present, announcing the topic, the purpose of the lesson, motivating students to study the topic).
2. Control of basic knowledge (written work, written test, frontal survey, etc.):
   - Requirements for students' theoretical readiness to perform practical classes (knowledge requirements, list of didactic units);

General neurology.
Practical experience:

1. Methods of research of surface reflexes
2. Methodology of research of deep reflexes
3. Methods of researching pathological foot and hand reflexes
4. Study of reflexes of oral automatism
5. Assessment of muscle strength
6. Methods of detecting signs of peripheral and central paralysis.
7. Techniques for detecting fibrillar twitching and muscle atrophy.
8. Methodology of examination of muscle tone and determination of spastic and plastic muscle hypertension.
9. Methods of detecting extrapyramidal disorders (hyperkinetic-hypotonic and hypokinetic-hypertonic syndromes).
10. Cerebellar function examination technique. Checking the coordination of movements.
11. Methods of detecting static ataxia.
12. Techniques for detecting dynamic ataxia.
13. Methodology of surface sensitivity research.
14. Deep sensitivity research methodology.
15. Methodology for the study of complex types of sensitivity (stereognosis, sense of localization, discrimination, two-dimensional spatial sense).
16. Techniques for examination of symptoms of sciatic and femoral nerve root tension (Nery, Laseg, Dezherin, Bekhterev, "landing", Wasserman, Matskevich).
17. Methods of research of the functions of the olfactory analyzer.
18. Methods of researching the functions of the visual analyzer (acuity, field of vision, color perception).
19. Study of the functions of the oculomotor nerves
20. Trigeminal nerve examination technique
21. Methodology of examination of the facial nerve (functions of mimic muscles, taste sensitivity).
22. Methodology of research of the vestibulo-cochlear nerve (hyperacusis and hypoacusis, tests of Rinne, Weber).
23. Methods of research of vestibular functions.

24. Methodology for determining aphasias.
25. Methods of detecting apraxias (kinetic, ideational, spatial, constructive).
26. Methods of detecting agnosias (visual, auditory, astereognosis, anosognosia).
27. Methods of examination of the autonomic nervous system, investigation of autonomic tone, autonomic reactivity (dermographism, Kerdo index, Aschner-Dagnini test, ortho-clinostatic).
28. Methodology for the study of meningeal signs (rigidity of the occipital muscles, symptoms of Kernig, Brudzinsky, Mendel, Bekhterev), the method of performing a lumbar puncture and assessing cerebrospinal fluid indicators in normal conditions and with meningitis of various etiologies.

1. Methods of research of surface reflexes: skin (abdominal, plantar, cremasteric, anal), mucous membranes (corneal, conjunctival, pharyngeal, soft palate).

Cutaneous reflexes are caused by line irritation of the skin with a blunt needle.

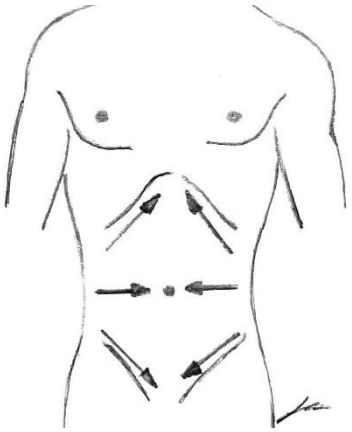

Fig. 1. Techniques for inducing abdominal reflexes

Abdominal reflexes are caused by rapid stroke irritation of the abdominal skin from the periphery to the middle (Fig. 1):

1) upper (Th 7- Th 8) – below the costal arch,
2) medium (Th 9- Th 10) - at the level of the navel,
3) lower (Th 11- Th 12) – above the Poupart ligament.

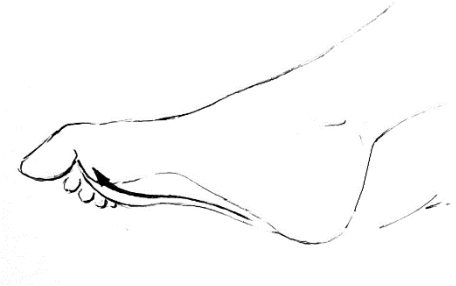

Fig. 2. The method of inducing the plantar reflex

The plantar reflex (L 5- S 1) is caused by line irritation of the outer edge of the sole and consists in the plantar flexion of all the toes (Fig. 2).

The cremasteric reflex (L 1- L 2) is caused by line irritation of the skin of the inner surface of the upper third of the thigh from the bottom up and consists in raising the testicle.

The anal reflex (S 4- S 5) is caused by irritation of the skin near the anus, in response, a contraction of the anal sphincter is observed.

Reflexes from mucous membranes are caused by :

By gently touching a thin strip of paper or cotton wool to the cornea above the iris (corneal) or to the conjunctiva (conjunctival), in response, the eye closes, a reflex arc is formed by V and VII pairs of cranial nerves.

The pharyngeal reflex and the palatal reflex are caused by irritation of the mucous membrane with a spatula, respectively of the back wall of the pharynx and the soft palate, in response there are vomiting movements (pharyngeal reflex) or lifting of the soft palate and tongue (palatal reflex), the reflex arc is formed by the IX and X pairs of cranial nerves

2. The technique of studying deep reflexes: tendon (flexor ulnar and extensor ulnar, knee, Achilles) and periosteal (superior, carporadial).

Tendon reflexes.

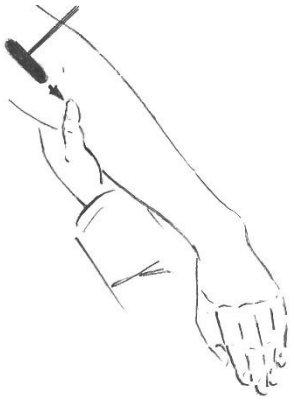

Fig. 3. The method of inducing the elbow reflex from the biceps

Elbow flexion reflex from the biceps (C5—C6) are caused by a short blow of the hammer on the tendon of the biceps muscle of the shoulder in the elbow bend. In response, the forearm bends at the elbow joint (Fig. 3).

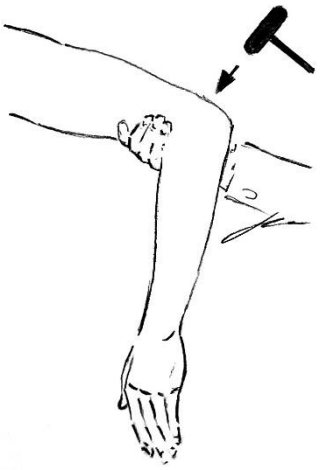

Fig. 4. Techniques for inducing the elbow flexion reflex from the triceps

The elbow extensor reflex from the triceps (C7—C8) is caused by a hammer blow on the tendon of the triceps muscle of the shoulder above the ulnar process (Fig. 4).

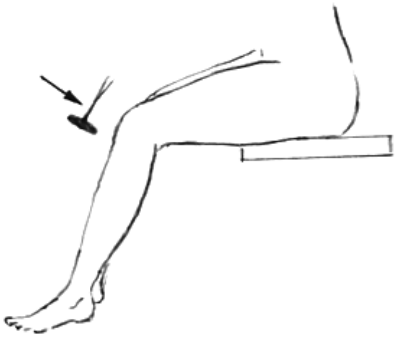

Fig. 5. Method of inducing the knee reflex

The knee reflex (L 3- L 4) is caused by a blow of the hammer on the tendon of the quadriceps muscle of the thigh, on the ligament of the patella. In response, the lower leg is extended (Fig. 5).

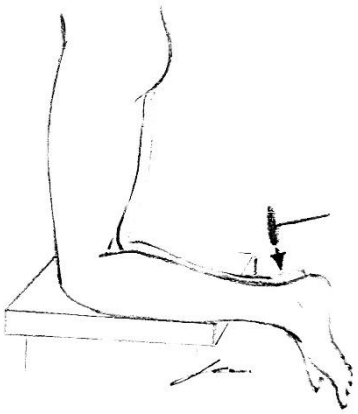

Fig. 6. Techniques for inducing the Achilles reflex

The Achilles reflex (S 1- S 2) occurs as a result of a hammer blow on the Achilles tendon, accompanied by plantar flexion of the foot (Fig. 6).

Periosteal reflexes:

The superbrow reflex is caused by hitting the brow arch with a hammer. The motor reaction consists in closing the eyelids. The reflex arc is formed by the trigeminal and facial nerves.

The carpal-radial reflex (C5-C8) is caused by a hammer blow on the styloid process of the radius. In response, there is flexion of the forearm in the elbow joint, flexion of the fingers and pronation of the hand, not all components of the response may be observed.

3. Methods of researching pathological reflexes: foot-extensor (Babinsky, Oppenheim, Gordon, Schaeffer, Pusep), flexor and their cetacean analogues (Rossolimo, Zhukovsky, Bekhterev). Foot extensor reflexes with extension of the big toe are early signs of organic damage to the pyramidal system.

Fig. 7. The technique of inducing the Babinski reflex

Babinski's reflex - in case of line irritation of the outer edge of the sole, there is an extension of the big toe, which is sometimes supplemented by simultaneous fan-like separation of all other toes (fan sign) (Fig. 7). The reflex is observed normally in children up to 1-1.5 years of age, with the formation of gait it disappears and the plantar reflex is triggered.

Fig. 8. The method of inducing the Oppenheim reflex

A similar movement of the fingers can be caused by pressing the front surface of the tibial crest with the thumb from top to bottom (Oppenheim's reflex) (Fig. 8).

Fig. 9. The method of inducing the Schaeffer reflex

A similar movement of the fingers can be caused by hand compression of the calf muscle (Gordon's reflex) or the heel tendon (Sheffer's reflex) (Fig. 9).

Fig. 10. The method of inducing the Rossolimo reflex

Flexion foot reflexes. The Rossolimo reflex is caused by intermittent blows of a hammer or fingertips on the plantar surface of the distal phalanges of the patient's toes. A quick plantar flexion of II — V toes occurs. A similar reaction occurs with light blows of a hammer on the back surface of the patient's foot (Bekhterev's reflex) and on the middle of the sole near the base of the fingers (Zhukovsky's reflex).

4. Study of reflexes of oral automatism (subcortical): sucking, proboscis, distance-oral, palmar, chin.

Reflexes of oral automatism are automatic, involuntary movements performed by the mouth muscles in response to mechanical irritation of various parts of the face. In the case of tapping with a hammer on the root of the nose, lip stretching occurs (naso-lip reflex). The same reaction is observed as a result of a light blow of the hammer on the upper or lower lips (oral reflex) and when the hammer approaches the patient's mouth (distance-oral reflex). The palmar-chin reflex of Marinescu-Radovich is caused by line irritation of the palmar area of the thumb (thenar) elevation. In response, there is a contraction of the chin muscles. Reflexes are normally observed in infants.

5. Assessment of muscle strength according to the point system (from 0 to 5 points).

The following scale is used to assess muscle strength:
0 points – no muscle contraction.
1 point - there is muscle contraction, but there is no movement in the joint.
2 points - there is movement in the joint, but it is impossible to overcome the force of gravity.
3 points - it is possible to overcome the force of gravity, but movement is impossible when overcoming the resistance.
4 points - it is possible to overcome some resistance.
5 points – muscle strength is normal.

6. Methods of detecting signs of peripheral and central paralysis.

Signs of central paralysis:

A) Hypertonia – increase in muscle tone of the spastic type, that is, the most significant at the beginning of the movement. Tone is assessed by performing passive movements in the limbs, that is, the movements are performed by the researcher, while the patient must be completely relaxed.

B) Hyperreflexia – increase in tendon and periosteal reflexes with simultaneous suppression of skin reflexes (abdominal and plantar). Reflexes are checked according to the standard method given above.

C) Pathological reflexes are evaluated according to the standard method given above.

D) Pathological synkinesis are reflex accompanying movements that are normally absent, the following synkinesis are distinguished:

Global - have a generalized nature, that is, in response to muscle contraction on the healthy side, as well as when coughing, straining, movements occur in the paralyzed limbs, for example, when the patient squeezes the doctor's palm with a healthy hand, there is bending of the forearm, hand and fingers in the paralyzed hand (shortening reaction), and in the leg - extension and adduction (lengthening reaction).

Coordination - contraction of paralyzed muscles when performing movement with other muscles that are functionally related to them, for example, Babinski's synkinesia - the patient from a supine position with arms crossed on the chest tries to sit down, while the paralyzed limb rises up.

Imitative synkinesis - when the healthy limb performs movements (for example, foot extension), a similar "mirror movement" occurs in the paretic limb.

D) Protective reflexes (reflexes of spinal automatism) are involuntary movements in the paralyzed limb when an intense irritation is applied, for example, a needle prick. Bending of the limb is usually observed - "reaction of shortening", sometimes - "reaction of lengthening".

7. Techniques for detecting fibrillar twitching and muscle atrophy.

Fibrillar muscle twitches are assessed visually, for this the patient must be undressed, muscle atrophy is also assessed, a centimeter tape is used to objectively assess muscle volume.

8. Methodology of examination of muscle tone and determination of spastic and plastic muscle hypertension.

Muscle tone is assessed by palpating them in a state of rest and complete relaxation, as well as by examining passive movements in the joints also under conditions of complete muscle relaxation.

With spastic muscle hypertonia, the tone increases according to the spastic type, or the "complex knife" type, that is, the resistance felt by the muscles is the greatest at the beginning of the movement, and then it decreases.

With plastic muscular hypertonia, the resistance felt by the muscles is constant throughout the entire movement in the form of plastic or waxy stiffness, and intermittent, stepwise stretching of the muscles can also be observed, which is called the "cogwheel symptom."

Hypotonia of the muscles mainly occurs when the peripheral motor neuron is damaged.

9. Methods of detecting extrapyramidal disorders (hyperkinetic-hypotonic and hypokinetic-hypertonic syndromes).

Visually assess the presence of involuntary movements (hyperkinesis in hyperkinetic-hypotonic syndrome), the speed of voluntary movements (slowed down in hypokinetic-hypertonic syndrome), the patient's posture, facial expressions, expressiveness of speech.

Muscle tone is evaluated by palpating them in a state of rest and complete relaxation, as well as by examining passive movements in the joints under conditions of complete muscle relaxation. To detect hidden extrapyramidal hypertension, the Neuke-Ganev test is performed - during passive movement in the elbow joint, the patient is asked to simultaneously raise the leg (bend at the hip joint), while an increase in tone in the muscles of the upper limb is observed.

10. Cerebellar function examination technique. Checking the coordination of movements.

Asynergy is detected using the Babinsky test: the patient, lying on his back with his arms crossed on his chest, is offered to sit down without the help of his hands. A patient with asynergia will not be able to sit down, because he does not have a synergistic contraction of the gluteal muscles, which fix the legs and pelvis to the surface of the couch. The Stuart-Holmes test, or the symptom of the absence of a return impulse: the patient is asked to clench the hand into a fist, bend the arm at the elbow joint and resist the doctor who is trying to unbend the patient's forearm.

During the sudden termination of the countermeasure by the doctor, the patient's fist hits the chest due to the lack of timely contraction of the antagonist muscles.

11. Methods of detecting static ataxia.

To detect static ataxia, Romberg's position is used: the patient stands with closed legs, arms extended forward, fingers spread, the test is performed with open and closed eyes. To detect minor violations, a complicated Romberg pose is used - the feet are on the same line, the toe touches the heel.

12. Techniques for detecting dynamic ataxia and coordination of movements.

Fig. 11. Finger-nose test

Finger-nose test: the patient stands with closed legs, arms extended forward, fingers spread, the patient should touch the tip of the nose with the index finger of the outstretched hand with closed eyes (Fig. 11).

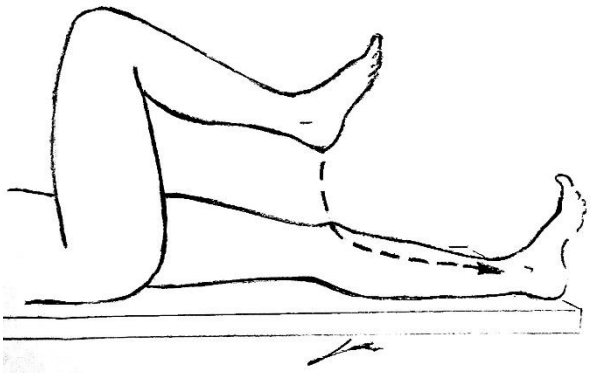

Fig. 12. Heel-knee test

Heel-knee test: the patient lies on his back, with his eyes closed, the heel of one leg touches the knee joint of the other leg, then he accurately runs the heel along the ridge of the tibial ridge from the knee to the foot (Fig. 12).

13. Methodology of surface sensitivity research.

Conditions: with closed eyes, irritation in symmetrical areas, once.

To test pain sensitivity, use a non-sharp, so as not to violate the integrity of the skin, preferably a disposable needle. Needles touch the skin with either a blunt or a sharp end. Injections should be short and not frequent. During the touch, the patient should answer: "sharp" or "dull". Injections are carried out in symmetrical parts of the body from top to bottom.

Temperature sensitivity is checked using test tubes with hot (40-45ºC) and cold (5-10ºC) water. The patient must determine the nature of the irritation and its intensity.

Tactile sensitivity is examined using a piece of cotton wool, gauze or a brush. The touch should be slight. A more advanced method is to use the Frey method with a set of hairs.

14. Deep sensitivity research methodology.

Musculoskeletal sensitivity is tested by passive small movements in the joints, starting with the distal parts of the limbs and small joints.

Vibration sensitivity is checked with a tuning fork with a frequency of oscillations of 64 or 128 Hz, the leg of which is placed on the bone protrusions and the period during which the patient feels vibration is noted, normally this period is 14-16 s.

The sensation of pressure and mass is investigated using a set of weights of different weights, which are applied to a certain area of the skin, normally a person perceives changes of 10 percent from the initial mass.

Kinesthesia of the skin is studied by displacing the skin trapped in the fold.

15. Methodology for the study of complex types of sensitivity (stereognosis, sense of localization, discrimination, two-dimensional spatial sense).

Stereognosis is tested by feeling things with the eyes closed. There are the following requirements for checking stereognosis:

A) A preliminary check of tactile and muscle-joint sensitivity should not reveal their disorders, since pseudostereognosis occurs when these types of sensitivity are affected.

B) The subjects to be checked must be familiar to the patient.

C) The objects being tested should not produce familiar sounds.

The two-dimensional spatial sensation is examined with closed eyes, drawing numbers, letters, and figures on the patient's skin, which he must recognize correctly.

The sensation of localization is checked with closed eyes, applying tactile stimuli to the patient, the localization of which he must determine, as a rule, errors do not exceed 1 см.

The feeling of discrimination is checked with the help of a Weber compass, simultaneously touching its legs to the skin of the patient and determining how the patient perceives the touch - as one or separate, by measuring the minimum distance between the legs of the compass, when the touch is still felt as separate.

16. Techniques for examination of symptoms of sciatic and femoral nerve root tension (Nery, Laseg, Dezherin, Bekhterev, "landing", Wasserman, Matskevich).

Neri's symptom - increased pain in the area of the affected roots when bending the neck (bringing the chin to the sternum).

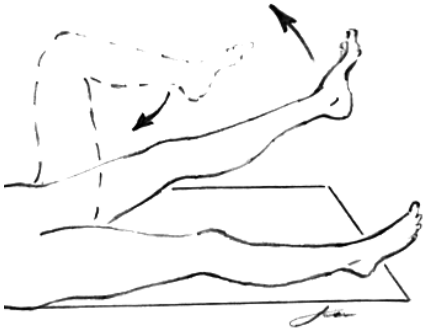

Fig. 13. Laseg's symptom

Laseg's symptom - the appearance or intensification of pain along the course of the sciatic nerve in a patient lying on his back during flexion in the hip joint of the lower limb, extended in the knee joint (first phase), when bending the limb in the knee joint, the pain decreases or disappears (second phase) (Fig. 13).

Dezherin's symptom is an increase in pain in the affected area during coughing and sneezing.

Bekhterev's symptom - the appearance of pain in the lumbar region during bending of the healthy leg in the hip joint

Symptom of "sitting" - in a patient who is lying on his back, an attempt to sit down leads to bending of the knee joint of the lower limb on the side of the lesion.

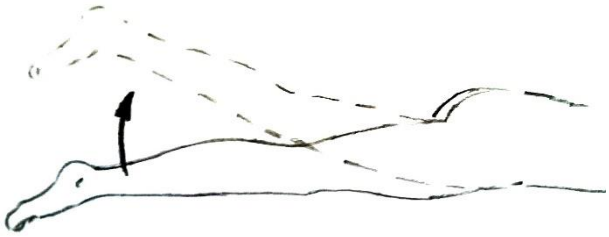

Fig. 14. Wasserman's symptom

Wasserman's symptom - the occurrence of pain or its intensification in the area of the front part of the thigh during the extension of the leg in the hip joint in a patient who is lying on his stomach.

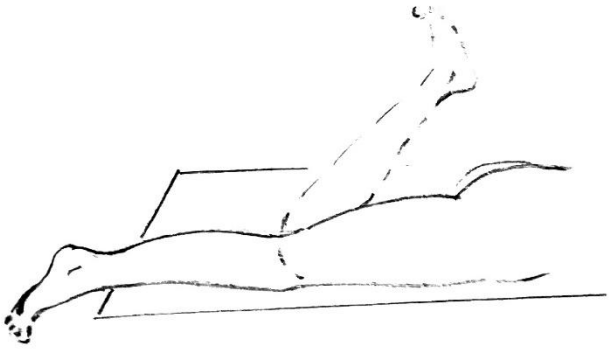

Fig. 15. Matskevich's symptom

Matskevich's symptom is the occurrence of pain or its intensification in the area of the front part of the thigh during bending of the leg in the knee joint in a patient lying on his stomach (Fig. 15).

17. Methods of research of the functions of the olfactory analyzer.

The patient is allowed to sniff an aromatic substance through each nostril, while avoiding harsh irritants (rubbing alcohol) that stimulate the receptors of the trigeminal nerve. It is necessary to find out whether the patient feels the smell and whether he recognizes it.

18. Methods of researching the functions of the visual analyzer (acuity, field of vision, color perception).

Visual acuity is checked separately for each eye using special tables consisting of 12 rows of letters or open rings or contour drawings. A healthy eye can distinguish letters in 10 lines from

a distance of 5 meters, this visual acuity is conditionally taken as 1. For example, if the patient's eye can distinguish letters in 6 lines, visual acuity is estimated as 0.6, if in 2 lines - 0.2.

Fields of vision are examined using a special device - a perimeter, which consists of a graduated arc rotating around a horizontal axis, an arc graduated from 0 to 90º in each direction from the center. In the center of the arch, a mark is placed on the inner surface, on which the patient fixes his gaze with the other eye closed, which is not tested at this time. Visual fields are evaluated separately for each eye. The patient informs the doctor when he notices a white mark, which is moved from the middle to the center in different planes, conducting research along the meridians every 15 degrees. The points drawn on the diagrams are combined and the boundaries of the field of view are obtained. Normally, the outer limit of the field of vision is equal to 90º, the upper and inner - 50-60º, the lower - about 70º.

Fig. 16. Evaluation of the field of vision by the control method.

An approximate assessment of the field of vision is possible using a sample with a towel, the center of which is placed in front of the eye (each eye is examined separately) in a horizontal plane, the patient should divide the towel in half, indicating its center, and a control method, when the doctor and the patient sit opposite each other, close different eyes and evaluate the perception of the object in the frontal plane, while everyone looks at each other (Fig. 16).

The condition of the fundus is determined using an ophthalmoscope. Normally, the optic disc is round, its boundaries are clear, and its color is pale pink. The ratio of diameters of arteries and veins is 2:3.

Color perception is assessed using the polychromatic tables of Rabkin et al.

19. Study of the functions of the oculomotor nerves.

The patient's complaints are collected (whether there is doubling in front of the eyes and when looking in which direction it appears or intensifies).

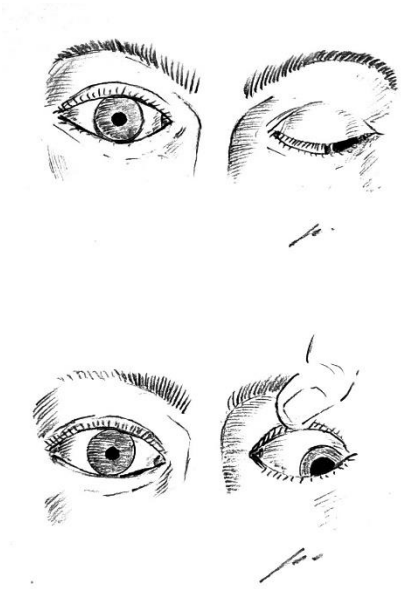

Fig. 17. Damage to the root of the oculomotor nerve on the left (compression genesis)

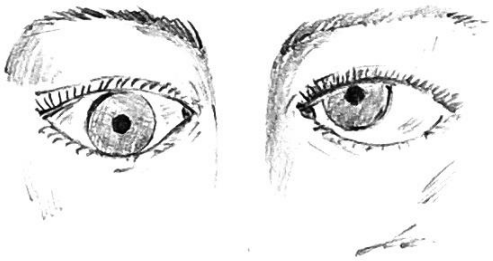

Fig. 18. Lesions of the block nerve on the left

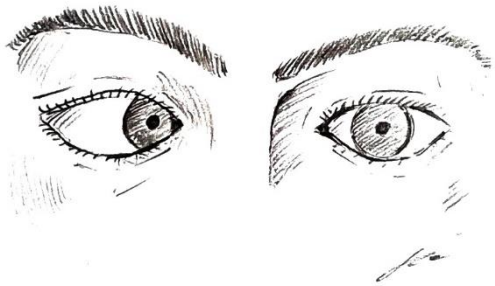

Fig. 19. Lesions of the abductor nerve on the left

Visually assess the patient's eyes (presence of ptosis, protrusion or depression of the eyeball, assess the shape and compare the diameter of the pupils, assess the presence of convergent or divergent strabismus) (Fig. 17-19).

With the help of a hammer, on which the patient must fix his gaze without turning his neck and head, the amount of active movements of the eyeballs to the right, left, up, down, and also when the hammer approaches the nose is checked (convergence check, while the axes of the eyeballs converge to the average lines).

Using a flashlight or covering the eye with the palm of the hand and moving the palm away, check the reaction of the pupils to light, illuminating the eyes one by one - normally the pupils narrow. Constriction of the pupil in the illuminated eye is a direct reaction to light, in the

contralateral eye it is a conjugal reaction. The reaction of the pupils to convergence is studied when the hammer approaches the bridge of the patient's nose, normally the pupils narrow. Pupils' reaction to accommodation is studied for each eye separately, the patient must monitor the approaching hammer, while the pupil narrows.

20. Trigeminal nerve examination technique (facial sensitivity, pain points, trigger zones, suprabrow, corneal, mandibular reflexes).

To examine the sensitivity on the face, the skin of the face is touched symmetrically on each side in the innervation zones of the branches of the trigeminal nerve with a needle, cotton wool and tubes with cold and warm water and in segments - from the ear area to the corner of the mouth in Zelder's zones.

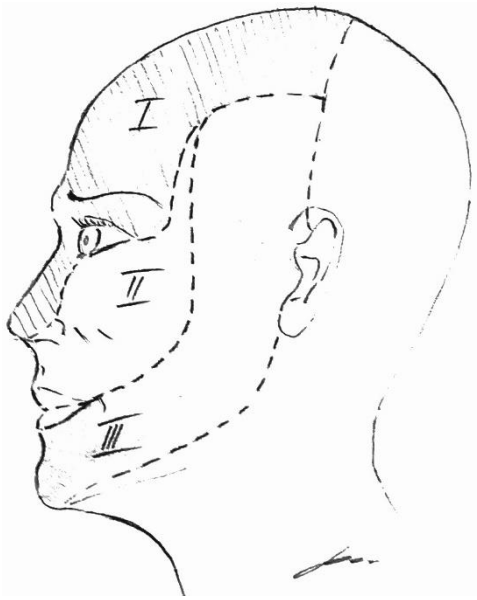

Fig. 20. Zones of innervation of branches of the trigeminal nerve

Palpate the exit points of the branches of the trigeminal nerve - for this, press with a finger on the supraorbital and infraorbital and mental points (Fig. 20).

The superbrow reflex is caused by hitting the brow arch with a hammer. The motor reaction consists in closing the eyelids. The reflex arc is formed by the trigeminal and facial nerves.

Corneal and conjunctival reflexes are caused by gently touching a thin strip of paper or cotton wool to the cornea above the iris of the eye (corneal) or to the conjunctiva (conjunctival), in response, eye closure is observed, the reflex arc is formed by the V and VII pairs of cranial nerves. (see item 1)

The mandibular reflex is caused by percussion with a hammer on the chin with the mouth half-open separately from the right and left sides, in response, the lower jaw is raised due to the contraction of the masticatory muscles, the reflex arc is formed by the mandibular nerve.

21. Methodology of examination of the facial nerve (functions of facial muscles, taste sensitivity).

Fig. 21. Lesions of the facial nerve on the left according to the peripheral type

The study of the function begins with a facial examination, checking the asymmetry of the face, the smoothness of the skin folds on the forehead and nasolabial folds (upper and lower level) (Fig. 21). Then the patient is offered to raise and frown his eyebrows, close his eyes, show his teeth, puff out his cheeks, pull out his lips with a tube, and whistle. With closed eyes, check for the presence of Bel's symptom (white strip of the sclera). After that, check for lacrimation or dryness of the conjunctiva of the eye, presence of hyperacusis, and taste sensitivity on the front two thirds of the tongue.

22. Methodology of research of the vestibulo-cochlear nerve (hyperacusis and hypoacusis, tests of Rinne, Weber).

The patient's complaints are investigated, hearing acuity, bone and air conduction are assessed.

Hearing acuity is tested for each ear separately using whispered speech. With closed eyes, the patient must repeat the words that are spoken in a whisper from a distance 6 м. Hearing acuity is studied in more detail with the help of audiography.

To assess damage to the sound-receiving or sound-conducting system, a tuning fork with an oscillation frequency of 128 hertz is used.

Try Rinne. The leg of the sounding tuning fork is placed on the nipple. After the patient stops hearing the vibration through the bone, the tuning fork is brought to the ear at a distance of 1- 2 см. A healthy person perceives sound through the air twice as long. Such a result is evaluated as a positive Rinne test. If the Rinne test is negative, that is, the patient does not hear the sound of the tuning fork, this indicates damage to the sound-conducting apparatus.

Weber's test. The leg of the sounding tuning fork is placed in the middle of the crown, normally the sound is equally perceived by both ears, i.e. there is no lateralization of the sound. When the sound-conducting apparatus is damaged, the sound is better felt by the affected ear (lateralization in the affected ear). When the sound-receiving apparatus is damaged, the sound is better felt by the healthy ear (lateralization in the healthy ear).

23. Methods of research of vestibular functions.

The examination of the function of the vestibular analyzer is carried out mainly in the clinic of otolaryngology and includes an assessment of the patient's complaints and a check for the presence of spontaneous nystagmus, balance disorders, performing coordination tests, determining the excitability of the vestibular analyzer using caloric and rotational tests. The leading complaint is systemic vertigo - a feeling of rotation of one's own body or surrounding objects in one direction (clockwise or counterclockwise). Vestibular nystagmus is an involuntary, rapidly repeating rhythmic twitching of the eyeballs.

24. Methods of detecting bulbar and pseudobulbar disorders (pharyngeal reflexes, soft palate reflexes, speech articulation, oral automatism reflexes).

Pharyngeal and palatal reflexes are caused by irritation of the mucous membrane of the back wall of the pharynx and the soft palate with a spatula, respectively: in response, vomiting and coughing movements occur in the case of a pharyngeal reflex and lifting of the soft palate and movement of the tongue during the palatal reflex.

Reflexes of oral automatism arise as a result of mechanical irritation of various parts of the face. In the case of tapping the hammer on the root of the nose, the lips are drawn out (naso-labial reflex), the proboscis reflex is caused by tapping the hammer on the upper or lower lip, the answer is the same as in the previous case. The same reaction is observed when the hammer approaches the patient's nose or mouth (distance-oral reflex).

The palmo-chin reflex of Marinescu-Radovich is caused by a stroke irritation of the palm in the thenar area (elevation of the thumb), in response to the irritation, a contraction of the chin muscles is observed.

Dysarthria is characterized by articulation disorders, indistinctness, unintelligibility of speech, but, unlike aphasia, the patient forms phrases and sentences correctly, the ability to write and understand oral and written speech is preserved. To assess articulation, the patient is offered words that are difficult to pronounce, for example, "three hundred and thirty-third artillery brigade" and others.

25. Methodology for determining aphasias.

The presence of fast spontaneous speech, understanding of speech and repetition of phrases are evaluated, the type of aphasia depends on the test results:

Table

Differential diagnosis of aphasias

Type of aphasia and localization of the pathological focus	Presence of rapid spontaneous speech	Language comprehension	Repetition of phrases
Total	There is none	There is none	There is none
Broca (motor), posterior part of the inferior frontal gyrus on the left in right-handed people	There is none	IS	There is none
Wernicke (sensory), posterior part of the upper temporal gyrus on the left in right-handed people	IS	There is none	There is none
Transcortical motor (premotor area on the left in right-handed people)	There is none	IS	IS
Transcortical sensory (temporal-occipital area on the left in right-handed people)	IS	There is none	IS
Conductive or conductive (subcortical area of the blood supply of the left middle cerebral artery in right-handed people)	IS	IS	There is none
amnestic (anomia)	IS	IS	IS

26. Methods of detecting apraxias (kinetic, ideational, spatial, constructive).

In order to detect apraxia, the patient is offered to perform complex actions with real objects (doing hair, etc.), with imaginary objects (showing how to pour water, etc.), then it is suggested to perform actions (threaten with a finger, draw a plan of a room, make a figure out of matches) . If the patient cannot perform a certain action, the doctor demonstrates it and offers to repeat it.

Kinetic apraxia occurs when the premotor zone of the cortex of the frontal lobe is damaged. It is characterized by a violation of both complex movements and actions according to the task,

following imitation. Motor perseverations are characteristic, that is, involuntary repetition of the same movement.

Ideational apraxia occurs as a result of damage to the supramarginal gyrus of the left parietal lobe in right-handed people and is bilateral. The patient loses the plan or idea of a complex action, its phases are rearranged, cannot perform certain verbal tasks (threats with a finger), but can repeat the doctor's actions.

Spatial apraxia occurs as a result of damage to the inferior parietal and parietal-occipital regions of the left hemisphere. Leads to a violation of spatial relations. For example, a patient cannot draw a plan of a room according to the task.

Constructive apraxia is caused by damage to the left angular gyrus. At the same time, the patient cannot make a whole out of parts, for example, make a geometric figure out of matches.

27. Methods of detecting agnosias (visual, auditory, astereognosis, anosognosia).

Visual (optical) agnosia is tested by showing objects that the patient must name.

Auditory agnosia is tested by recognizing objects by their characteristic sounds (a clock ticks, a dog barks).

Stereognosis is examined by feeling things that the patient must name with closed eyes. There are the following requirements for checking stereognosis:

A) A preliminary check of tactile and muscle-joint sensitivity should not reveal their disorders, since pseudostereognosis occurs when these types of sensitivity are affected.

B) The subjects to be checked must be familiar to the patient.

C) The objects being tested should not produce familiar sounds.

Anosognosia is checked by detecting the patient's awareness of his defect (for example, paresis).

28. Methods of examination of the autonomic nervous system.

Skin manifestations of vegetative disorders: discoloration, erythema, pigmentation, moisture, swelling, greasiness, hair growth (hypertrichosis, alopecia), nail trophic (thickening, delamination, fragility, Mesa strips), trophic ulcers, skin changes after herpes. Determination of iris color and pigmentation, size (mydriasis, miosis), uniformity (anisokoria). Claude-Bernard-Horner syndrome - ptosis, miosis, enophthalmos - occurs when cervical sympathetic nodes are affected.

Vascular reflexes in case of mechanical irritation of the skin. Distinguish between white (sympathicotonia); pink (Eytonia); red, elevator (vagotonia).

Reflex dermographism – stroke pressure with a sharp object. It disappears when the responsible segments of the spinal cord are affected.

Sweat reflexes are studied when the body is warmed up, the introduction of diaphoretic substances (pilocarpine, aspirin). Minor's method (iodostarch test). Research of electrocutaneous resistance (SHGR - galvanic skin response or evoked skin sympathetic potentials - VSHSP).
Study of the permeability of blood vessels (hydrophilicity) by McClure and Aldrich. Pharmacological tests (histamine, adrenaline, etc.). The most convenient is the study of vegetative-cardiac reflexes.

- Study of autonomic and vascular reactions of the skin with the help of ultraviolet radiation - acceleration and deceleration of the reaction. Suppression of erythema in segmental lesions of the central nervous system.

- Thermal imaging study - objectifies some reflex-vascular reactions during scanning of infrared radiation against the background of viscero-cutaneous connections, during pain reactions, etc.

Currently, the study of the "vegetative portrait", which includes:

The study of vegetative tone (special tables) allows to assess the state of the nervous system at rest.

The study of autonomic reactivity - with the help of a large number of cardiovascular tests (for example, Dan'ini-Aschner, Thomas-Roux) - evaluates the speed of response to irritation and recovery after bringing the brain out of a state of rest, which is important for assessing the severity of damage to the central nervous system, prognosis and others

Vegetative maintenance of activity (physical, mental, emotional) - assesses the ability of the central nervous system to long-term support of vegetative reactivity at a certain level.

It is also necessary to palpate the projection of nodes of the sympathetic trunk, plexuses (periarterial, cervical, solar, etc.).

Research of vegetative algic points of Markelov-Birbrair, which are symmetrically located on the sides of the body and occur in the pathology of different departments of the central nervous system. Sliding palpation is performed. Pain reactions are assessed on a five-point scale. Construction of "algic curves" can determine regional, generalized, symmetrical, asymmetrical syndromes.

The study of sensitivity with the help of Zakharyin-Hed zones (viscero-cutaneous ligaments) indicate irritation of the segmental-peripheral visceral apparatus of the ANS.

Identify and differentiate paroxysmal autonomic disorders (dizziness, fainting, acrocyanosis, Quincke's edema, urticaria, vasomotor rhinitis, hypothalamic crises, trigeminal pain, migraine, sleep attacks, autonomic-visceral auras, etc.).

Cardiovascular tests:

The Kerdo index is used to assess tone in the cardiovascular system, the Kerdo index is calculated according to the following formula: K = (1 – D/P), where K is the Kerdo index, D is diastolic pressure, P is pulse rate, eutonia, or vegetative equilibrium is observed at the value of the index from -10 to +10, sympathikotonia - at values greater than +10, parasympathikotonia - at values less than -10.

The Aschner-Danini pericardiac reflex allows you to assess the autonomic reactivity of the parasympathetic nervous system. The reflex is induced by pressing for 20 seconds with the pads of the doctor's fingers on the front-side surfaces of the patient's eyeballs. Normally, the pulse rate decreases by an average of 8 beats in 1 minute. In the case of vagotonia, the pulse slows down by more than 10 beats per minute, in the case of sympathicotonia it does not change or becomes more frequent.

The Chermak reflex is very similar in mechanism to the Aschner reflex and occurs when the mm is pressed. sternocleidomastoidei.

Thomas-Roux solar reflex – pressing on the cells of the solar plexus projection, the reaction is similar to Aschner's reflex.

Prevel's orthostatic reflex consists in an increase in pulse rate and blood pressure as a result of a change in the position of the body in space - from horizontal to vertical and characterizes the vegetative support of activity. Normally, the pulse increases by 8-12 beats per minute, and the blood pressure increases by 5-10 10 ммmmHg.

Danielopolo's clinostatic reflex is characterized by a slowing of the pulse by 6-8 beats in 1 minute and a decrease in blood pressure by 5- 10 ммHg. in response to a change in body position from vertical to horizontal.

29. Methodology for the study of meningeal signs (rigidity of the occipital muscles, symptoms of Kernig, Brudzinsky, Mendel, Bekhterev), the method of performing a lumbar puncture and assessing cerebrospinal fluid indicators in normal conditions and with meningitis of various etiologies.

Fig. 22. Assessment of stiffness of the occipital muscles

Stiffness of the occipital muscles - an attempt to bend the patient's head causes a sharp increase in the tone of the occipital muscles (Fig. 22).

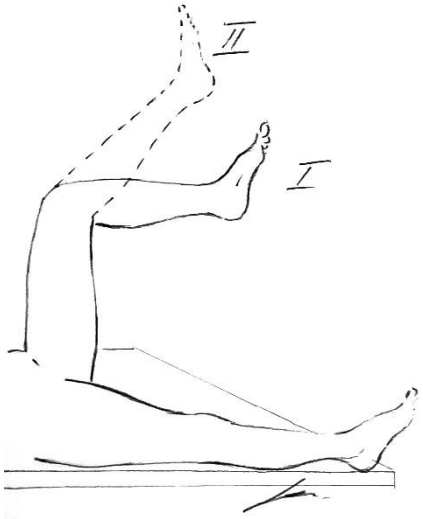

Fig. 23. Kernig's symptom

Kernig's symptom - the patient's leg, which is in a horizontal position facing up, is bent at a right angle in the hip and knee joints, the extension of the lower limb in the knee joint is impossible due to the increased tone of the muscles of the back surface of the thigh (Fig. 23).

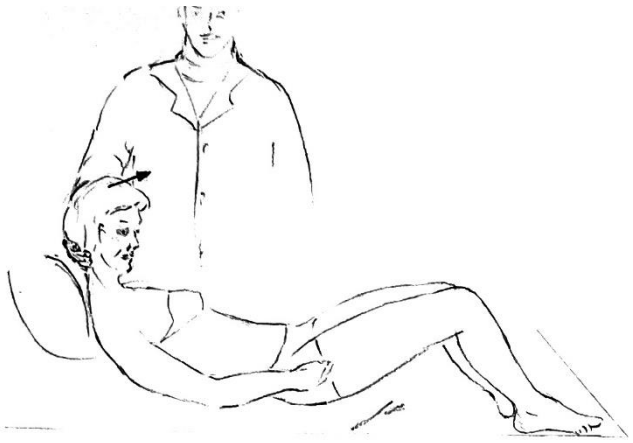

Fig. 24. Brudzinsky's upper symptom

Brudzinsky's upper symptom - during the examination of the stiffness of the occipital muscles, the patient's lower limbs involuntarily bend in the hip and knee joints (Fig. 24).

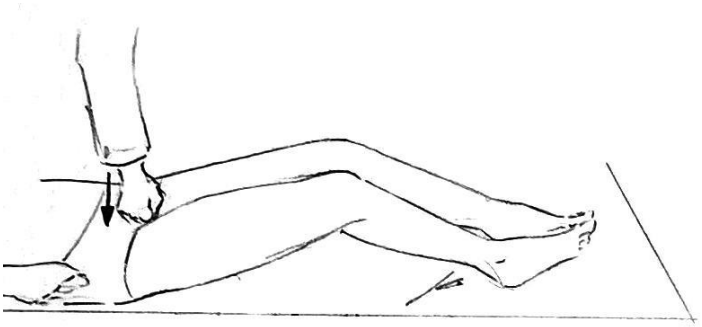

Fig. 25. Brudzinsky's average symptom

Brudzinsky's average symptom - when pressing on the area of the pubic symphysis, the patient's lower limbs involuntarily bend in the hip and knee joints (Fig. 25).

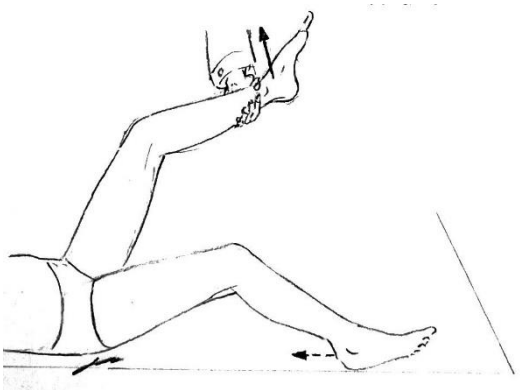

Fig. 26. Brudzinsky's lower symptom

Brudzynski's lower symptom - during Kernig's symptom check, the second lower limb of the patient involuntarily bends in the hip and knee joints (Fig. 26).

Mendel's symptom - when pressing on the front wall of the external auditory canal, there is a sharp pain and a grimace of pain.

Bekhterev's symptom - percussion of the zygomatic arch causes a sharp headache and painful grimace.

Lumbar puncture:

For the timely detection of drug allergies and prevention of complications when using anesthetics in accordance with the instructions on the procedure for diagnosing drug allergies (order of the Ministry of Health and the AMS of Ukraine No. 127/18 dated 02.04.2002), a skin test must be performed (according to the methodology given in the appendix 2 to Order No. 127/18).

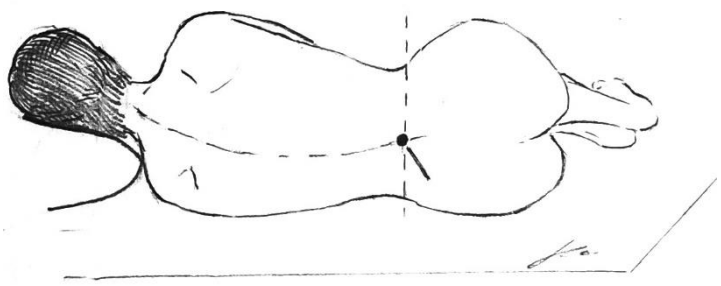

Fig. 27. Position of the patient during a lumbar puncture and guidelines for finding the city of its performance.

Lumbar puncture technique: the patient lies on his side, the hips are brought to the body, the head is tilted forward, the assistant holds the patient firmly (Fig. 27). The skin of the lumbar area is disinfected with a 5% alcohol solution of iodine, then thoroughly wiped with alcohol, the skin and subcutaneous fat in the puncture area are infiltrated with a 1% solution of novocaine. The needle is inserted between the spinous processes of the L3-L4 vertebrae. A needle with a mandrel is inserted sagittally in a slightly upward position, at the depth approximately 5 смthe resistance of the yellow ligament is felt, after passing through which the needle pierces the dura mater, which is accompanied by a characteristic crunch, deepening by 2 - the 3 ммneedle ends up in the subarachnoid space. In order to carry out chemical and bacteriological research, 5-10 ml of cerebrospinal fluid are collected in two sterile test tubes. After that, the needle is removed and the puncture site is covered with a sterile napkin.

Tasks for self-checking the rising level of knowledge-skills .

List the arcs of the main spinal reflexes
To list the research methods of individual CH
List methods of sensitivity research
List the methods of research of the coordinating sphere
List the methods of research of cortical functions

The list of educational practical tasks that must be completed during the practical session:

explore:

- Reflexes
- Sensitivity
- Coordination
- Cranial nerves
- cortical functions

Tests of different levels.

1. Methodology of the study of surface reflexes
2. Methodology of research of deep reflexes
3. Research methodology of pathological foot and hand reflexes
4. Study of reflexes of oral automatism
5. Assessment of muscle strength
6. Techniques for detecting signs of peripheral and central paralysis.
7. Techniques for detecting fibrillar twitching and muscle atrophy.
8. Techniques for examination of muscle tone and determination of spastic and plastic muscle hypertension.
9. Methods of detecting extrapyramidal disorders (hyperkinetic-hypotonic and hypokinetic-hypertonic syndromes).
10. Techniques for examination of cerebellar functions. Checking the coordination of movements.
11. Methods of detecting static ataxia.
12. Techniques for detecting dynamic ataxia.
13. Methodology of surface sensitivity research.
14. Deep sensitivity research methodology.
15. Methodology for the study of complex types of sensitivity (stereognosis, sense of localization, discrimination, two-dimensional spatial sense).
16. Techniques for examination of symptoms of sciatic and femoral nerve root tension (Nery, Laseg, Dezherin, Bekhtereva, "landing", Wasserman, Matskevich).
17. Methodology for researching the functions of the olfactory analyzer.
18. Methods of researching the functions of the visual analyzer (acuity, field of vision, color perception).

19. Study of the functions of the oculomotor nerves
20. Trigeminal nerve examination technique
21. Facial nerve examination technique (functions of facial muscles, taste sensitivity).
22. Vestibulo-cochlear nerve research methodology (hyperacusis and hypoacusis, Rinne, Weber tests).
23. Methods of research of vestibular functions.
24. Methods of detecting bulbar and pseudobulbar disorders (reflexes: pharyngeal, soft palate, speech articulation, reflexes of oral automatism).
25. Methods of determining aphasias.
26. Techniques for detecting apraxias (kinetic, ideational, spatial, constructive).
27. Techniques for detecting agnosias (visual, auditory, astereognosis, anosognosia).
28. Methods of examination of the autonomic nervous system, investigation of autonomic tone, autonomic reactivity (dermographism, Kerdo index, Aschner-Dagnini test, ortho-clinostatic).
29. Methodology for the study of meningeal signs (rigidity of the occipital muscles, symptoms of Kernig, Brudzinsky, Mendel, Bekhterev), the method of lumbar puncture and assessment of cerebrospinal fluid indicators in normal conditions and with meningitis of various etiologies.

Special neurology

Lesson goals:

Specific goals:

1) Learn the principles of classification of vascular diseases of the brain.
2) Interpret the peculiarities of transient disorders of cerebral blood circulation.
3) To interpret the features of hemorrhagic strokes.
4) To analyze the features of ischemic strokes.
5) Learn the principles of undifferentiated (basic) and differentiated stroke treatment.
6) Learn the principles of prevention of acute disorders of cerebral circulation.
7) Interpret the modern classification of epileptic and non-epileptic paroxysmal states.
8) Diagnose status epilepticus and provide emergency care.
9) To interpret the main types of cephalgia and the tactics of their treatment.
10) To explain modern ideas about the mechanisms of action of chemical and physical agents on the nervous system.
11) Diagnose neurological manifestations of craniocerebral and spinal trauma.
12) Carry out examination of patients, formulate a preliminary and carry out a differential diagnosis of neurological diseases.
13) Learn the principles of classification of infectious diseases of the nervous system.
14) Master the clinic of the main nosological forms of infectious diseases.
15) Interpret forms of neurosyphilis.
16) Analyze damage to the nervous system in the presence of HIV infection.
17) To learn modern aspects of etiopathogenesis, clinical forms, treatment of demyelinating diseases.
18) Draw up schemes for treatment and prevention of infectious diseases of the nervous system.
19) Learn the principles of formation of vertebral and non-vertebral diseases of the peripheral nervous system.
20) Interpret clinical features in perinatal damage to the nervous system.
21) Analyze the neurological manifestations of hereditary degenerative diseases of the neuromuscular, pyramidal, extrapyramidal, and cerebellar systems.
22) Interpret neurological syndromes in diseases of internal organs, paraneoplastic syndromes.
23) Analyze congenital defects of the spine and spinal cord.
24) Learn the drugs that are used in patients with a neurological profile.

Based on theoretical knowledge of the topic:

Be able:

Carry out examination of patients, formulate a preliminary and carry out a differential diagnosis of neurological diseases.

Topic content:

When examining the patient, first of all, it is necessary to appreciate the following: Complaints of the patient: mainly only those neurological complaints due to which the patient was hospitalized.

Medical history. The onset of the disease, the cause of the disease (infections, injuries, intoxication, tuberculosis, venereal diseases, cooling, psychophysical overstrains, rheumatism, hypertension, diabetes, and others). Development of symptoms of the disease, features of the course, influence of previous therapy, dynamic laboratory and paraclinical data (ECG, EEG, etc.).

History of life. In the history (childhood infections, influenza, tonsillitis, tuberculosis, venereal diseases, craniocerebral and spinal cord injuries, operations). Inherited history, hypertension, diabetes. For women - menstrual cycle (regularity, pain, duration of menopause), pregnancy, childbirth, menopause. Marital status. Harmful habits (smoking, alcoholism, drug use, etc.).

General condition. Body temperature. Position in bed. Face expression. Constitutional features. Subcutaneous fat layer. Skin. Lymph nodes. A hundred joints. Pulse characteristics. Blood pressure. The limits of the heart. Heart activity and tones. Respiratory organs. The Digestive System. Genitourinary system.

Neurological status.

I pair - olfactory nerve (smell of each nostril)

II pair - optic nerve. Visual acuity is checked in accordance with the table, the field of vision is checked by the perimeter or a control method, color perception occurs in accordance with the Rabkin table.

III pair - oculomotor, IV block and VI pair - abductor nerve are checked simultaneously. The position of the eyes, the size, movement of the eyeballs, and the size of the pupils and their uniformity, the quality of the direct and cooperative reaction of the pupils to light (live, lethargic, absent), the presence of the Argyle-Robertson symptom.

V pair - trigeminal nerve (act of chewing, tone of masticatory muscles, corneal and conjunctival reflexes, sensitivity on the face, soreness at the exit points of the trigeminal nerve).

VII para-facial nerve (functions of mimic muscles are checked; taste sensitivity on the front 2/3 of the tongue, hearing, lacrimation).

VIII para-auditory nerve (whisper voice is checked from a distance of 5-6 meters for each ear separately, Rinne and Weber test)

IX pair - glossopharyngeal and X pair - vagus nerves.

Speech sounds, the presence of swallowing and reflexes from the soft palate, the act of swallowing, the taste on the back third of the tongue, the movement of the soft palate during phonation, the position of the tongue and soft palate, the rhythm of respiratory movements and heart activity

XI pair - additional nerve (head position, level of shoulder elevation, head rotation, tone and appearance of m. sternocleidomastoideus and trapezius, fibrillation and fasciculation in them).

XII pair - hypoglossal nerve (tongue mobility, volume of tongue muscles, fibrillation, deviation) deviation of the tongue during its movement. Language articulation.

Motor functions.

Gait (spastic, ataxic, other) of patients. The volume and speed of active movements in all joints. Muscle strength (dynamometry and patient resistance according to a 6-point scale) in all muscle groups. Muscle volume (atrophy is manifested by studying and measuring the volume of the limbs with a tape). The volume of passive movements and muscle tone (hypotonia, hypertension, muscle dystonia). The phenomena of "cogwheel" and "folded knife". Presence of hyperkinesis and their type (chorea, athetosis, myoclonus, tremor, tics, and others).

Coordination of movements (finger-nose and knee-heel test, intentional tremor during movements, adiadochokinesis, Stewart-Holmes test, Babinsky test, Romberg's pose, hyper and dysmetria, asynergia).

Sensory functions.

Palpation of nerve exit points and nerve trunks. Symptoms of nerve and root tension (Neri, Laseg, Bekhterev, Wasserman, Matskevich, etc.). Zones of hypoesthesia (hypo- and anesthesia). Deep sensitivity: joint-muscular and vibrational sensitivity, feeling of pressure and weight.

Complex sensitivity: presence of astereognosis, violation of two-dimensional spatial and discriminative sensitivity. A sense of localization. Hyperpathies and paresthesias.

Meningeal signs (rigidity of the neck muscles, Kernig's sign and symptoms of Brudzinsky and Lesage for young children).

Reflective sphere.

Tendon reflexes (flexor-ulnar, extensor-ulnar, knee, Achilles), periosteal reflexes (carporadial). The level of reflexes, their symmetry, the presence of hypo-, hyper- and areflexia.

Cutaneous reflexes (upper, middle, lower abdominal, plantar and cremasteric in men).

Clonus of the feet and kneecaps. Symptoms of Babinski, Oppenheim, Rossolimo, Zhukovsky. Analogues of pathological reflexes on the hands. Reflexes of oral automatism (Marinescu-Radovichi, nasolabial).

Organs of the small pelvis. Delayed and paradoxical enuresis, imperative urination disorders. Frequent urination. Fixed

Vegetative functions. Sweating (general and local hyperhidrosis), pilomotor reaction. Dermographism (red, white, pink, raised). Asymmetry of skin temperature. Fat secretion.

Oculocardial reflex, tenderness of the solar plexus upon deep palpation, ichthyosis, brittle nails, hirsutism and baldness (alopecia), focal alopecia.

State of consciousness.

Mood. Thinking and intelligence. WARNING. Memory. Orientation in the environment, place, time and own personality. Emotions. Behavior. Delusions, hallucinations, symptoms of obsession and phobia. Sleep. Speech - (motor and sensory aphasia and their characteristics in the patient; slurred speech, dysarthria, stuttering, etc.).

Clinical and laboratory research.

The patient's medical history includes tests of blood, cerebrospinal fluid, urine, and feces; biochemical studies, X-ray examination data (chest X-ray, X-ray of the skull, spine, etc.); data of paraclinical examination methods (EEG, EEG, EMG EMG, CT, MRI and others) diagnoses of consultants (therapist, ophthalmologist, ENT and others).

Summary data on the disease syndrome and symptoms.

Topographic diagnosis.

Based on the data obtained through an objective examination of the patient and the syndrome of the disease, the doctor must mark (show) the localization (topography) of the damage to the brain and spinal cord, peripheral nervous system.

Differential diagnosis.

Differential diagnosis is carried out by comparing similar symptoms of different diseases, and then analyzing the signs on which these diseases are differentiated. After differential diagnosis, a final neurological diagnosis is made. Concomitant diseases unrelated to the main pathology are not taken into account.

Prognosis of the disease.

The prognosis of this disease is given in relation to life, recovery of working capacity, in general, and, in particular, for the controlled patient.

Epicrisis. Epicrisis contains the name and surname of the patient, age, profession, date of admission to the clinic, main complaints, data of objective examination and laboratory tests, which make it possible to make a correct diagnosis. The treatment of this neurological disease and its results, the course of the disease, recommendations for the future and the final clinical diagnosis are shown.

Appendices: a temperature graph, a scheme for researching fields of vision and sensitivity, drawings, schemes and a list of the studied special literature on this disease by the student who treated the patient.

Materials for classroom self-training:

List of educational practical tasks:

1. Methods of research of surface reflexes
2. Methodology of research of deep reflexes
3. Methods of researching pathological foot and hand reflexes
4. Study of reflexes of oral automatism
5. Assessment of muscle strength
6. Methods of detecting signs of peripheral and central paralysis.
7. Techniques for detecting fibrillar twitching and muscle atrophy.
8. Methodology of examination of muscle tone and determination of spastic and plastic muscle hypertension.
9. Methods of detecting extrapyramidal disorders (hyperkinetic-hypotonic and hypokinetic-hypertonic syndromes).
10. Cerebellar function examination technique. Checking the coordination of movements.
11. Methods of detecting static ataxia.
12. Techniques for detecting dynamic ataxia.
13. Methodology of surface sensitivity research.
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18. Methods of researching the functions of the visual analyzer (acuity, field of vision, color perception).
19. Study of the functions of the oculomotor nerves
20. Trigeminal nerve examination technique
21. Methodology of examination of the facial nerve (functions of mimic muscles, taste sensitivity).
22. Methodology of research of the vestibulo-cochlear nerve (hyperacusis and hypoacusis, tests of Rinne, Weber).
23. Methods of research of vestibular functions.
24. Methodology for determining aphasias.
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Recommended literature:

Basic:

- Neurology:textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Herasymchuketal.; editedbyI.A. Hryhorova, L.I. Sokolova. – Kyiv : AUSMedicinePublishing, 2019. – 624 p.

- Netter Atlas of Human Anatomy: Classic Regional Approach: Professional Edition with NetterReference Downloadable Image Bank (Netter Basic Science) 8th Edition By Frank H. Netter MD / Publisher : Elsevier; 8th edition (April 25, 2022). - 712 p. ISBN-10 : 0323793738 ISBN-13 : 978-0323793735

- Neuroanatomy through Clinical Cases 3rd Edition By Hal Blumenfeld / Publisher : Sinauer Associates is an imprint of Oxford University Press; 3rd edition (February 28, 2021).- 1056 p. ISBN-10 : 16053596299ISBN-13 : 978-1605359625

- Pocket Neurology (Pocket Notebook Series) Third Edition By M. Brandon Westover MD PhD Publisher : LWW; Third edition (October 16, 2021). - 390 p. ISBN-10 : 1975169034 ISBN-13 : 978-1975169039

Additional:

- Topical Diagnosis in Neurology. Anatomy, Physiology, Signs, Symptoms / Mathias Baehr, Michael Frotscher (6 edition) – Thieme, 2019 - 332 p.

- Adams and Victor's Principles of Neurology / Allan Ropper, Martin Samuels, Joshua Klein, Sashank Prasad (11th edition). - McGraw-Hill, 2019. - 1664 p.

- Clinical Neuroanatomy Made Ridiculously Simple: Color Edition 6th Edition by Stephen Goldberg M.D. / Publisher: MedMaster; 6th edition (September 14, 2022).- 112 p. ISBN-10 : 1935660519 ISBN-13 : 978-1935660514

- Clinical Neurology and Neuroanatomy: A Localization-Based Approach, Second Edition 2nd Edition by Aaron Berkowitz / Publisher : McGraw Hill / Medical; 2nd edition (July 21, 2022).- 384 p. ISBN-10 : 1260453367 ISBN-13 : 978-1260453362

- Handbook of Neurosurgery 9th Edition by Mark S. Greenberg / Publisher : Thieme; 9th edition (October 23, 2019).- 1784 p. ISBN-10 : 1684201373 ISBN-13 : 978-1684201372

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