Federal State Budgetary Educational Institution
higher education
" NORTH-OSSETIAN STATE MEDICAL ACADEMY "
Ministry of Health of the Russian Federation

DEPARTMENT OF PSYCHIATRY WITH NEUROLOGY, NEUROSURGERY AND MEDICAL REHABILITATION

Topical diagnosis of diseases of the nervous system

Textbook for students of medical, pediatric, medical-preventive and dental faculties of a medical university

Vladikavkaz 2023

Study guide approved at the meeting of the Central Committee for Medical Education of the Federal State Budgetary Educational Institution of Higher Education of the NOSMA of the Ministry of Health of Russia

Recommended for students of medical, pediatric, medical-prophylactic and dental faculties of medical universities

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Neurology is a young medical specialty, rooted in depth and breadth in fundamental medical sciences, such as physiology, anatomy, etc., without the knowledge of which it is impossible to study nervous diseases. The basis of the study of neurology is the propaedeutics of nervous diseases, the knowledge of which allows you to navigate in the topical diagnosis of diseases.

In the methodological work, the authors presented the anatomy and physiology of the nervous system, the theoretical foundations of propaedeutics and topical diagnosis of diseases of the nervous system.

CONTENT

Reflexes and their pathology. Arbitrarily - the motor sphere and its defeat. Paresis and paralysis.

The purpose of the lesson is to study the structural and functional features of the central and peripheral motoneurons, master the methodology for the study of motor functions.

The student should know:

anatomy and physiology of the pyramidal pathway, the structure of the segmental apparatus of the spinal cord and the peripheral nervous system;

reflex arc (for example, the knee reflex);

clinical characteristics of central (spastic) and peripheral (flaccid) paralysis;

modern ideas about the phasic and tonic motor systems, variants of the pyramidal syndrome;

concepts: monoparesis, hemiparesis, triparesis, tetraparesis;

features of disorders of motor functions, depending on the level of damage;

clinical variants of disorders of neuromuscular transmission (syndromes of myasthenia gravis, myoplegia, myopathy, etc.);

basic and additional research methods (dynamometry, electromyography, electroneuromyography);

methods of medicinal rehabilitation therapy of movement disorders in various forms of pathology of the nervous system.

The student should be able to:

To study strength in various muscle groups, muscle tone, periosteal, tendon and superficial (from the skin and mucous membranes) reflexes, pathological signs of the flexor and extensor group, synkinesis, clonuses of the hand, patella, feet, protective reflexes;

Assess the nature of movement disorders in the patient and determine the localization of the pathological process.

Control questions:

1. What is the anatomy and physiology of a central motor neuron?
2. What are the anatomy and physiology of a peripheral motor neuron?
3. What are the signs of central motor neuron damage?
4. What are the signs of peripheral motor neuron damage?
5. What is the semiotics of the lesion of various levels of the central motor neuron (cortex, corona radiata, internal capsule, trunk, spinal cord)?
6. What is the semiotics of damage to the peripheral motor neuron at its various levels (anterior horn, anterior root, plexus, peripheral nerves)?
7. What additional methods are used in the study of the motor system?

REFLEXES AND THEIR CHANGES

The nervous system controls the work of all systems and organs, affects the level of energy processes, and ensures the functional unity of the body. It receives

information about the state of the external and internal environment, stores the information received (memory function), converts this information into regulatory influences. This ensures the interaction of the organism with the external environment, balancing with it and actively overcoming it.

The basis of the functions of the nervous system is reflex activity. "All acts of conscious and unconscious life by the mode of origin are reflexes" (IM Sechenov).

The structural unit of the nervous system is a neuron.

Note that all efferent systems normally function in concert, obeying the principle of subordination (subordination).

CLASSIFICATION OF REFLEXES

All reflexes are subdivided into unconditioned and conditioned; between the one and the other there is a relationship associated with the history of the development of the nervous system.

Unconditioned reflexes are innate and constant for a given type of reaction. They can be simple (for example, withdrawing a limb when applying strong pain or thermal stimuli - a protective reflex) or very complex (such as instincts, automatic actions). In the process of phylogenesis, there was an improvement and complication of the receptor and effector, and especially the combined functions of the reflex systems. Fixing by inheritance, they passed into the fund of unconditioned reflex activity.

Conditioned reflexes arise in ontogenesis in the course of individual development and the accumulation of new skills. The development of new temporary connections depends on changing environmental conditions. Conditioned reflexes are created on the basis of unconditioned ones and are formed in the brain with the indispensable participation of its higher divisions. “The cerebral hemispheres have a special function, the formation of conditioned reflexes, ie. binding with a known physiological activity of such agents that were not previously associated with this activity. All these new connections are formed with the help of innate connections "(IP Pavlov). Thus, unconditioned reflexes, including the simplest ones, are constant components in all complex acts of life.

In humans, there is a huge number of permanent, innate connections and reactions, unconditioned reflexes, carried out through the spinal cord, hind and midbrain, cerebellum, subcortical regions and cerebral cortex. Therefore, a distinction is made between spinal, stem, cerebellar, subcortical, and cortical reflexes.

By the nature of the response, reflexes are divided into motor (with the participation of striated muscles) and vegetative (secretory, trophic, vasomotor, respiratory, gastrointestinal, neuro-endocrine, etc.).

The type of reception determines the release of painful, visual, auditory, gustatory, interoceptive, etc. reflexes. According to the location of the receptors, reflexes can be divided into superficial (skin, from mucous membranes) and deep (tendon, periosteal, muscle proper).

The study of deep and superficial reflexes of the segmental apparatus is of great importance for neurological diagnosis; they are devoted to two sections of this chapter. And in other chapters, we will also get acquainted with reflex functions, since the reflex principle lies at the foundation of all nervous activity.

DEEP AND SURFACE REFLEXES

Deep reflexes cause irritation of receptors embedded in the tendons, periosteum, joints and in the muscles themselves, which is achieved by hitting a neurological hammer or stretching the tendons, muscles, joint capsules.

For example, hitting lig with a hammer. patelae excites the receptors of this tendon, as well as the receptors of the quadriceps muscle itself (due to its slight stretching). The flow of afferent impulses through the femoral nerve, the intervertebral ganglion and the posterior root enters the posterior horn, then switches to the cells of the anterior horn, from which it goes along the anterior roots and motor fibers of the femoral nerve to the same quadriceps muscle, causing its contraction.

Many deep reflexes have a two-neuronal reflex arc.

Superficial reflexes cause irritation of the skin or mucous membranes by touch (with a cotton swab, brush, paper), a stroke, an injection or thermal effects (hot, cold).

So, with streak irritation of the abdominal skin, the abdominal muscles on their side contract.

Reflex arcs of superficial reflexes contain intercalary neurons.

The direction of the strokes when examining the reflexes of the abdominal and cremaster.

In a person, the number of unconditioned reflexes that can be caused by certain stimuli is quite large. The ones that deserve the greatest attention are those that are distinguished by significant constancy. Therefore, we will confine ourselves to considering only that small part of the reflexes that is constantly studied in neurological practice and is included in the mandatory program for the study of the nervous system. In our presentation, we will adhere to the order of research from top to bottom, denoting the name of the reflex, the nature of the reaction, the reflex arc and the method of evocation.

The superciliary reflex is triggered by striking the hammer along the edge of the superciliary arch. Refers to deep, periosteal reflexes. The response is the closure of the eyelids (m.orbicularis oculi).

Reflex arc: n.ophtalmicus (1 branch of the trigeminal nerve), sensory nucleus of the trigeminal nerve, motor nucleus n.facialis. The corneal reflex is obtained by gently touching the cornea with a cotton swab or soft paper over the iris (but not over the pupil); the reflex is superficial, from the mucous membrane. The motor reaction consists in the same closing of the eyelids, the reflex arc is the same as in the superciliary reflex.

The mandibular, or mandibular, reflex (ankylosing spondylitis) is triggered by tapping the chin with a hammer or a spatula placed on the lower teeth with a slightly open mouth. The reflex is deep, periosteal. The response is a contraction of

the masseter muscles (m. Masseteres), which causes the jaws to close (raising the lower jaw). Reflex arc: sensitive fibers n.mandibularis. (III branch of the trigeminal nerve), the sensory nucleus of the trigeminal nerve, its motor nucleus in the bridge. The reflex is not very constant in the norm and sharply increases in pathological cases (with pseudobulbar paralysis).

The pharyngeal reflex is triggered by touching the back wall of the pharynx with a rolled-up piece of paper; there are swallowing, sometimes coughing and gagging. Reflex arc: sensory fibers and the nucleus of the IX and X nerves (nn.glosspharyngeus and vagus), the motor nucleus and the fibers of the IX and X nerves.

The soft palate reflex (palatal) results from touching the soft palate; the response is to raise the latter and the uvula. The reflex arc is the same as for the pharyngeal reflex.

The pharyngeal and palatal reflexes are not very consistent and may be absent in healthy individuals. A unilateral decrease or absence of a reflex is diagnostically valuable; therefore, it is necessary to touch separately the right and left sides of the mucous membrane of the palate and pharynx. Both reflexes are superficial, from mucous membranes.

Flexor flexor, or reflex from the tendon m.bicipitis, is caused by a hammer blow on the tendon of the biceps muscle in the elbow bend. The response is to contract the named muscle and flexion in the elbow joint. Reflex arc: n.musculocutaneus, V and VI cervical segments of the spinal cord. Deep, tendon reflex. To evoke it, the examiner takes the subject's hands with his left hand and bends his arms at the elbow joints. The musculature of the arms should be relaxed, not tense. Ular is applied with a hammer shortly, abruptly and, moreover, exactly along the tendon of the biceps muscle (you can first feel it with your finger). It is clear that the blow should be of the same force when examining the right and left hands. Sometimes it is beneficial to feel the tendon with the large palnom of the left hand, press it with the pulp of the terminal phalanx and apply a blow with a hammer to the thumbnail of the examiner. In this case, the researcher's forearm rests on the researcher's left forearm.

The extensor, or reflex from the tendon n.tricipitis, is obtained as a result of a blow with a hammer on the tendon of the triceps muscle, which causes its contraction and extension of the forearm in the elbow joint; the blow is applied 1.5-2 cm above the olecranon. The reflex is investigated as follows: the hand is grasped by the examiner's left hand, the subject's hand is not tense and bent at the elbow joint at a right angle, sometimes (better) at a slightly obtuse angle. You can explore the reflex in another way: take the subject's hand by the shoulder slightly above the elbow and hold it, requiring complete relaxation of the muscles; the forearm and hand should hang freely; flexion at the elbow joint at a right or slightly obtuse angle; this is followed by a hammer blow over the olecranon.

The metacarpal, or carporadial, reflex is caused by the ular of the malleus along the processus stiloideus and consists in flexion in the elbow joint, pronation and flexion of the fingers. Not all of the named reactions are obtained constantly: pronation is usually most clearly expressed. When evoking a reflex, the subject's

arm should be bent at a right or slightly obtuse angle at the elbow joint, the hand should be in the middle position between supination and pronation. The hand is either grasped by the examiner's left hand and held in weight, or both hands are freely and symmetrically positioned in the desired position on the hips of the seated subject.
spinal cord. The scapular-shoulder reflex (ankylosing spondylitis) is obtained by striking the inner edge of the scapula with a hammer: adduction and rotation outside the shoulder are noted, which should hang freely.
Superficial abdominal reflexes are caused by dashed irritation of the skin of the abdomen: below the costal inflation - the upper abdominal reflex, at the level of the navel - the middle abdominal reflex, above the pupar ligament - the lower abdominal reflex.
Stroke irritations should be quick, applied with a somewhat pointed object (quill pen, match).
Deep abdominal reflexes are caused (according to A.V. Triumfov) by tapping with a hammer on the pubis, 1 - 1.5 cm to the right and left of the midline; the result is a contraction of the corresponding side of the abdominal wall.
A deep abdominal (periosteal) reflex is the osteo-abdominal reflex described by V.M. Bekhterev; contraction of the abdominal muscles occurs as a result of tapping with a hammer along the edge of the costal arch, somewhat inside from the nipple line.

The cremaster reflex, or testicular, is caused by streak irritation of the skin of the inner surface of the thigh; there is a contraction of m.cremasteris and pulling up the testicle of the corresponding side. The knee reflex, or patellar, is obtained by striking the lig with a hammer. patellae below the patella, resulting in shin contraction and extension. Knee reflexes are best examined when the patient is supine. It is more convenient to approach the subject from his right side; the left hand is brought under the knee joints of the subject's legs bent at an obtuse angle; while his feet are resting on the couch, the muscles of the legs should be relaxed. In this position, they strike with a hammer with the right hand on the patellar tendon on the right and left. In the position of the patient on his back, one can examine the knee reflexes separately, with one leg thrown over the other; the intensity of contraction of the quadriceps muscle is determined by placing the left hand on the thigh on top. Finally, knee reflexes can be examined while the subject is sitting; the legs should hang freely over the edge of the couch or bed and be at right angles to the thighs; feet should not rest on the floor.
Sometimes knee reflexes are caused with difficulty due to the inability of the patient to sufficiently relax the muscles of the legs. In such cases, the Endrashik technique is usually used: the subject is invited to clasp the fingers of both hands and forcefully pull the hands to the sides; you can offer the patient to clench their teeth, count, and ask him questions to distract attention.

The Achilles reflex is triggered by striking the Achilles tendon with a hammer; there is a reduction in m.tricipitis and flexion of the foot. The best way of research is as follows: the patient kneels on a couch or chair so that his feet hang freely over the edge without tension; hands against the wall or the back of a

chair. You can examine the patient and in a supine position, on the stomach; both feet are grasped by the fingers and held in a bent at a right angle in the ankle joints with the examiner's left hand (it is more convenient to approach from the patient's right side), after which successive hammer blows are applied to the left and right Achilles tendon.

The plantar reflex is obtained in response to streak stimulation, which is applied with the handle of a malleus or with a pointed object to the inner or, better, the outer edge of the sole.

The direction of the stroke can be from the bottom or from top to bottom; it is better to carry out the stroke with some pressure, with an increase in it towards the end of the irritation. The response is flexion of the toes, and with a high reflex with flexion in the knee and hip joints, withdrawal of the leg. The study is performed on a patient lying on his back; the leg lies calmly on the couch or is held by the examiner's left hand in a slightly bent position.

The plantar reflex in the form in which it is described here and which is inherent in the intact human nervous system, starting from persons who have reached the age of 1-1.5 years, is a type of reaction modified in connection with the development of the cerebral cortex and the simultaneous development of a vertical body position and walking. In small children, there is, and in patients with a damaged pyramidal system (disconnection from the cerebral cortex), that type of plantar reflex, or the Babinsky phenomenon, arises, which will be discussed below.

The anal reflex is triggered by a prick in the skin near the anus; its circular muscle contracts.

CHANGES IN REFLEXES

Reflexes can change in the direction of: 1) lowering or losing them; 2) enhancements and 3) perversions. Finally, in pathological conditions, new reflexes may appear that are not normally evoked.

Pathological reflexes. This group includes perverted reflexes or those that do not normally exist and are caused only in pathological conditions, i.e. with damage to the nervous system.

The pathological reflexes carried out by the oral muscles include the following.

Nasolabial reflex, caused by tapping with a hammer on the bridge of the nose. The response is to contract the orbicularis oris, the orbicularis muscle of the mouth (pulling the lips forward).

The proboscis reflex is the same reaction that occurs when a hammer is struck on the upper or lower lip.

The sucking reflex is obtained as a result of touching the lips or stroking them; sucking movements with the lips are observed in response.

The distance-oral reflex, the clinical significance of which was emphasized by S.I. Karchikyan, is caused not by direct irritation of the lips, but only by the approach to the mouth of the sick malleus: even before the impact, a "proboscis" extension of the lips occurs forward

The palmar-chin reflex (Marinescu-Radovicha) is caused by dashed irritations of the skin of the palm over the thenaris, a contraction is obtained on the m.mentalis on this side with an unsharp displacement of the skin of the chin upward.

The listed "oral" reflexes are rarely observed in healthy adults; they are normally found in newborns and infants. Their appearance is characteristic of the so-called pseudobulbar paralysis, when, due to the disconnection of the reflex centers from the cerebral cortex, the inhibitory effects of the latter on the automatic sucking reactions associated with the segmental apparatus of the brain stem fall out. They are also observed in people of old age.

The group of oral reflexes refers to the so-called "axial" reflexes, i.e. to those types of motor reactions that are caused from the "axis" of the body (head, neck, trunk). Together with other axial reflexes, oral reflexes can be increased in the so-called Parkinsonian syndrome, i.e. with extrapyramidal lesions

A number of new, pathological reflexes may appear on the upper limbs with damage to the pyramidal pathways.

Carpal pathological reflexes can be found in the mildest lesions of the pyramidal system. The upper reflexes of Rossolimo, Zhukovsky and Bekhterev-Mendel, the reflexes of Hoffmann, Jacobson-Lask, symptoms of Mayer and Venderovich (motor ulnar defect) are of practical importance. The most constant of them is Rossolimo's reflex, it is better to induce the Venderovich technique: with the supinated hand of the examined person, the blow is applied to the ends of the slightly bent II-V fingers.

The main and practically very important group of pathological reflexes is made up of pathological reflexes found on the foot. The cardinal ones are the following.

Babinsky's symptom is a perverted plantar reflex, or a symptom of extension of the thumb. Normally, with streak stimulation of the sole, reflex flexion of all five fingers is obtained. In pyramidal lesions, the same irritation causes extension of the thumb, sometimes isolated, sometimes with simultaneous dilution of the remaining fingers ("fan sign"). The symptom is very constant with central paralysis and paresis; is one of the earliest and most subtle manifestations of the violation of the integrity of the pyramidal pathway in the brain or spinal cord above the segments of the reflex arc of the plantar reflex.

Symptom Rossolimo - reflex flexion of the II-V toes as a result of a short blow to the tips of the said fingers with the examiner's fingers or a hammer.

Ankylosing spondylitis - Mendel's symptom - the same flexion of the fingers when tapping with a hammer on the front of the non-outer surface of the rear of the foot.

Zhukovsky's symptom is caused by a hammer blow on the sole under the toes; the response reflex movement is plantar flexion of the II-V fingers.

Oppenheim's symptom results from pressing with the flesh of the thumb over the anterior surface of the tibia from top to bottom; the phenomenon consists in the same extension of the thumb as in Babinsky's symptom.

Gordon's symptom is the same reflex extension of the thumb, but as a result of squeezing the hand of the examining the mass of the gastrocnemius muscle.

Schaeffer's symptom - reflex extension of the thumb with pinched irritation or strong compression of the Achilles tendon;

Hirschberg's symptom is flexion and inward rotation of the foot, caused by dashed irritation of the inner edge of the sole.

Pusepp's symptom - abduction of the V toe in case of streak irritation of the outer edge of the foot. Described as a symptom of extrapyramidal paresis; in our opinion, it is a particular (incomplete) manifestation of the "sign of the fan", i.e. a symptom of pyramidal lesions.

The pathological reflexes indicated here, found on the foot, are characteristic of lesions of the pyramidal system and represent the forms of reactions of the underlying motor apparatus, disconnected from the cerebral cortex. Normally, these reflexes are observed in children up to the age when the vertical position of the body and the function of walking are developed, i.e. up to 1-1.5 years. In connection with the development of the cerebral cortex, the old types of reactions are modified and can be revealed again in their former form when the underlying devices are released from the influences of the cerebral cortex regulating and rearranging their activity (subordinating).

Here are the main types of pathological foot reflexes, which are practically more valuable and are of the greatest importance in clinical practice. A large number of other pathological foot phenomena have been described: they differ from those described usually only in the place and nature of the stimulation applied. Responses are reduced mainly to the same dorsal flexion of the thumb or flexion of the remaining fingers.

Defense reflexes are also one of the symptoms of pyramidal tract damage. They are especially pronounced in transverse lesions of the spinal cord (separation of the underlying segments of the latter from the brain). They are best caused by a pinch, as well as an injection (sometimes only a series of consecutive injections) or a sharp plantar flexion of the toes, produced by the researcher (V.M. Bekhterev). They are characterized by a rather long latent period (slow response); the paralyzed lower limb with the above stimuli "pulls back", involuntarily bending in the hip, knee and ankle joints ("shortening reaction"); the opposite limb (previously bent) is straightened, unbending at the joints ("elongation reaction"). By alternately applying irritation to one or the other leg, you can get an imitation of the "automatism" of walking.

Protective reflexes can be of some importance in determining the level of transverse lesions of the spinal cord: sometimes they can be obtained with irritations applied to the entire surface of the body below the location of the spinal cord interruption (compression by a tumor, trauma, etc.). It should be noted that it is still difficult to obtain precise indications of the lower border of the transverse lesion of the spinal cord based on the study of only protective reflexes, if any.

Tonic, or postural, reflexes (reflexes of position) are manifestations of a special complex innervation system that automatically regulates muscle tone to maintain body position.

There are mechanisms that reflexively regulate balance, both at rest and in motion. The redistribution of muscle tone largely depends on a change in position in the head space (labyrinthine and cervical tonic reflexes). Experiments on animals have shown that the vestibular and red nuclei in the brain stem are of major importance in the regulation of muscle tone when standing and

walking. Normally, tonic reflexes are largely inhibited by the higher parts of the brain and are revealed with particular intensity when they are turned off.

A weakening or strengthening of postural reflexes with damage to certain parts of the nervous system can also be observed in humans, but not with such constancy and regularity as in experimental animals. Symptoms that can be considered a manifestation of disorders of postural reflexes and are somewhat more consistent include the following:

1. The subject stretches: hands forward and with closed eyes makes the maximum turn of the head to the right, while both hands are slightly deviated to the right, and the right hand, in addition, is also raised somewhat upward. Corresponding hand position change
occurs at the next, then the task to turn the head to the left.

The phenomenon is enhanced on the side where there are cerebellar disorders, and weakened or lost when the extra pyramidal system is affected.

2. Automatic raising of the arm (more than the right one) occurs after a while, if the subject is asked to keep his arms outstretched
forward with closed eyes. An increase in lifting occurs in the case of a brain
gallbladder lesion on the side corresponding to the focus.

3. Pronation phenomenon. With eyes closed, the hands are embroidered forward in a full supination position (palms up); slight pronation gradually develops. Strengthening of the phenomenon is observed in pyramidal and cerebellar lesions.

Without citing other (very numerous) symptoms of the manifestation of postural reflexes, we point out that they have not acquired great importance in the clinic of nervous diseases and have not received widespread use in the study of the nervous system.

Movement, paralysis and paresis

The human motor function seems to be extremely complex. A number of parts of the nervous system are involved in the implementation of movements. In some cases, the movements are primitive, occur involuntarily according to the type of a simple reflex act and are carried out due to the activity of the segmental apparatus (spinal cord, brain stem). The reflexes discussed above are an example of such simple automatic movements in humans. Movements resulting from cortical innervation, developed in life experience and are, in essence, conditioned reflexes, are usually called "voluntary"; automatic reflex movements - "involuntary".

For the implementation of "voluntary" movement, it is necessary, in particular, that the impulses arising in the cerebral cortex should be conducted to the muscle. Conduction of an impulse from the cortex occurs along a chain consisting of two neurons: 1) a central motor 2) a peripheral motor neuron. The entire path is called cortico-muscular.

The central motor neuron starts from the area of the cortex located anterior to the Roland groove in the anterior central gyrus, in the posterior parts of the superior and middle frontal gyri.

Nerve cells that provide fibers for the innervation of individual muscle groups have an arrangement opposite to the location of parts of the human body: the projection

of movements of the lower limb - in the upper parts of the anterior central gyrus, the upper limb - in its middle part, and the head, face, tongue, pharynx and larynx - in the bottom. In the posterior section of the superior frontal gyrus, the projection of trunk movements is presented, and in the posterior section of the middle frontal gyrus, the rotation of the head and eyes in the opposite direction. The innervation of the muscles is cross, i.e. the right hemisphere is connected with the muscles of the left half of the body, and the left hemisphere is connected with the right (the cross of the conductors, which will be discussed below).

The central motor neuron originates from the large Betz pyramidal cells located in the fifth layer of the cortex of the projection motor zone.

The inner capsule is located between the large ganglia of the base; it is a strip of white matter in which there are conductors going from the cerebral cortex to the lower parts of the central nervous system, and ascending conductors that ascend to the cortex. In their downward direction, both of these conductors pass from the inner capsule into the legs of the brain, occupying the middle two-thirds of their base. In the bridge, the motor conductors are also located at the base. Passing here transversely and crossing fibers of the middle legs of the cerebellum, the pyramidal pathways are divided into a number of separate bundles, merging again into a common conductor in the medulla oblongata.

In the medulla oblongata, the pyramidal bundles are located at the base, separating into two ridges visible on the surface of the brain - pyramids

On the border of the medulla oblongata and the spinal cord, the pyramidal bundles undergo incomplete intersection. The large, crossed part of the path passes into the lateral column of the spinal cord and is called the main, or lateral, pyramidal bundle, the smaller, uncrossed part passes into the anterior column of the spinal cord and is called the direct uncrossed bundle. The fibers end in the motor nuclei of the cranial nerves - in the anterior horns of the spinal cord. Here, impulses from central motor neurons are transmitted to peripheral ones. Peripheral motor neurons for the muscles of the chewing, facial, tongue, larynx and pharynx consist of motor cells of the motor nuclei of the cranial nerves with their axons that make up the motor fibers of the roots and nerves V, VII. IX, X and XII (trigeminal, facial, glossopharyngeal, vagus, sublingual). Peripheral motor neuron cells for the muscles of the neck, trunk, limbs and perineum are located in the anterior horns of the spinal cord; their axons go out as part of the anterior motor roots and make up the motor fibers of the peripheral spinal nerves. The cortical innervation of the muscles is predominantly cross, since the fibers of the central motor neurons for the most part move to the opposite side and establish connections with the opposite nuclei of the cranial nerves and the anterior horns of the spinal cord.

The crossing of fibers, tractus corticospinalis occurs immediately on the border of the medulla oblongata and the spinal cord; the fibers are crossed sequentially, as they pass to the corresponding nuclei of the cranial nerves ("supranuclear" crossover).

With the defeat of the central motor pathways, movement disorders, of course, arise in the muscles of the opposite side of the body, but not in all: a number of muscle groups remain unaffected. This is due to the presence of bilateral cortical

innervation that exists for the oculomotor, masticatory muscles, muscles of the pharynx, larynx, neck, trunk and perineum. The bottom line is that to a part of the motor nuclei of the cranial nerves and to some levels of the anterior horns of the spinal cord, the fibers of the central motor neuropes approach not only from the opposite side, but also from their own, thus providing the approach of impulses from the cortex not only of the opposite, but also your hemisphere. It is clear that with a unilateral central lesion, the functions of only muscle groups that are unilaterally (only from the opposite hemisphere) innervated are lost. These are the limbs, tongue and lower part of the facial muscles, which we observe in the so-called hemiplegia; the muscles of the neck, trunk. the perineum and most of the mouse, innervated by the cranial nerves, remain unaffected.

For the implementation of the movement, it is necessary that the motor impulse be freely conducted from the cerebral cortex to the muscle. When the cortico-muscular pathway is interrupted, the impulse cannot be conducted and the corresponding muscles are in a state of paralysis. Incomplete loss of movement (a decrease in their strength and volume) is called not paralysis, but paresis.

According to their prevalence, paralysis is divided into monoplegia (one limb is paralyzed), hemiplegia (paralysis of one half of the body), paraplegia (damage to two symmetrical limbs, upper or lower); tetraplegia (all four limbs are paralyzed).

Paralysis or paresis caused by damage to individual nerve trunks is designated as paralysis of the corresponding nerve, for example, radial, ulnar, etc. Similarly, paralysis of the plexuses (brachial, lumbar) or their individual trunks is distinguished.

With damage to the central motor neurons, central paralysis occurs; with damage to peripheral neurons - peripheral. Common to them is only the very fact of paralysis, otherwise their symptomatology is sharply different.

PERIPHERAL PARALYCH

Peripheral paralysis is, as just said, the result of damage to peripheral motor neurons. those. cells of the anterior horns of the spinal cord (or motor nuclei of the cranial nerves), anterior roots and motor fibers of the spinal and cranial peripheral nerves. This type of paralysis is characterized by loss of reflexes, hypotension and degenerative muscle atrophy, accompanied by the so-called rebirth reaction.

The loss of reflexes (or their weakening with incomplete damage) becomes clear if we remember that the peripheral motor neuron is at the same time a centrifugal, efferent part of the reflex arc. When any part of the latter is interrupted, the reflex act is impossible or (with an incomplete interruption) is weakened.

Atony or hypotonia of the muscles is also explained by the interruption of the reflex arc, as a result of which the muscle loses its characteristic constant, so-called contractile, tone, which is normally maintained by the same reflex arc with the participation of the gamma loop. In addition, atony can be exacerbated by the resulting atrophy of muscle mass. Atonic muscles feel flabby, sluggish, passive movements are excessive, joints are "loose". This condition of the musculature gives reason to call peripheral paralysis also flaccid, or atonic.

Muscle atrophy occurs as a result of dissociation with the anterior horn cell, from where neurotrophic impulses flow to the muscle along the motor nerve fiber,

stimulating the normal exchange of muscle tissue. The presence of muscle atrophy leads to another definition of peripheral paralysis - as atrophic.
Mouse atrophy follows the degeneration and death of the nerve motor fibers, and muscle denervation occurs. As a result, motor fibers disappear in the nerves from top to bottom; a degenerative process develops in the muscle, characterized by changes in muscle fibers, their death, the development of adipose and connective tissue. There are changes in the electrical reactions of the affected nerves and muscles, characteristic of peripheral paralysis, called the reaction of degeneration or degeneration (RD).
With incomplete damage to the peripheral motor neuron, a partial reaction of degeneration may occur, when the excitability of the nerve to both currents is not lost, but only weakened, as well as the faradic excitability of the muscle; contraction of the same muscle during irritation with galvanic current also occurs slowly.
A complete reaction of rebirth is not yet a bad prognostic sign: provided that the nerve fiber is restored (regenerated), it can be replaced by normal electroexcitability through the phase of a partial reaction. But if a muscle with peripheral paralysis remains completely denervated for more than 12-14 months (sometimes longer), then as a result of progressive degeneration of muscle fibers, they die completely, are replaced by adipose and connective tissue, and cirrhosis of the muscle sets in with the loss of its reaction to galvanic current , i.e. complete loss of electroexcitability develops. The latter indicates the irreversibility of the changes that have occurred in the muscle. The degeneration reaction is observed with atrophy, which develops as a result of damage to the peripheral motor neuron. Other atrophic processes in the muscles. The study of the reaction of degeneration has a certain value in the clinic and allows for differential diagnosis of muscle atrophy of various nature. In addition, the study of electroexcitability makes it possible to establish early the diagnosis of nerve conduction disorders, muscle contractility and makes it possible to judge the dynamics of the process,
reactions. With myotonia, the excitability of the nerve remains normal, while the muscle relaxes extremely slowly after the resulting contraction. For myasthenia gravis, extreme muscle fatigue is characteristic, which manifests itself in the rapid depletion of its contractile ability with repeated stimulation of the current.
Peripheral paralysis, as mentioned above, is the result of damage to either the anterior horns of the spinal cord, or its anterior roots, or plexus trunks, or, finally, the peripheral nerves themselves (also the motor nuclei of the cranial nerves, their roots and the cranial nerves themselves). To resolve the issue of localization and distribution of the process that caused peripheral paralysis, it is necessary to know the pattern of innervation of movements and muscles by segments of the spinal cord and individual nerves,

CENTRAL PARALYCH

Central paralysis occurs as a result of damage to the central motor neuron in any of its parts. Since the arrangement of the cells and fibers of the pyramidal bundles is rather close, the central paralysis is usually diffuse, spreading to the whole limb or half of the body. Peripheral paralysis can be limited to the defeat of some muscle

groups or even individual muscles. However, there may be exceptions to this rule. So, a small focus in the cerebral cortex can cause the occurrence of an isolated central paralysis of the foot, face, etc.; conversely, multiple diffuse lesions of the nerves or anterior horns of the spinal cord sometimes cause widespread paralysis of the peripheral type.

As mentioned above, the symptomatology of central paralysis differs sharply from that of peripheral paralysis: pronounced muscle atrophy is not characteristic here and there is no degeneration reaction, neither mouse atony nor loss of reflexes is observed.

Slight diffuse muscle atrophy can sometimes be observed with central paralysis (for example, with lesions of the parietal lobes), but it never reaches such a degree as with peripheral paralysis, and is not accompanied by the reaction of degeneration typical of the latter. This atrophy may be the result of a lack of activity in the mouse, but sometimes it develops early, following a lesion; in this case, it can be explained as a trophic disorder resulting from damage to the cortex. In cases of acute central paralysis (trauma, hemorrhage), muscle hypotonia and loss of reflexes are possible at first.

The absence of disorders characteristic of flaccid paralysis is understandable, since the peripheral motor neuron (and the segmental reflex arc) with central paralysis remains intact; consequently, there are no symptoms depending on its defeat. The segmental apparatus of the spinal cord that remains intact retains its reflex activity. When the pyramidal system is damaged, the inhibitory effects of the cerebral cortex do not reach the segmental apparatus.

The main features of central paralysis are mouse hypertension, increased deep reflexes, the so-called concomitant movements (or synkinesis) and pathological reflexes.

Hypertension, or muscle spasticity, defines another name for central paralysis - spastic. Muscles are tense, tight to the touch; with passive movements, a clear resistance is felt, which can sometimes be overcome with difficulty. This spasticity is the result of increased reflex tone and is usually unevenly distributed, resulting in typical contractures. With central paralysis, the upper limb is usually brought to the trunk and bent at the elbow joint; the hand and fingers are also in a flexion position. The lower limb is extended at the hip and knee joints, the foot is bent and turned by the sole inward (the leg is extended and "lengthened"). This position of the limbs with central hemiplegia creates a kind of Wernicke-Mann posture.

An increase in deep reflexes (hyperreflexia) is also a manifestation of increased, disinhibited, automatic activity of the spinal cord. Reflexes from the tendons and periosteum are extremely intense and easily triggered by even minor irritations.

Concomitant movements, or synkinesis, observed with central paralysis, can occur reflexively in the affected limbs, in particular, with the tension of healthy muscles. Their occurrence is based on a tendency to irradiation of excitation in the spinal cord to a number of adjacent segments of its own and opposite sides, which is normally moderated and limited by cortical influences. With disinhibition of the segmental apparatus, this tendency to propagate excitement is revealed with

particular force and causes the appearance of "additional" reflex contractions in the paralyzed muscles.
There are a number of synkinesis associated with central paralysis. Here are some of them:
if the patient, on assignment, resists the extension in the elbow joint produced by the examiner with his healthy hand, or strongly shakes his hand with his healthy hand, then in the paralyzed hand , concomitant reflex flexion occurs .
Also flexing the affected hand occurs when coughing, sneezing, yawning.
Under these conditions, in the paralyzed leg (if the patient sits with the legs hanging over the edge of the couch or table), involuntary extension is observed;
Lying on his back with outstretched legs patient offer lead and take a healthy pace in what he resist. In the paralyzed leg, an involuntary corresponding adduction or abduction is observed .
Pathological reflexes are a group of very important and persistent symptoms of central paralysis. Of particular importance are pathological reflexes on the foot, which are observed, of course, in cases where the lower limb is affected. The most sensitive are the symptoms of Babinsky (perverted plantar reflex), Rossolimo and Bekhterev-Mendel. The rest of the pathological reflexes on the foot (see above) are less constant. Pathological reflexes on the hands are usually less pronounced. Pathological reflexes on the face (mainly a group of "oral" reflexes) are characteristic of central paralysis or paresis of the muscles innervated by the cranial nerves, and indicate bilateral supranuclear lesions of tractus corticonuclearis in the cortical, subcortical or trunk regions.
Symptoms such as increased tendon reflexes of the extremities, weakening of abdominal reflexes and Babinsky's symptom are very subtle and early signs of a violation of the integrity of the pyramidal system and can be observed when the lesion is not enough for the occurrence of the paralysis itself or even paresis. Therefore, their diagnostic value is very high. EL Venderovich described the symptom of "ulnar motor defect", indicating a very mild degree of pyramidal lesion: on the affected side, the patient's resistance to forced abduction towards the little finger, which is maximally reduced to the fourth finger, is weaker.
The methodology for the study of movements consists of: 1) studying the general appearance, facial expressions, speech, posture and gait of the patient; 2) determining the volume and strength of active movements; 3) checking passive movements and muscle tone; 4) studies of coordination of movements; and 5) testing of electrical excitability of nerves and muscles.
Already one external examination of the patient can give a lot of essential and direct the attention of the investigator to a particular defect in the state of the musculature and motor function. So, atrophy of the mouse, contractures of the extremities can immediately be established. Sometimes the patient's posture, low or, conversely, excessive mobility, draw attention to themselves. In a conversation with a patient, paresis of mimic muscles, speech disorders, phonation disorders can be noticed. Trembling, convulsive twitching, etc. are noticeable. The patient's gait, which may be upset, is necessarily examined.

In particular, with hemiparesis of the central type, there is a "hemiplegic, circumferential" gait, the Wernicke-Mann posture, as mentioned above. With spastic lower paraparesis, there is a "spastic" or "spastic-paretic" gait, when the patient walks with straightened legs, slightly lifting the soles from the floor; during the movements of the legs, the tension existing in them is noticeable. With flaccid paraparesis, the feet usually hang down, and the patient, in order not to touch the floor with his toe, is forced to raise his leg high (the so-called “cock”, or peroneal, gait).

Active movements are examined in order from top to bottom; usually the volume of only some of the basic movements is determined.

In raspberries, wrinkling of the forehead upward, closing of the eyelids, movements of the eyeballs, opening the mouth and pulling the corners of the mouth outward, protruding the tongue are investigated.

The volume of head turn to the sides is determined. It is offered to the subject to make the movement of raising the shoulders ("shrug" the shoulders). The arms are raised to the horizontal and higher; flexion and extension in the elbow, wrist and finger joints; pronation and supination of the hands; pinching and spreading fingers; to determine the mild degree of paresis and disorder of fine movements, it is advisable to invite the subject to make quick flexion and extensor movements with the fingers, fingering them in the air with the arms extended forward. This is followed by flexion and extension of the trunk, tilt to the right and left.

Flexion and extension in the joints of the hip, knee, ankle, finger, walking on heels and on toes are performed.

In some cases, it is necessary to check more subtle and isolated movements concerning individual mice.

The presence of a full range of active movements does not always exclude the possibility of the existence of mild paresis, which in such cases may be limited by the weakening of muscle strength. Therefore, the study of the range of active movements of the limbs is usually accompanied by a simultaneous study of muscle strength, for which the subject has a certain resistance to the produced movement. Determined, in particular, the force of compression of the hand, which can be measured with a dynamometer.

Passive movements, of course, will not be limited if there is a full range of active movements. Their study is necessary when establishing the absence or limitation of active movements in a particular muscle group. It may turn out that movements are limited not due to paresis, but due to joint damage, pain, etc. The study of passive movements is also carried out in order to determine muscle tone. The tone is determined, first of all, by the palpation of the muscle at rest. With atony or hypotension, the muscles are flabby, sluggish to the touch, with hypertension - dense, tense. With passive movements in the case of atony, excursions in the joints are completely free, even redundant; the joints are loose. With an increase in tone, passive movements encounter significant resistance, which requires a certain amount of tension to overcome. With spasticity of the muscles accompanying central paralysis, there is a condition called the "jackknife symptom"; if we make a quick passive movement, then the resistance exerted by the rigid muscles is not the

same throughout the movement; it is especially felt at the beginning and decreases later.
Coordination of movements is impaired as a result of damage to the cerebellar system and with the loss of "a sense of position and movement" (joint-muscle sense).

SYMPTOMIC COMPLEXES OF DISORDERS IN DIFFERENT SECTIONS OF THE MOTOR WAYS

I. Damage to a peripheral nerve causes peripheral paralysis in the area of the mouse innervated by this nerve. Since the vast majority of nerves are mixed, i.e. has not only motor, but also sensory fibers, then in this case, in addition to paralysis, pain and sensory disorders are also observed.
P. The defeat of the cervical, brachial, lumbar and sacral plexuses also gives a combination of peripheral paralysis, pain and sensory disorders.
Symptoms of damage to individual plexus trunks correspond to the pattern of damage to the peripheral nerves emanating from them (in a certain combination).
III. The defeat of the anterior horns and anterior roots of the spinal cord (equally the motor nuclei of the cranial nerves) causes only peripheral paralysis without pain and without sensory disturbances. The prevalence of movement disorders corresponds to the affected segments. In chronic progressive processes, fibrillar or fascicular twitching in atrophying muscles is usually observed.
With the defeat of other parts of the motor tract, consisting of central motor neurons, central paralysis is observed.
IV Defeat of the lateral column of the spinal cord with a pyramidal bundle passing through it causes diffuse (downward from the level of the lesion) central paralysis of the muscles on the side of the focus. If the process is localized in the thoracic region, then paralysis of the leg is observed; with the defeat of the pyramidal bundle above the cervical thickening - central paralysis of the arm and leg. Simultaneously with the aforementioned movement disorders, with the defeat of the lateral column, there is a loss of pain and temperature sensitivity on the opposite side of the body.
V. A transverse lesion of the spinal cord gives central paraplegia of the lower extremities (bilateral lesion of the pyramidal bundles) - when localized in the thoracic region, or tetraplegia, i.e. defeat of all four limbs, - with higher (upper cervical) localizations.
Vi. The defeat of the pyramidal bundle in the brainstem (medulla oblongata, pons pons, cerebral peduncles) gives central hemiplegia already on the opposite side, since the pyramidal pathways below, on the border with the spinal cord, intersect. Usually, in this case, the nuclei of the cranial nerves located here are involved in the process. This creates a picture of the so-called alternating (cross) paralysis: on the side of the focus - the defeat of certain cranial nerves, on the opposite side of the central hemiplegia.

Vii. The defeat of the pyramidal fibers in the inner capsule causes central hemiplegia on the opposite side of the body, in the same place is the central paresis of the lower part of the facial muscles and tongue.
With lesions in the inner capsule, the Wernicke-Mann posture is especially often observed. The defeat of the motor projection area in the anterior central gyrus of the cerebral cortex also causes central paralysis on the opposite side of the body. Unlike capsular lesions, here more often than not hemiplegic, but monoplegic type is observed, i.e. predominant loss of function of the arm or leg.

Sensitivity and senses.

The purpose of the lesson is to study the pathways of the afferent systems, to master the methods of studying various types of sensitivity and pain syndromes.

The student should know:

classification of receptors, pathways of general sensitivity;

the visual hillock as a collector of all types of sensitivity, the principle of somatotopic projection in the conducting systems and cortical zones of the analyzers, clinical variants of sensitive disorders depending on the level of lesion and the functional state of the analyzer, classification of pain syndromes and methods of their drug correction.

The student should be able to:

to explore superficial (pain, temperature, tactile) and deep sensitivity (feeling of pressure, weight, vibration, kinesthetic and joint-muscular feeling),

complex types of sensitivity (feeling of localization, discrimination two-dimensional - spatial, stereognosis);

draw up and draw a diagram of sensitive disturbances

to assess the nature of sensitive disorders in the patient and determine the topic of the pathological process.

CONTROL QUESTIONS:

1. Classification of different types of sensitivity.
2. What are the conductors of sensitivity?
3. What types of surface sensitivity do you know?
4. How is surface sensitivity investigated?
5. What kinds of deep sensitivity do you know?
6. How is deep sensitivity investigated?
7. What kinds of complex sensitivities do you know?
8. How is complex sensitivity investigated?
9. What types of sensory disorders do you know?
10. What is the significance of the law of eccentric arrangement of long conductors for topical diagnostics?
11. What does non-aural type of sensory impairment mean?
12. What does polyneuritic type of sensitivity disorder mean ?
13. What does radicular sensory disorder mean?
14. What type of sensory disorder is called segmental?
15. What is Dissociated Sensory Disorder?
16. What does spinal conduction type sensory disorder mean?
17. What are the signs of Brown-Séquard syndrome?
18. What type of sensitivity disorder occurs when the brain stem, the inner capsule is damaged?
19. Where and what types of sensitivity fall out with damage to the optic hillock?
20. What is a cortical sensory disorder?
21. What are the manifestations of irritation of the posterior central gyrus?
22. What are the zones of peripheral and segmental innervation of the facial skin?
23. In what areas will pain and temperature sensitivity be impaired when the caudal part of the nucleus of the descending root of the trigeminal nerve is damaged?
24. The axons of which cells form the olfactory nerve?
25. How is the sense of smell tested?
26. When affected, which parts of the olfactory tract is anosmia?
27. Where is the pathological process localized in olfactory hallucinations?
28. What is the method of examining the function of vision?
29. What does the eponymous and opposite hemianopsia mean?

30. What are the main techniques for studying cochlear and vestibular function?
31. What neuron of the auditory pathway is affected by unilateral hearing loss?
32. What symptoms occur when the peripheral part of the vestibular analyzer (labyrinth, nerve, nuclei) is damaged?
33. What are the ways of conducting the gustatory sensations?
34. What is the research methodology for taste?
35. What are the symptoms of a lesion in the taste paths and centers?

In physiology, the entire set of afferent systems is united by the concept of reception. Taking this definition in full, we in the clinic also distinguish the concept of sensitivity within the limits. Indeed, not every irritation conducted within the central nervous system is felt, although it leads to certain reactions - changes in tone, motor, secretory, vascular reflexes, biochemical shifts, mental reactions, etc. Consequently, the concept of reception is broader than the concept of sensitivity. Not everything that is received is felt; the receptors of the cerebellum can be cited as an example.

Paths afferent to the cerebellum do not reach the cerebral cortex; irritations from the organs of movement, carried out along these paths, are not felt, although they cause response, regulating and coordinating reflexes to the muscles due to the automatism of the cerebellar system.

The significance of reception, of sensations in particular, is extremely great: through sensations (sensitivity), a connection is established between the organism and the environment, an orientation in it. It cannot be assumed that sensations, "feelings" about which we judge by the assessment and statements of the researched, refer only to the subjective world. At the same time, they reflect the objective relationship of the organism with the external environment.

Sensitivity should be considered from the point of view of the teachings of I.P. Pavlov about analyzers. The analyzer, as already mentioned, is a complex neural mechanism that begins with a perceiving device and ends in the brain; this device has the task of decomposing (analyzing) the complexity of the external world into separate elements. The analyzer consists of receptors, nerves, conductors, and perceiving brain cells; the connection of all these parts into one mechanism, into a single functional system, and bears the general name of the analyzer. The cortical department of the latter, where the highest function of analysis and synthesis is carried out, is what in the clinic until now bears the name of cortical sensory and gnostic centers.

Peripheral apparatuses (nerve endings) are special (for each type of sensitivity) transformers, each of which converts a certain type of energy into a nervous process. Each individual afferent fiber coming from a specific element of the peripheral receptor apparatus, i.e. the nerve endings, conducts impulses into the cortex that arise when exposed to only a certain type of energy; Accordingly, this

afferent fiber in the cortex must have a special cell associated with a separate specific nerve receptor.

Nerve endings located in tissues are very different in their histological structure. It is assumed that cold sensitivity corresponds to one type of endings, a feeling of pressure, joint-muscular feeling - others, etc. The nerve endings of the interoreceptor system are also diverse in structure.

TYPES OF SENSITIVITY AND METHODS OF RESEARCH

According to one of the classifications, based on determining the place of origin of irritations, sensitivity is divided into exteroceptive, proprioceptive and interoceptive,

1.Exteroceptors are divided into: a) contactceptors, which perceive stimuli applied from the outside, and fall directly on the tissues of the body (pain, temperature, tactile, etc.), and b) distantceptors, which perceive stimuli from sources that are at a distance (light, sound),

2. Proprioceptors perceive irritations arising inside the body, in its deep tissues, associated with the function of maintaining the position of the body during movement. This type of receptor is presented in muscles, tendons, ligaments, joints, periosteum, impulses arise in connection with a change in the degree of tendon tension, muscle tension and orientate in relation to the position of the body and its parts in space: hence the name - "joint-muscle feeling", or "a sense of position and movement (kinesthetic sense)".

3. Interoceptors perceive irritations from internal organs, normally rarely causing distinct sensations; interoceptive afferent systems belong to the section of visceral innervation.

With another division of sensitivity - into superficial: pain, temperature and tactile and deep: muscular-articular, vibration and pressure. The first should include exteroceptors, the second - proprioeptors and interoceptors.

In the clinic, another classification based on biological data has become quite widespread. From this point of view, sensitivity is considered as the ratio and interaction of two systems.

One, more ancient, dual to the more primitive nervous system, serves for the conduction and perception of strong, sharp, threatening the integrity of the organism of irritations; this includes gross pain and temperature irritations associated with the ancient "sensory" organ - the visual hillock. This sensitivity system is called protopathic, vital, nociceptive, thalamic.

Another system is connected entirely with the cerebral cortex. Being more new and perfect, it serves for subtle recognition of the quality, character, degree and localization of irritation. This includes such types of sensitivity as touch, determination of position and movement, shape, place of irritation, discrimination of subtle temperature fluctuations, the quality of pain, etc. The name of this sensitivity system is epicritic, gnostic, cortical.

More commonly used in clinical practice is a descriptive classification based on distinguishing between the type of irritation and the sensation arising in connection with it. From this point of view, the sensitivity can be divided into the following types. Tactile sensitivity, or the sense of touch, touch. Its study is carried out using a cotton swab or a brush with soft hair. As in the study of other types of sensitivity, the subject is invited to close his eyes in order to better concentrate on the registration and analysis of the received stuns, as well as to exclude the possibility of determining the type of visual irritation. Each touch applied sequentially to different parts of the surface.

Sensitivity and her body disorders, the subject should immediately register the word "yes" or "feel." Irritations should be applied sparingly and at irregular intervals. In addition, touches with a cotton swab or a brush should not be "smearing" (to avoid the summation of irritations), but tangents.

Pain sensitivity is investigated with the point of a pin or the end of a pointed goose feather. Painful irritations are applied, alternating with tactile; the subject is given the task to mark the injection with the word "sharp", the touch - with the word "stupid".

Thermal sensitivity is made up of two different types of sensitivity: the feeling of cold and the feeling of warmth. For research, usually two test tubes are used, into one of which cold water is poured, into the other - heated water.

The listed types of sensitivity are the main types of the so-called surface sensitivity, when irritation falls on the surface tissues of the body - the skin and mucous membranes.

Frey's method is a very subtle and accurate way to study tactile and pain sensitivity and the feeling of pressure. With the help of a set of specially selected graduated hairs and bristles attached at right angles to the handle, it is possible to establish individual sensitive points corresponding to the localization of receptors, determine their number per 1 cm^2 of a given skin area, and establish the threshold for irritation of the points.

Frey's method gave a lot of new things in the teaching of physiology and pathology and sensitivity. In practical neurological work, it is of little use due to the extreme diligence and duration of the study.

To determine the feeling of localization, the subject is asked to point with his finger exactly where the irritation is applied with his eyes closed.

The difference between two simultaneous stimuli (or discriminatory feeling) is investigated using the Weber compass. Either, bringing them closer together, then pushing the legs of the compass apart, they simultaneously touch the skin or mucous membrane with both points, noting whether the investigated person distinguishes both touches or perceives them as one. The most sensitive are the tongue, lips, fingertips. There are tables indicating the distances between the legs of the compass, distinguished in the norm, with which the results are compared.

The ability to recognize two-dimensional stimuli is determined by writing numbers, letters, figures on the skin, which the subject must recognize with his eyes closed.

Receptors embedded in the musculoskeletal system (in muscles, tendons, joints, periosteum), conductors from these receptors and cortical areas, where the analysis and synthesis of stimuli arising in the organs of movement, constitute the kinesthetic (motor) analyzer.

The joint-muscular sense, or sense of position and movement, is determined by the recognition of passive movements in the joints. The study begins with the movements of the terminal phalanges, then the fingers, then in the wrist, ankle joints and above. Disorders of the musculoskeletal feeling are noted with the following record: "upset up to the elbow (knee or other) joint, inclusive."

Loss of musculoskeletal sensation causes a movement disorder called sensory ataxia. The patient loses the idea of the position of parts of his body in space: the idea of the direction and volume of movement is lost. Both static and dynamic ataxia are possible, especially if the control of vision is excluded.

Static ataxia is investigated using Romberg's technique: the patient is asked to stand with his feet drawn together and arms extended forward, while instability and staggering are observed, which intensify when the eyes are closed. If there is a disorder of the musculoskeletal feeling in the upper extremities, then the spread fingers of the arms extended forward involuntarily change the accepted position, producing spontaneous movements (pseudoathetosis). Dynamic ataxia in the hands is examined with the fingernose test, in the legs with the calcaneal-knee test. The examinee is invited to touch the coccyx of his nose with his forefinger with his eyes closed or draw the heel of one leg from the knee of the other leg down the front surface of the lower leg. It is essential that when the heel moves downward, it only touches the surface of the lower leg; pressure with the heel can produce a known jerky motion that mimics ataxia. Movements with ataxia lose their smoothness, become irregular, awkward and inaccurate. With ataxia in the legs and trunk, gait is sharply upset: ataxia of the upper limbs leads to a disorder of fine movements, a change in handwriting, etc.

The vibrational feeling is investigated with a vibrating tuning fork, the leg of which is placed on bones covered with thin integuments (the back of the fingers, the back of the hand and foot, the tibia, the spinous processes of the vertebrae or joints).

The feeling of pressure is determined by simple finger pressure or a special device - a baresiometer. The subject must distinguish between touch and pressure and the difference between pressure of different strengths.

The feeling of weight is investigated with the help of weights (weights) applied to an outstretched arm. Normally, there are differences in weight of 15-20 g.

Stereognostic feeling is a complex pitchfork of sensitivity. The examinee is invited to identify the object placed in his hand, by touch, with his eyes closed. Separate perceptions of the qualities of a given object (temperature, weight, shape, surface, dimensions) are combined in the cerebral cortex (synthesis) into a certain complex idea of the object. If the object offered for feeling is familiar to the investigated person (watch, box of matches, coin, key), then it is "recognized", the received perception from the object is compared with the previously existing idea

of it (analysis and synthesis). Since a number of different types of sensitivity are involved in the process of stereognosy, astereognosia also occurs as a result of the loss of the named types of sensitivity, especially tactile and articular-muscular (false astereognosis).

But an isolated disorder of the stereognostic feeling (with damage to the parietal lobe) is also possible, when the patient can describe the individual qualities of the object, but cannot recognize it by touch as a whole.

SENSITIVITY GUIDES

The cells of the first, or peripheral, sensory neurons are embedded in the intervertebral spinal ganglia, their processes - sensory fibers of peripheral nerves - conduct an impulse from the periphery from the nerve sensory endings. Fibers from deep receptors also go to the posterior columns, the rest of the conductors from the cutaneous and cerebellar receptors end in the posterior horns, the following neurons form the ascending conductors of the spinal cord.

From the cells of the intervertebral nodes, the impulse along the fibers of the posterior sensory root is carried to the spinal cord, where fibers of different types of sensitivity diverge in different directions.

The conductors of the joint-muscular sense, vibrational and part of the tactile, without entering the gray matter of the spinal cord, excluding the fibers forming the arcs of segmental reflexes, enter directly into the posterior column of their side and, as part of the Gaulle and Burdach bundles, rise without interruption up to the medulla oblongata , where they end in the cores of the rear pillars, or the cores of Gaulle and Burdach. Consequently, the first, or peripheral, neurons of these types of sensitivity provide impulse conduction from the periphery to the medulla oblongata, and the conductors follow all the time along their side of the spinal cord.

Since the conductors of the articular-muscular sense, tactile and others enter the posterior columns from their outer side, next to the posterior horn, there is a gradual displacement of the fibers previously traveling in the posterior column (from the underlying segments) to the midline, and therefore medially in the located Gaulle's bundle, there are conductors from the lower extremities, in the lateral Burdakh bundle - conductors from the higher segments, mainly the upper extremities.

The conductors of pain and temperature feelings, as well as some of the tactile, enter from the dorsal root into the dorsal horn of the spinal cord, where the axons of the first, or peripheral, neuron end, in contact with the cells located here (the second neuron). The axons of these cells pass through the anterior gray commissure to the opposite side, making a cross here, and enter the lateral column, making up the spinothalamicus tractus. The named conductor is, therefore, composed of the fibers of the second neurons of pain and temperature sensation (partly and tactile) of the opposite side.

The transition of the crossing fibers does not occur in the horizontal plane, at the level of this segment, but obliquely upward, as a result of which the fibers enter the spinothalamicus tractus 2-3 segments higher. This determines the

corresponding decrease in the upper level of conduction disorders of pain and temperature sensation on the opposite side with damage to the lateral column.

Without interruption, the spinothalamicus tractus passes along the lateral column of the spinal cord into the oblong, into the pons varoli and the cerebral peduncles, ending in the lateral nucleus of the optic tubercle, the fibers from the cells of the posterior horn enter the spinothalamicus tractus from the inside, thereby pushing the fibers running in the bundle from the underlying segments outwards. As a result, in the spinothalamic tract, the fibers are arranged in such a way that in its outer section, sensitivity is carried out from the lower segments (sacral, lumbar), in the inner part - from the upper ones (the so-called law of the eccentric arrangement of longer pathways). This circumstance is important for the tonic diagnosis of spinal cord diseases: during intramedullary processes emanating from the gray matter in the direction of the lateral column, conduction disorders of pain and temperature, starting from the level of the affected segments, go down as the process develops. In the case of extramedullary processes affecting the spinothalamicus tractus from the outside, conduction disorders grow from the bottom up.

Together with the fibers of pain and temperature sensation from the posterior root pass into the posterior horn and fibers of the cerebellar proprioceptors. Peripheral neurons end in the gray matter of the spinal cord. The cells of the second neurons are laid here: their axons go out into the lateral column of their side and are located here on the periphery, making up the Flexig bundle and the Govers bundle, the tractus spinocerebellaris dorsalis. These conductors rise up the spinal cord and end in the cerebellar vermis.

Let us return to the consideration of the course of the pathways of the joint-muscular and tactile sense. As mentioned above, the fibers of the first neurons ended in the medulla oblongata, in the nuclei of Gaulle and Burdach. From here, from the cells of the named nuclei, the axons of the second neurons are directed ventrally and to the midline, making a cross at the level of the olives (in the inter-olive layer). Passing to the opposite side, these fibers join, adjoining from the inside, to the spinothalamicus tractus. A bundle of fibers of the second neurons of the articular-muscular and tactile sensitivity is called the tractus bulbothalamicus. The fusion of both sensory pathways - the spinothalamicus tractus and the bulbothalamicus, starting in the medulla oblongata, is finally completed only in the bridge. The common sensory path of the brain stem, made up of the two named bundles, is called the medial loop.

The medial loop is a collection of fibers of the second neurons of all types of sensitivity of the opposite side of the body (the intersection of the fibers of pain and temperature sensitivity occurs sequentially along the segments throughout the spinal cord in the anterior gray commissure, and the fibers of the articular-muscular and tactile sense - in the inter-olivny layer of the medulla oblongata) ... The medial loop is located in the middle floor of the trunk: in the medulla oblongata and the bridge - above the pyramids, in the legs of the brain - above the nigra substance. Initially located close to the midline, both medial loops in the bridge begin to diverge, positioning themselves more and more laterally. Fibers

from the nuclei of the sensory cranial nerves are attached to the loops: glossopharyngeal, vagus and trigeminal (also after crossing). The fibers of the medial loop (the spinothalamicus tractus and the bulbothalamicus tractus) end in the lateral nucleus of the optic tubercle. Along the way in the brain stem, they give off collaterals to the reticular formation. Some of the fibers also end in the medial nucleus; they, apparently, establish reflex connections within the sub-tuberous and subcortical formations. In the visual hillock, there are cells of the third sensory neurons, the fibers of which make up the thalamocortical tractus, heading through the inner capsule (posterior third of the hind thigh) and the radiator crown into the cerebral cortex, into the posterior central gyrus and parietal lobe. The projection into the cortex of the receptor fields of the opposite side of the body is carried out as follows; in the upper part of the posterior central gyrus are receptors of the leg, in the middle - the arms and in the lower part - of the head, i.e. in the reverse order of the body parts. Analysis and synthesis of sensations from both skin receptors (exteroceptors) and articular-muscular (propriopeptors) occurs not only in the posterior central gyrus, but also in much wider areas of the cortex, in particular in the parietal lobe, and the latter is mainly represented by deep sensitivity.

SENSITIVITY DISORDERS

Sensitivity changes include the following.

1. Anesthesia, i.e. loss, loss of one or another type of sensitivity. There is tactile anesthesia, pain (analgesia), temperature anesthesia (thermal anesthesia), loss of a sense of localization (topanesthesia), stereognostic feeling (astereognosia), joint-muscular feeling (batianesthesia), etc. With the loss of all types of sensitivity, they speak of general total anesthesia.

2. Hypesthesia is not a complete loss, but only a decrease in sensitivity, a decrease in the intensity of sensations. Hypesthesia can concern both all sensitivity and its individual types.

3. Hyperesthesia, i.e. hypersensitivity arises as a result of the summation of the irritation applied during the study and the irritation existing due to the pathological process on the path of the sensitive impulse.

4. Dissociation, or splitting of sensitivity disorders, is an isolated violation of some types of sensitivity, while other species are preserved in the same territory.

The presence of pain in an area that is not sensitive to external pain stimuli is called dolorosis anesthesia. Such a disorder can occur as a result, for example, of a complete transection of a peripheral nerve, if its central segment is in a state of irritation with a scar, neuroma, etc. The latter circumstance causes severe pain felt in the zone of nerve innervation; painful irritations applied to this territory are not perceived, since impulses from here do not penetrate into the brain due to an interruption in the nerve.

In the clinic, a kind of perversion of sensitivity is quite often observed, which can be defined as a qualitative change in sensitivity. This type of disorder is called hyperpathy.

5. Hyperpathy is characterized, first of all, by an increase in the thresholds of perception. Subtle distinctions of weak stimuli fall out: slight tactile stimuli are not felt, there is no sensation of warm or cool; the most differentiated types that

require subtle analysis suffer: determination of the place of irritations (localization), individual qualities and their nature. The irritation must reach a significant degree to be perceived (the threshold is raised); often only sharp pain and temperature irritations are felt. From the moment of application of irritation to its perception, there is a long latent period; typical "explosive", a sharp nature of pain with an indefinite, but intense "feeling of unpleasantness." There is no exact localization: irritation has a tendency to irradiation, it “seems to spread or crumble”. There is an aftereffect, i.e. prolonged sensation after the irritation has already stopped.

The idea that hyperpathy is the result of a dissociation between epicritical and protopathic sensitivity (in the sense of disinhibition, revealing the latter when epicritic sensitivity falls out) should be recognized as discredited (E.K. Sepp, K.M. Bykov). Hyperpathy is detected in a variety of cases of disorders of the sensitive system, with the defeat of its various links or in certain phase states of the disease process. So, hyperpathy can occur with certain degrees of conduction disturbance and irritation of the peripheral nerve, in a certain phase of regeneration of its sensitive fibers after an interruption, with sympathetic, lesions of the posterior columns, medial loop, visual hillock, its connections with the cortex, the cerebral cortex itself. Hyperpathy occurs especially clearly and most regularly with damage to the optic hillock and with causalgia.

There is no doubt that the basis of hyperpathy is a violation of the analytical, cortical function. Normally, sensitivity is a complex functional system, and all links of this system, from the periphery to the cortex, take part in every normal sensation. The visual hillock also plays a certain role in this system. “Likewise,” writes K.M. Bykov, - as integral reflex acts in the “ascending series” acquire new qualities, so the “afferentation in the ascending series” acquires new qualitative features ”. According to K.M. Bykov, with the restoration of impaired sensitivity (this should be added - or with the loss of one or another link of the sensitive system) there are periods of increased and decreased cortical sensitivity associated with phase changes in nerve conductors and, probably, in nerve cells both thalamic ganglion apparatus and cortical. "The extraordinary diversity of changes in sensitivity is associated with a complex interaction of cortical and thalamic centers, the appearance of a peculiar sensitivity, changes in the latency period and the" explosive "nature of sensitivity (hyperpathy) should be associated with a violation of the interaction of ganglion apparatus of different floors" (K.M. Bykov). Ultimately, sensitivity is always a cortical process, all types of sensitivity are closely related to each other, making up a complex complex. When one or another link of the sensitive system is damaged, especially when the system of the optic tubercle is damaged, the analysis process is disturbed, a "breakdown of the analyzer" occurs (I.P. Pavlov) and a completely different, qualitatively changed sensitivity is created, a particular manifestation of which is, for example, hyperpathy ...

Other forks of sensitivity changes include the following.

6. Dysesthesia, which consists in perverting the perception of irritation: touch is perceived as pain, cold as warmth, etc.

7. Polyesthesia, when the idea of several irritations arises, although in fact one was inflicted.

eight. Synesthesia is a sensation of irritation not only at the site of its application, but also in any other area (usually in the segment of the same name - the dermatome of the opposite side).

From the forms of sensitivity disorders discussed above, established by research, one should distinguish those sensory disorders that arise without causing external stimuli. This category includes paresthesias and so-called spontaneous pains.

1. Paresthesias are abnormal sensations experienced without receiving external irritation. They can be extremely varied: a feeling of numbness, crawling creeps, heat or cold, tingling, burning, etc.

2. The pains arising in the body as a result of certain pathological processes and felt without applying external stimuli are the result of irritation of receptors, sensitive conductors or centers. Although damage to any part of the sensory system can cause pain or paresthesias, it should be emphasized that the most distinct pain phenomena occur when peripheral nerves, posterior sensory roots of the spinal cord and roots of the sensory cranial nerves, the meninges of the spinal cord and brain, and, finally, the optic tubercles are affected.

By localization, pain can be divided into: a) local, b) projection, c) radiating and d) reflected.

With local pain, the localization of the felt pain coincides with the localization of the pathological process. So, with inflammation of the nerve, pain can be felt throughout it, corresponding exactly to the anatomical location of the nerve trunk.

With projection pains, their localization does not coincide with the localization of local irritation in the sensitive system. For example, in case of injury or tumor of the proximal nerve trunk, pain is projected into the zone of peripheral innervation of the nerve; It is known that when the ulnar nerve is bruised in the region of the elbow joint, pain is felt in the fourth and fifth fingers, that irritation of the posterior sensory roots of the spinal cord gives pain that projects, “shoots” at the limbs or “girdles” the body, etc. An example of the same pain is the "phantom" pain of the amputated: irritation of the ends of the severed nerves in the stump creates a false sensation of pain in the distal parts (fingers) of the missing limbs, etc.

Irradiating are called, in particular, those pains that are caused by the spread of irritation from one branch of the nerve to another. So, with a sharp irritation of one of the branches of the trigeminal nerve, pain can spread along its other branches (for example, with toothache).

Irradiation of irritation also results in reflected pain in diseases of internal organs. In this case, irritation spreads from visceral receptors to pain sensitivity cells in the posterior horns of the spinal cord; as a result, pain is felt as transient

from the area innervated by this segment, "projected" into the area of this segment. Such pains are called viscerosensory phenomena, and the territories where they arise are called Zakharyin-Ged zones. In addition to pain, hyperesthesia can also be observed here.

Reflected pain and hyperesthesia can be a valuable auxiliary symptom in the diagnosis of diseases of internal organs.

A special category of painful phenomena is the so-called causalgia: burning, intense pain that sometimes occurs as a result of injuries to the peripheral nerves, especially the median and sciatic. A painful burning sensation often forces patients to continuously moisturize the affected limb. In the area of the skin innervated by the irritated nerve, hyperpathy phenomena are observed in the study of sensitivity. This zone usually extends beyond the innervation of the affected nerve, acquiring the character of a "glove" or "stocking". At the heart of causalgia is an incomplete violation of the conduction of the nerve with the phenomena of its irritation. The peculiar nature of painful phenomena is explained by the involvement of sympathetic elements in the process ("sympathetic"). According to M.I. Astvapaturov, causalgia arises mainly as a result of over-irritation of the visual hillock, whose participation in the symptom complex of causalgia is undoubtedly.

In addition to the considered category of pains that arise without applying external stimuli, there are so-called reactive pains, which are caused as a result of certain influences. Common methods of inducing reactive pain include pressure and traction on the nerve trunks, often used in research. The pressure on the nerve is usually produced in those areas where it lies more superficially and closer to the bone.

An example of determining the pain of a nerve by the traction method is the Lasegue technique: the examiner raises the leg of the lying patient, straightened in the knee joint, bending it in the hip; in the presence of irritation of the sciatic nerve, the patient experiences pain along the sciatic nerve during this method (stretching the nerve). Otherwise, the soreness of the femoral nerve is experienced: when the patient is on his stomach, the examiner bends the leg in the knee (or hip) joint, when the nerve is irritated, pain occurs in the anterior thigh and groin (Wasserman symptom).

SYNDROMES OF SURFACE AND DEEP SENSITIVITY DISORDERS

The defeat (complete) of the trunk of the peripheral nerve is characterized by a violation of all types of sensitivity in the area of cutaneous innervation of this nerve, since the fibers of all types of sensitivity in the peripheral nerve pass together. The defeat of the mixed or sensory nerve is usually accompanied by pain or paresthesia. The defeat of the plexus trunks (cervical, brachial, lumbar and sacral) causes anesthesia or hypesthesia of all types of limb sensitivity in the territory innervated by the sensitive fibers of those nerves that originate from the affected trunk (or trunks) of the plexus. Pain is also common here.

The defeat of the posterior sensory root of the spinal cord also barks loss or decrease in all types of sensitivity, but the zones of sensory disorders are already of a different, namely segmental nature; circular on the body and longitudinal stripes on the limbs. The defeat of the roots is also accompanied by pain.

With the simultaneous involvement of the intervertebral ganglion in the process (ganglionitis or ganglioneuritis), herpes zoster vesicles may erupt in the area of the corresponding segments.

The defeat of the dorsal horn of the spinal cord causes the same segmental disorders of sensitivity as the defeat of the dorsal root, but in contrast to the defeat of the root, split, or dissociated, disorders are observed here . It should be remembered that when the posterior sensory root enters the spinal cord, only the fibers of pain and temperature sensitivity enter the posterior horn, while the fibers of the tactile and musculoskeletal sense bypass it, entering directly into the white conductors of the posterior column. Therefore, when the posterior horn is damaged, only the pathways for conducting pain and temperature sensations from this segment are interrupted. As a result, pain and temperature anesthesia occurs while tactile sensitivity is preserved in the same territory (dissociation). It was believed that, in contrast to the defeat of the posterior root, with the defeat of the posterior horn, pain is not so characteristic; however, even with the defeat of the posterior horns, very intense painful sensations are often observed.

The defeat of the anterior gray commissure of the spinal cord, where the intersection of the fibers of pain and temperature sensation occurs, also causes dissociated disorders (loss of pain and temperature sensitivity while the tactile sensitivity is preserved); areas of anesthesia are segmental; they are bilateral and symmetrical (butterfly type).

Unlike lesions of the dorsal roots, posterior horns and anterior gray commissures, which cause segmental distribution of anesthesia, damage to the white conductors of the central nervous system will give a conductive sensory disorder .

The defeat of the posterior column of the spinal cord, where the Gaulle and Burdach beams pass, causes the loss of the articular-muscular and vibrational feeling on the side of the lesion of the conductive type, i.e. from the level of the lesion to the end down: disorders of tactile sense can also occur . As a result of a violation of the sense of position, a sensitive ataxia sets in, which was mentioned above. Lesions of the posterior columns of the spinal cord often cause the appearance of hyperpathy when painful and temperature irritations are applied.

The defeat of the lateral column of the spinal cord causes pain and temperature anesthesia of the conductive type due to the defeat of the spinothalamicus tract passing here. The loss of these types of sensitivity occurs on the opposite side, since the fibers of the second neurons of pain and temperature sensation, before their entry into the lateral column, are crossed in the anterior gray commissure. The defeat of half of the spinal cord gives on the side of the focus: a violation of the musculoskeletal feeling in the presence of central paralysis from

top to bottom from the level of the lesion, on the opposite side, conductive pain and temperature anesthesia.

This symptom complex is called Brown-Sekar's paralysis. The defeat of the entire diameter of the spinal cord, causing a break in all sensitive conductors from the lower segments of the body, gives a picture of anesthesia of all types of sensitivity of the conductor type on both sides, downward from the level of the lesion.

At the same time, bilateral central paralysis with urination disorder is observed .

The defeat of the medial loop in the brain stem after the complete fusion of the spinothalamicus tractus and the bulbothalamicus (in the bridge, in the brain pedicles) causes loss of all types of sensitivity on the opposite side of the body and sensitive ataxia

in the opposite limbs due to the loss of joint-muscular feeling (hemianesthesia and hemiataxia).

The fibers for various types of sensitivity in the medial loop are located in such a way that the conductors of the articular-muscular feeling are located most medially, outward from them - tactile, even more lateral - temperature, and finally, painful feeling. Therefore, with incomplete damage to the medial loop, predominantly certain types of sensitivity may drop out on the opposite side of the body. This is all the more possible with damage to the medulla oblongata, where the spinothalamicus and bulbothalamicus tractus have not yet merged and go separately. The law of the eccentric arrangement of longer pathways also applies to the medial loop: the conductors from the underlying segments (lower extremities) are located ventrolaterally from the overlying ones.

The defeat of the visual hillock - causes, like the defeat of the medial loop, hemianesthesia of all types of sensitivity and hemiataxia on the opposite side; in addition, due to the defeat of the subcortical visual centers , hemianopsia of the opposite visual fields also occurs, i.e. “Three-tee syndrome”: hemianesthesia, hemiataxia and hemianopsia.

Often, with the defeat of the visual hillock , a kind of thalamic pain occurs in the opposite half of the body - hemialgia; there is a painful and extremely unpleasant feeling of coldness or burning, which can hardly be described by the patient and is poorly localized by him; vagueness, irradiation of pain is observed. These pains usually do not respond well to therapeutic influences. When examining sensitivity on the half of the body opposite the focus, hyperpathy is usually found. Sometimes these pains are worse at rest and are better with movement. The defeat of the sensory pathways in the inner capsule, where the fibers of the third sensory neurons (tractus thalamocorticalis) pass in the posterior third of the hind thigh, also causes the “three hemi syndrome”: hemianesthesia, hemiataxia and hemianopsia. Often, the processes in the inner capsule are more diffuse, and then, due to the defeat of the pyramidal pathways, hemiplegia is also observed on the opposite side and the central type, i.e. “Three hemi syndrome” of a different nature: hemiplegia, hemianesthesia and hemianopsia. If, with a lesion of the posterior femur of the inner capsule , the visual hillock is also involved in the

process , then hemianesthesia is the same as with the defeat of the optic hillock, i.e. applies to all types of sensitivity. If the visual hillock is not affected, then such types of sensitivity as tactile, sense of position, localization, etc. fall out on the opposite side, and hyperpathy occurs when sharp pain and temperature irritations are applied.

The defeat of the central posterior gyrus of the cerebral cortex causes prolapse on the opposite side of the same types of sensitivity as with the defeat of the inner capsule; often, and at the same time there is hyperpathy. The half type of anesthesia is observed here not as regularly as in the case of damage to the medial loop, optic tubercle and internal capsule, since it is often not the entire posterior central gyrus that is affected, but only one or another of its parts. In such cases, it is not hemi - but monoanesthesia that occurs: with a lesion, for example, of the middle sections of the gyrus - the arms, the upper - the legs, etc. For cortical sensitivity disorders, they are also characterized by their greater severity in the distal parts: on the hand, on the foot, while sensitivity disorders with damage to the capsule or medial loop are distributed more evenly over the entire opposite half of the body.

Foci in the corona radiatae area cause sensory disorders either mono - (if they are closer to the cortex) or hemianesthetic type (closer to the inner capsule). And in these cases, hyperpathy is observed.

Irritation of the posterior central gyrus causes the appearance, usually in the form of seizures, paresthesia in the half of the body opposite the focus, in the territory corresponding to the "irritated" area. Further, paresthesias "spread" into neighboring areas and may result in convulsions and a general epileptic seizure.

CRANIAL NERVE DAMAGE AND ANALYZER FUNCTIONAL DISTURBANCES.

The purpose of the lesson is to study the anatomical and physiological syndromes of lesion and methods of studying the cranial nerves.

The student should know:

structural and functional features of I and II cranial nerves and groups of oculomotor nerves (III, IV, VI pp.); the system of the posterior (medial) longitudinal beam, ensuring friendly movements of the eyeballs, options for paresis of the gaze at different localizations of the lesion;

structural and functional features of the trigeminal (V) nerve, ensuring the act of chewing and articulation, lesion syndromes;

structural and functional features of the facial (VII) nerve system, lesion syndromes at different levels, participation in providing facial expressions and emotional reactions;

structural and functional features of the bulbar group of nerves (IX, X, XI, XII pp.): ensuring the functions of breathing, swallowing, chewing, speech, etc.;

bulbar and pseudobulbar syndromes;

symptoms of oral automatism;

alternating syndromes of the midbrain, pons and medulla oblongata.

The student should be able to :

to investigate the functions of the olfactory, optic and oculomotor nerves, the direct and friendly reaction of the pupils to light, convergence and accommodation, to reveal anisocoria, diplopia, paresis of gaze;

to investigate the functions of the motor portion of the trigeminal nerve (tension of the masticatory muscles, movement of the lower jaw);

to investigate the state of the facial muscles, taste disorders and differentiate the central and peripheral lesions of the facial muscles;

to investigate the functional state of the bulbar group of nerves (the act of breathing, swallowing, chewing, articulation, mobility of the tongue, soft palate), to identify atrophy and fibrillar twitching in the tongue, choking when swallowing, etc.;

to identify paresis of the sternocleidomastoid and trapezius muscles;

solve problems of topical diagnostics.

Test questions :

1. Which cranial nerves are motor nerves?
2. In which mixed cranial nerves are motor fibers present?
3. What structures of the spinal cord are analogous to the nuclei of the motor cranial nerves?
4. In which part of the brain stem are the nuclei of the oculomotor nerve located?
5. What functions are provided by the motor portions of the trigeminal nerve?

6. In which part of the brain stem lies the nucleus of the facial nerve, and what functions are provided by it?
7. What is the difference in the clinical lesion of the facial nerve in the central and peripheral types?
8. What localization of the process is indicated by the presence of hyperacusis and taste disorder in the anterior two-thirds of the tongue?
9. Which cranial nerves make up the caudal group?
10. Where does the nucleus lie and what functions are provided by the accessory nerve?
11. What are the symptoms of bulbar palsy?
12. What are the signs of the difference between bulbar and pseudobulbar palsy?
13. What formations are the peripheral apparatus of the visual analyzer represented by?
14. What cytoarchitectonic fields of the cerebral cortex belong to the cortical centers of the visual analyzer?
15. What is included in the concept of hemianopsia and with the defeat of which structures does it arise?
16. What formations is the peripheral apparatus of the olfactory analyzer represented by?
17. What cytoarchitectonic fields of the cerebral cortex belong to the olfactory analyzer?
18. At what localization of the pathological process does anosmia occur?
19. What systems of cranial nerves are used for the taste analyzer?

Twelve pairs of cranial nerves are divided into 3 purely sensitive pairs, 6 motor and 3 mixed, having both sensitive and mixed fibers. Strictly speaking, purely motor cranial nerves do not exist, since each of them has a certain amount of sensory fibers (deep sensitivity).

In their origin, structure and function, the last 10 pairs of cranial nerves (III-XII) do not differ significantly from the spinal ones. Thus, the sensory fibers of the cranial nerves are nothing more than the fibers of sensory cells located in special ganglia, which are equivalent to the intervertebral saginal nodes; the axons of these cells pass as part of the sensory root of the corresponding cranial nerve (homologue of the posterior sensory spinal root) and enter the sensory nuclei of the

brain stem (correspond to the posterior horns of the spinal cord), ending in the cells of the latter the first (peripheral) sensory neuron.

Sensory fibers of the cranial nerves are the peripheral parts of the analyzers (cutaneous, kinesthetic, gustatory, auditory, vestibular, visual, olfactory).

The motor fibers of the cranial nerves start from the cells of the motor nuclei (homologues of the anterior horns of the spinal cord) and emerge as part of the motor root from the brain stem (equivalent to the anterior root of the spinal cord).

Corresponding to the aforementioned similarity, in the pictures of the lesion of the cranial nerves, their roots and nuclei, we see familiar features of the lesion of the spinal nerves and the gray matter of the spinal cord. So, damage to the motor nucleus, root or the motor cranial nerve itself gives a symptom complex of peripheral paralysis of the corresponding muscles. The defeat of the trigeminal nerve root from the defeat of its nucleus in the brain stem differs in the same features that allowed us to differentiate the defeat of the posterior sensitive spinal root from the posterior horn: pain, violation of all types of sensitivity - in the first case, and split sensitivity disorders - in the second. Consider the caudal group of nerves.

Caudal group of nerves.

XI couple, n. accessorius - motor nerve. N. accessorius. (accessory nerve) can rightfully be considered a spinal nerve. The cells that give rise to its fibers are located in a long nucleus located in the gray matter of the spinal cord at level IV (partly also VI-V II) of the cervical segments at the base of the anterior horn. Its thin roots (6-7 in number) emerge along the lateral surface of the spinal cord between its anterior and posterior roots at the level of C1 — C V1 — C VP segments and merge into one common nerve stem, following inside the spinal canal along the lateral surface of the spinal cord upward. Further, the nerve enters the cranial cavity through the foramen occipetale magnum, passes towards the foramen jugular, from where it leaves the skull again. Innervates m. Sternocleidomastoideus and m. Trapezius. The function of this nerve is to turn the head in the opposite direction, raising the shoulder, scapula and acromial part of the clavicle upward ("shrug"), pulling back the shoulder girdle and bringing the scapula to the spine, as well as raising the shoulder above the horizontal. When the nucleus, root or nerve is damaged, peripheral or atrophic paralysis of the innervated muscles develops. In this case, the sternocleidomastoideus and the upper section of the trapezium are atrophied, it is difficult to turn the head to the healthy side, the shoulder on the affected side is pubescent, the scapula moves away from the spine with its lower angle outward and upward, the patient has difficulty shrugging the shoulder, raising the hand above the horizontal line is limited.

Convulsions of the muscles innervated by the XI nerve are very rare in isolation; they are often unilateral and are the result of cortical or subcortical irritations. Tonic spasm gives a picture of "torticollis" (torticolis spasticus); clonic

- twitching of the head in the opposite direction, sometimes with simultaneous lifting of the shoulder.

Bilateral clonic spasm leads to nodding movements of the head (salaam spasm, nutas spasm).

XII couple, n. hypoglossus - motor nerve. Core n. hypoglossi is located at the bottom of the rhomboid fossa, located dorsally in the depth of trigonum n. hypoglossi, with its caudal section it reaches down to 1-1 of the cervical segment. The roots (10-15 in number) protrude between the pyramids and olives of the medulla oblongata and merge into a common stem, which leaves the skull through the hypoglossi canalis. N. hypoglossus is the motor nerve of the tongue. When it is damaged, peripheral paralysis or paresis of the corresponding half of the tongue develops with atrophy and thinning of the muscles (with damage to the nucleus, fibrillar twitching is also observed). When protruding the tongue, it deflects with its end towards the affected muscle. This is because the healthy side, pushing the tongue forward more strongly, pushes the tongue towards the weaker half. Unilateral damage to the tongue (hemiglossoplegia) does not cause noticeable dysfunctions, which is explained by the significant interlacing of the muscle fibers of both halves, i.e. the latter entering the middle line to the other side. Bilateral damage to the language (glossoplegia) leads to speech impairment, which becomes indistinct, insufficiently understandable, intertwined (dysarthria); in mild cases, this can be detected only when pronouncing words that are difficult to articulate (for example, curdled milk serum "). With a complete bilateral defeat of the tongue, speech becomes impossible (anarthria): the tongue is motionless, cannot be stuck out of the mouth. It is clear that at the same time the process of eating is also sharply hampered; the food bolt cannot be moved in the mouth for chewing, but moved to the throat for swallowing.

With the same in the main picture of peripheral paralysis resulting from damage to the nucleus, root or seal, the level of damage can usually be established more accurately. For chronic progressive processes in the nucleus itself, fibrillar twitching is characteristic, as already mentioned. In addition, with a nuclear lesion of the XII nerve, simultaneously with the tongue, m. Orbicularis oris (thinning, folding of the lips, impossibility of whistling) is affected (in isolation from the rest of the facial muscles). This circumstance can presumably be explained by the fact that the motor fibers for the circular muscle of the mouth, going to the periphery as part of the facial nerve, start from the cells located in the nucleus N. hypoglossi and suffer if it is defeated. Finally, if the more peripheral part of the nerve itself is damaged, after it leaves the cranial cavity, lesions of the muscles that fix the larynx, innervated by the upper cervical nerves that are anastomosed with n, can join the atrophy of the tongue . hypoglossus. When swallowing, in this case, a displacement of the larynx to the side is noticeable.

IX pair, n. glossopharyngeus is a mixed nerve. It has to do with both somatic and autonomic innervation; contains motor, sensory, special taste and secretory fibers. Accordingly, it has nuclei in the medulla oblongata, many of which are common with n. vagus. It has two ganglia - the superius ganglion and the petrosum

ganglion (homologues of the sensitive spinal intervertebral nodes), which include the cells of the first, or peripheral, sensitive neuron. Sensory fibers of the glossopharyngeal nerve in the roots of the IX nerve (4-5 in number) include

the medulla oblongata between the olive and the corpus are restiform and end in the nucleus ale cinerea (common with the X nerve) and the nucleus tractus of the solitary - the "gustatory" nucleus (common with the XIII nerve). Taste fibers of pairs IX and XIII, their common core, the nucleus of the solitary tract, represent the peripheral part of the taste analyzer. Its cortical section is located in the temporal lobe, deep in the Sylvian groove, around the islet of Reil (Penfield).

Motor fibers (somatic, for innervation of striated muscles) originate from the ambiguus nucleus (shared with the X nerve). Finally, secretory, or rather salivary, nerve fibers begin in the lower part of a special nucleus - the salivatorius nucleus (common with the XIII nerve).

So, the IX nerve has 4 nuclei in the medulla oblongata: nucleus alecynerea, nucleus tractus solitarium, nucleus salivatorius and nucleus ambiguus. The roots, as already indicated, emerge in the medulla oblongata between the olive and the corpus of the restiform; further, the common nerve trunk leaves the cranial cavity through the foramen jugular (in which and near which the sensitive ganglia of the nerve are located - the ganglion supfriis and the ganglion petrosum.

N. glossopharyngeus is:

1) a sensitive gustatory nerve for the posterior third of the tongue and palate;

2) the sensitive nerve of the middle ear and pharynx (together with N. vagus);

3) the motor nerve of the pharyngeal muscles (together with the X nerve) and the secretory one for the parotid salivary gland (glandula parotis).

With the defeat of N. glossopharyngeus are observed:

1) loss of taste on the side of the same name (or ageisia) on the back third of the tongue (to study taste, use a set of bottles with bitter, sweet, sour solutions: a drop of solution is applied to the tongue with a pipette, first from one side, then from the other side);

2) anesthesia of the mucous membrane of the upper half of the pharynx;

3) disorders of swallowing, which are usually insignificant or not given at all, since it is of greater importance in the innervation of the pharyngeal muscles. vagus.

Switching off (unilateral) function of the glandula parotis is compensated by the activity of the other salivary glands, therefore dry mouth may be absent or insignificant.

The phenomena of irritation of the IX nerve include a spasm of the pharyngeal muscles - pharyngismus or pharyngospasm, which is already the result of damage to the higher parts of the central nervous system or a manifestation of neurosis.

X pair, n. vagus is a mixed nerve. Like IX, X nerve is mixed and not only somatic, but also visceral, vegetative. It has a very diverse and complex function, it includes motor fibers for striated and smooth muscles, sensory, secretory, etc. It

has, accordingly, several nuclei in the medulla oblongata, some of them are common with the IX nerve. Sensory fibers of the X nerve begin from the cells of the jugular ganglion and nodosum ganglion, go to the medulla oblongata and enter it as part of 12-16 root filaments between the olive and the corpus cord below the glossopharyngeal nerve roots. The first, or peripheral, sensory neuron ends in the sensitive king of the 1X-X nerves - the nucleus ale cinerea. Motor fibers - somatic for the striated muscles - start from the nucleus ambiguus common with the IX nerve; vegetative motor fibers for smooth muscles (internal organs, vessels) - from the nucleus (dorsalis n. Vagi). From the cranial cavity n. vagus goes out together with n. glossopharyngeus and n. acecessorius through foramen jugular, in which and below which the named ganglia of the nerve are located - ganglion jugular, ganglion nodosum. The vagus nerve with its motor fibers innervates the muscles of the pharynx (together with the IX nerve), soft palate, larynx and epiglottis, smooth muscles of the trachea and bronchi, esophagus, stomach, small and upper large intestines. Its sensitive fibers end in the meninges, deep in the external auditory canal, pharynx, larynx, trachea, bronchi, lungs, gastrointestinal tract and other organs of the abdominal cavity. In addition, it contains secretory fibers to the stomach and pancreas, inhibitory fibers of the heart and, finally, vasomotor fibers to the vessels.

With unilateral lesion of the vagus nerve, there is a drooping of the soft palate on the side of the lesion, immobility or lagging behind in this half when pronouncing the sound "a". The uvula is deflected to the healthy side. In addition, with unilateral damage to the X nerve, paralysis of the vocal cord is observed, established by laryngoscopic examination; the voice becomes hoarse. The pharyngeal reflex from the mucous membrane of the affected side of the pharynx may be lost. This usually limits the symptom complex of prolapse with unilateral lesion of N. vagi. With bilateral damage to the X nerve, there is a nasal, nasal tone of voice, pouring out of liquid food through the nose (paralysis of the soft palate); voice changes can reach full aphonia (silent, whispering speech).

Due to the paralysis of the epiglottis, choking occurs when eating, coughing; the penetration of food particles into the trachea and bronchi often infects the lungs. Swallowing disorders (dysphagia) set in, sometimes requiring the use of artificial nutrition. From the side of the heart, tachycardia can be observed, slowing and irregular breathing are possible.

Complete bilateral loss of vagus nerve function causes death (cessation of cardiac activity and respiration).

The phenomena of irritation should include convulsions in the larynx, pharyngo -, esophago - cardio - pylorospasmus, cardiac dysfunction, etc. These disorders can arise as a clarification of neurosis or be tabic crises; finally, they can arise as a result of subcortical and cortical irritations.

The symptom complex of movement disorders resulting from damage to the considered nerves of the caudal group (IX, X, XII) is called boulevard paralysis. The latter arises as a result of damage to the nuclei in the medulla oblongata and roots or nerves (inside or outside the skull). At the same time, paresis or paralysis of the tongue with corresponding speech disorders, impaired

swallowing, choking, pouring of fluid through the nose, nasal tone of voice, its hoarseness (or aphonia) are observed. In full measure, all these disorders are observed in a bilateral process and bear all the features of peripheral paralysis. Therefore, atrophy of the tongue is typical here, fibrillar twitching in it, the reaction of degeneration are frequent; the corresponding reflexes (pharyngeal) fade away or rise.

It is clear that similar disorders are observed in supranuclear lesions, i.e. with the involvement of the central motor neurons in the process in any part of their length - from the anterior central gyri to the nuclei 1X, X and XII pairs - tractus corticobulbaris. True, in this case, with unilateral damage, the prolapse is extremely insignificant: no functional disturbances from the side of the IX and X pairs (and XI) occur due to the bilateral cortical innervation of the nuclei, with the cells of which the central motor neurons from both hemispheres are in contact, with their and the opposite side.

The only thing that can be noted with a unilateral lesion of the anterior central gyrus or tractus corticonuclearis is the deviation of the tongue when it protrudes towards the weak muscle (in this case, with central paresis, in the direction opposite to the lesion focus). At the same time, speech disorders do not occur.

But with bilateral lesions of the central motor neurons, the entire symptom complex of bulbar disorders appears in the same form in which it is observed with bulbar localization of the process. Unlike the latter, it is called pseudobulbar. Functional dysfunctions in both cases will be the same; however, with pseudobulbar, as with any central paralysis, there is no atrophy and the reaction of degeneration; reflexes of oral automatism, as opposed to tabloid paralysis, here will come to light with particular intensity.

NERVES OF THE BRIDGE-CEREBREL ANGLE.

The main nerves of the cerebellopontine angle are n. facialis (XII nerve) with n. intermediate (VIII nerve) and n. vestibulocochlearis (VIII nerve). This group often includes those who go out in the immediate vicinity of N. abducens (VI nerve) and n. trigeminus (V nerve). In processes in the region of the cerebellopontine angle (for example, with tumors), in addition to the VII and VIII nerves, these nerves are often involved in the process. Pair VI will be considered in the group of oculomotor nerves.

VII couple, n. facialis - motor nerve. Core n. the facialis is located quite deep in the lower part of the pons of varoli, on the border with the medulla oblongata. Fibers emanating from the cells of the nucleus rise dorsally to the bottom of the rhomboid fossa and bend around the nucleus located here n. abducentis (VI nerve), forming the so-called knee (internal) of the facial nerve. Further, the fibers are directed downward and come out with a root at the base between the bridge and the medulla oblongata, lateral to the olive, in the cerebellopontine angle (together with the intermediate and N. acousticus), following in the direction of the porus acousticus internus. At the base of the

metatus acousticus of the facial and wrisbergs, the nerves depart from the auditory and enter the canalis of the facialis of Fallopia. Here, in the pyramid of the temporal bone, the VII nerve again forms the knee (external) and, finally, exits the skull through the stylomastoideum foramen, dividing into a number of terminal branches ("goose foot", dog anserinus). N. facialis is a motor nerve of the facial muscles and innervates all facial muscles (except for m. Levator palpebre superioris - III nerve), m. Digastricus (posterior abdomen), m. Stylochioideus and, finally, m. Stapedius and m. Platysma on the neck. For a considerable extent, a fellow traveler of the facial nerve is N. intermediate, also called the XIII cranial nerve. This is a mixed nerve with centripetal sensory, more precisely - taste, and centrifugal secretory salivary fibers. In terms of its meaning, it is largely identical to the glossopharyngeal nerve, with which it has common nuclei. Sensory taste fibers originate from cells located in the temporal bone. They go to the periphery together with the fallopian canal and leave the latter in the composition: later they enter the trigeminal nerve system and through reach the tongue, supplying its anterior two-thirds with taste ends (the posterior third is innervated from the glossopharyngeal nerve). Axons of cells n. sideshows from ganglion genikuli together with n. the facialis enter the cerebellopontine angle into the brain stem and end in the common "taste" nucleus with the ninth nerve — the nucleus tractus of the solitary.

Secretory salivary fibers of the XIII nerve originate from the nucleus salivatorius common with the IX nerve and pass together with n. facialis, leaving the canalis facialis as part of the same tympani chord; they innervate the submandibular and sublingual salivary glands.

Except n. interludes, for a certain extent, accompany the facial nerve and secretory lacrimal fibers, starting from a special secretory nucleus located near the nucleus of the VII nerve. Together with n. facialis, these fibers enter the fallopian canal, which soon leave as part of n. petrosus major. In the future, lacrimal fibers enter the trigeminal nerve system and through n. lacrimalis (V nerve) reach the lacrimal glands. When these fibers are damaged, there is no tearing and dryness of the eye is observed.

Somewhat below, the discharge is separated from the facial nerve and leaves the fallopian canal and n fibers. stapedia. With the defeat of the muscle of the same name innervated by it, hypeacusis is observed (unpleasant, enhanced perception of sound, especially low tones).

Below the named two branches leaves the bony canal and is separated from the facial nerve by the tympanic notochord - continuation of n. interludes with its taste fibers for the anterior two-thirds of the tongue and salivary fibers for the submandibular and sublingual glands.

The defeat of the VII nerve causes peripheral paralysis of the facial muscles (prosopoplegia). Already with a simple examination, the sharp asymmetry of the face is striking. The affected side is mask-like, the folds of the forehead and the nasolabial fold are smoothed here, the palpebral fissure is wider, the corner of the mouth is pubescent. When the forehead is wrinkled, no folds are formed on the side of the paralysis (m. Frontalis with ventral frontalis m. Occipitofrontalis are

affected); when screwing up, the palpebral fissure does not close due to the weakness of the m. orbicularis of the oculi. In this case, the upward movement of the eyeball is visible (Bell's phenomenon). With lagophthalmos, increased lacrimation is usually observed. When showing teeth, the corner of the mouth on the affected side is not pulled backward (m. Risorius), m. Platysma on the neck is not strained . Whistling is impossible, speech is somewhat difficult (m. Orbicularis oris). As with any peripheral paralysis, a rebirth reaction is observed, the superciliary reflex (carneal) is lost or weakened.

The height of the lesion of the facial nerve should be determined depending on the symptoms accompanying the described picture.

With damage to the nucleus or fibers inside the brain stem, the defeat of the facial nerve is accompanied by central paralysis or paresis of the extremities of the opposite side (alternating Miyard-Gubler syndrome), sometimes with the addition of a lesion. abducentis (Fauville syndrome). The defeat of the root n. facialis and in the place of its exit from the brain stem is usually combined with a lesion of N. vestibulocochlearis (deafness) and other symptoms of damage to the cerebellopontine angle.

Paralysis of the facial nerve in these cases is not accompanied by lacrimation (dry eye), there is a violation of taste in the front two-thirds of the tongue, dry mouth may be felt. Hyperacusis is not observed due to a combined lesion of the VIII nerve.

During processes in the area of the bone rope up to the gene n. facialis, i.e. above the discharge, simultaneously with paralysis, dry eyes, disorders of taste and salivation are also noted; from the side of hearing, hyperacusis is observed here (damage to the fibers of n. stapedi). With a lesion in the bone canal below the discharge of n. petrosi majoris are observed along with paralysis, the same disorders of taste, salivation and hyperacusis, but instead of dry eyes, increased lacrimation occurs. In case of damage to the facial nerve in the bone canal below the discharge of the n. stapedia and above the tympani chord, paralysis, lacrimation, disorders of taste and salivation and salivation are observed. Finally, when the nerve is damaged in the bone below the outlet of the tympani chord or after it leaves the skull through the stylomastoideum foramen, only paralysis with lacrimation is observed without those concomitant symptoms that were discussed with higher lesions.

The most frequent are the latter cases with peripheral localization of the process, and the paralysis is usually unilateral. Cases of diplegte facialis are rare. It should be noted that with peripheral paralysis of the facial nerve, especially at the onset of the disease, pain in the face, in the ear and around it (especially often in the area of the mastoid process) is very often observed.

This is due to the presence in this area of rather intimate connections (anastomoses) with branches of the trigeminal nerve, the possible passage of sensory fibers of the V nerve into the facies canal (tympani chord N. petrosom major), the simultaneous involvement of the facial nerve and the root of the trigeminal nerve or his mind during processes based on the brain.

Central paralysis (paresis) of the facial muscles are observed, as a rule, in combination with hemiplegia. Isolated lesions of the facial muscles of the central type are rare and are sometimes observed with damage to the frontal lobe or only the lower part of the anterior central gyrus. It is clear that central paresis of facial mice is the result of a supranuclear lesion of the tractus corticonuclearis in any of its parts (cerebral cortex, radiata corona, intern capsule, cerebral peduncles, bridge). With central paralysis, the upper facial muscles are almost not affected, and only the lower (oral) muscles are affected. This is due to the fact that the upper cell group of the nucleus of the VII nerve has bilateral cortical innervation, in contrast to the lower one, to the cells of which the fibers of the central nerves mainly come from the opposite hemisphere.

With central paralysis of the facial muscles, in contrast to peripheral paralysis, the reaction of degeneration will not be observed; the superciliary reflex is preserved and even strengthened.

The phenomena of irritation in the area of the facial muscles include various kinds of tics (manifestation of neurosis or organic disease), contractures that may be a consequence of peripheral paralysis of the VII nerve, localized spasm, and other clinical and tonic convulsions (cortical or subcortical hyperkinesis).

VIII couple, n. acousticus with n. vestibulocochlearis. Under the general name n. acousticus combines two completely independent sensory nerves with different functions - n. pars cochlearis and n. pars vestibularis.

Pars cochlearis. A true auditory nerve that has a spiral ganglion that is located in the cochlea of the labyrinth. The dendritic cells of the named sensory node are directed to the organ of Corti, to its hairy auditory cells. Axons emerge from the temporal bone into the cranial cavity through the pore acoustics internus and as part of the root pars cochlearis with pars vestibularis, n, facialis, and n. the intermediates enter the brain stem in the cerebellopontine angle. They end (the first auditory neuron) in two nuclei: the cochlearis ventralis nucleus (located in the ventral part of the pons) and the cochlearis dorsalis nucleus, or acousticum tuberculum (in the dorsal part of the pons). At the same level, there are a number of nuclear formations that take part in the formation of further pathways for conducting auditory stimuli (the core of the trapezoidal body, the upper olive, the core of the lateral loop). The fibers of the second auditory neurons, starting from both nuclei of the cochlear nerves and partly intersect in the bridge, passing to the opposite side, partly they go along their side of the brain stem, attaching to themselves third neurons from the above-mentioned nuclear formations (trapezoidal body, etc.). This path, called the lateral loop, the lemniscus lateralis, ends in the subcortical auditory centers located in the posterior tubercles of the quadruple and in the corpus of the geniculum medial with the optic tals. From here, from the cells of the corpus of the geniculum medial originates the last auditory neuron, the axons of which pass through the inner capsule and crown of the radiate, ending in the temporal lobe of the cerebral cortex (the posterior part of the superior temporal gyrus and Heschl's gyrus), located in the depths of the sylvian sulcus; here is the cortical auditory projection area or the cortex of the auditory analyzer.

Carrying out auditory stimuli on both sides of the brain stem (its own and the opposite) and, therefore, the representation in each lateral loop of paths from each ear explains the fact that unilateral hearing damage occurs only in the case of damage to the middle and inner ear of the partis cochlearis and its nuclei: with unilateral the same defeat of the lateral loop, subcortical and cortical auditory centers and the internal capsule - clear hearing disorders do not arise at all, because in this case, irritations from both ears are carried out into one of the hemispheres of the cortex on the unaffected side).

Pathological phenomena on the part of the hearing aid and the study of hearing are discussed in detail in the course of otolaryngology; it should be mentioned that hearing loss is denoted by the term hypacusis, its loss, i.e. deafness - anacusis or surditas and increased perception - hyperacusis. It is always important for an otiatrist and a neuropathologist to distinguish between hearing loss depending on pathological processes in the middle ear (eardrum, auditory ossicles), from “nervous” hearing loss or deafness (organ of Corti, cochlear nerve and nucleus). In the first case, a greater hearing loss in low tones and preservation of bone conduction are characteristic; in the second, loss of perception, mainly of high tones, and weakening or loss of bone conduction. Because of this, in the study of bone conduction according to the Weber method (a tuning fork is installed on the crown of the investigated person), when the sound-conducting apparatus is damaged (for example, with otitis media), the sound is more intensely perceived by the diseased ear, otherwise it is "lateralized" to the diseased side; in case of damage to the nervous apparatus of the ear - in a healthy one.

Phenomena of irritation of partis cochlearis are expressed in the occurrence of spontaneous noises, whistling, buzzing, etc. However, the same can be observed with diseases of the middle ear. When the temporal lobe cortex is irritated, auditory hallucinations can occur - from simple noises to complex sound phenomena (music, voices).

Pars vestibularis. Vestibule nerve, sensory nerve; has a vestibular ganglion located in the bottom of the internal auditory canal. The processes of the cells of this node end in the ampullae of the semicircular canals, urticulum and sacculus. Axons, entering the cranial cavity, like pars cochlearis through the acousticus internus porus, are part of the root of the partis vestibularis in the cerebellopontine angle in the brainstem and end the first neuron in the system of the nucleus of the vestibular nerve located in the pons lining on the border with the medulla oblongata in the lateral regions bottom of the IV ventricle. The most important cell group of this nucleus is the so-called Deiters nucleus and the ankylosing spondylitis nucleus, through which the vestibular apparatus (semicircular canals, sacculus and utriculus) establishes a number of connections with other parts of the nervous system. So, the vestibular nuclei are connected:

1) from the nucleus fastigia of the cerebellar vermis, mainly from its side (through the pedunculus of the cerebellum inferior, rope body);

2) through the system of the posterior longitudinal bundle, the Deiters nucleus is connected with the nuclei of the oculomotor nerves;

3) with the optic hillock and further - with the cerebral cortex (temporal lobes);

4) by the spinal cord, its anterior horns, the vestibular nucleus is connected by special conductors - the vestibulospinalis tractus;

5) with vegetative centers of the trunk, formatio reticularis, nucleus n. wagi, etc.

The vestibular apparatus is one of the organs that orientate relative to the position and movement of the body (head) in space. Its receptors, nerve and nuclei make up the peripheral part of the vestibular (vestibular-spatial) analyzer.

When it is damaged, balance disorders (connections with the cerebellum), nystagmus (with the nuclei of the oculomotor nerves), dizziness, vomiting (connections with the nucleus of N. vagia, etc.) both as a result of diseases of the inner ear, and during processes in the pontine-cerebellar corner (pars vestibularis), brain stem, cerebellum and cerebral cortex.

Have a couple, n. trigeminius. Being a mixed nerve, it has motor and sensory nuclei in the brain stem. Sensory fibers begin from a powerful Gasser node located on the anterior surface of the temporal bone pyramid between the leaves of the hard corneal sheath. The dendrites of the cells of this node are sensory fibers of the trigeminal nerve, which consists of three branches: n. ophthalmicus, n. maxillaris and n. mandibularis. The axons of the cells form the sensory root n. trigemini, which enters the bridge in its middle third, near the middle legs of the cerebellum. Further, fibers for pain and temperature sensitivity in the form of a descending root come to the nucleus of the nucleus tractus spinalis n. trigemini, where they end. The conductors of tactile and articular-muscular sensitivity do not enter this nucleus; they end in another nucleus - the nucleus terminalis, located orally to the nucleus of the descending root.

The nucleus tractus spinalis is a direct continuation of the posterior horns of the spinal cord; this long nucleus can be traced throughout the medulla oblongata; with its front end (oral), it enters the bridge, into its posterior third.

Consequently, the first, or peripheral, sensory neurons end in the nuclei. Further conduction of sensitive stimuli from the face is carried out by the second neurons, the cells of which are embedded in the nuclei. Their fibers are directed through the midline of the trunk into the trigemini lemniscus, which enters the opposite medial loop and ends with it in the optic tubercle. Due to the fact that the fibers of tactile sensitivity pass into the loop of the opposite side, without entering the nucleus of the descending root, the defeat of only this nucleus (similar to the defeat of the posterior horn) causes loss of pain and temperature sensitivity while maintaining the tactile one. Further holding the sensitivity away from the face like

And from the whole body, it is carried out by means of third neurons coming from the optic tubercle through the inner capsule and into the posterior central gyrus of the hemisphere opposite to the nucleus of the cerebral cortex.

The motor nucleus - the nucleus motorius, or the nucleus masticatorius, is located in the dorso-lateral section of the bridge tire; its fibers leave the bridge in the form of a thin root, next to the sensitive, adjoin the Gasseri ganglion and join

the III branch of the nerve, i.e. to n. mandibularis, as part of which they are sent to the chewing muscles. Thus, only the III branch of the trigeminal nerve is mixed, i.e. sensitive motor; the first two branches are purely sensitive.

1.N. Ophthalmicus leaves the skull through the fissure orbitalis superior, supplies sensitive endings to the skin of the forehead and the anterior scalp, upper eyelid, inner corner of the eye and the back of the nose, the eyeball, mucous membranes of the upper part of the nasal cavity, frontal and ethmoid sinuses, meninges ...

In N. ophthalmic neuralgia, the pain point is felt in the foraminis supraorbitalis area.

11. N. maxilaris emerges from the skull through the rotundum foramen. Provides sensitive endings to the skin of the lower eyelid and the outer corner of the eye, part of the skin of the lateral surface of the linden tree, the upper part of the theca, the upper lip, the upper jaw and its teeth, mucous membranes of the lower part of the nasal cavity, the maxillary cavity.

With neuralgia of the II branch, a painful point is felt in the infraorbital foramen, where n. infraorbitalis from n. maxilaris.

III. N. mandibularis - mixed nerve;

a) sensitive fibers innervate the lower lip, lower

part of the cheek, chin, back of the lateral surface of the face,

the lower jaw, its gums and teeth, the mucous membranes of the cheeks, lower part of the oral cavity and tongue;

b) motor fibers innervate the chewing muscles - m. masseter, m. temporalis, mm. pterygoid externa and interni m. digastricus (anterior abdomen).

With neuralgia of the III branch, one of the pain points is the mentale foramen, from where N. mandibularis - n. mentalis.

Sympathetic and parasympathetic fibers from special ganglia located in the n system are attached to an extensive network of branches of the trigeminal nerve. trigemini. For 1 branch - n. ophthalmicus - such a node is the ciliary ganglion located in the orbit: for II-n. maxilaris - sphenopalatinum ganglion, located in the fossa of pterygopalatin, for III - n. mandibularis - oticum ganglion (below the oval foramen).

Sympathetic fibers originate from the corresponding arteries of the perivascular plexus (mainly from the plexus carotikus) and consist of vasomotor, secretory and trophic fibers.

When one of the branches of the trigeminal nerve is damaged, sensitivity disorders (anesthesia, hyperesthesia, etc.) occur in the zone innervated by this branch; the corresponding reflexes fade away or decrease. So, with a violation of conduction , the corneal reflex disappears.

When the Gasser node or root of the V nerve is involved in the process on the basis of the brain, loss of sensitivity is observed in the area of the zone of all three branches. And in this case, and with an isolated lesion of one of the branches, pains (trigeminal neuralgia) are usually observed, often reaching exceptional intensity. At the same time, pressure on the painful points of NN is sometimes painful. supraorbitalis, infraorbitalis and mentalis. Due to the presence of

sympathetic fibers in the V system of the seal, sweating disorders, vasomotor phenomena, trophic disorders, especially dangerous areas of the cornea (the so-called neuroparalytic keratitis) are possible; when the gasser's node (ganglioneuritis) is involved in the process, the appearance of herpes zoster is observed.

There is a special form of trigeminal neuralgia - quinty major neuralgia - with extremely severe pains that come on in paroxysms, but with usually mild nerve conduction disturbances; the disease is characteristic mainly of old age.

Differential diagnosis between the defeat of the root of the V nerve and the nucleus tractus spinalis N. trigemini is based on the same data that distinguish the lesion of the spinal posterior sensory root from the posterior horn. When the root is involved in the process, there are disorders of all types of sensitivity on the face, as well as pain. For the defeat of the nucleus, pain is less characteristic; on the face there are dissociated or split sensory disorders, i.e. loss of pain and temperature while maintaining tactile feeling. Finally, with incomplete damage to the nucleus, segmental annular zones of anesthesia can be seen: with damage to the oral, late part of this nucleus, sensitivity falls out in the circumference of the nose and mouth; in case of violation of the integrity of the caudal, posterior part of the nucleus, the anesthesia strip adjoins the zones of the upper cervical segments, located in the lateral circumference of the face and leaving its oral part free.

When the motor fibers of the III branch, motor root or motor nucleus are affected, paralysis of the masticatory muscles develops on the affected side. The onset of this atrophy of M. masseteris and M. temporalis is detected when probing the muscles below and above the zygomatic arch after the subject clenches the jaw; when the mouth is opened, the jaw shifts towards the weak muscle. A rebirth reaction is found in the masticatory muscles.

Central paralysis of the masticatory muscles is also possible with supranuclear lesions of the pathways of the central neurons connecting the lower part of the anterior central gyrus of the cortex with the motor nuclei of the V nerve in the bridge. However, with a one-sided process, no functional impairments occur. This is explained by the approach of central fibers to each nucleus from both hemispheres of the cortex (bilateral cortical innervation). There is a complete analogy here with what we saw in relation to the cortical innervation of the nuclei of the bulvar. As with pseudobulbar paralysis, central chewing paralysis is only bilateral if both supranuclear pathways are affected .

Spasms of the masticatory muscles are bilateral. Topical spasm - trismus - is characteristic of tetanus, rabies, tetany. In addition, convulsive phenomena in the masticatory muscles are sometimes found with trigeminal neuralgia and as a manifestation of subcortical and cortical hyperkinesis.

ORAL MOTOR NERVES

VI pair, n. abducens - motor nerve. Nucleus (motor) n. The abducentis is located dorsally in the pons varoli at the bottom of the rhomboid fossa, under the eminence known in the descriptive name as the colliculus facialis, or eminence teres. The elevation is formed due to the presence here of the fibers of the facial nerve braiding from above the nucleus of the VI nerve, as already mentioned

above. Radicular fibers are directed from the nucleus to the base and emerge as a stem at the border of the bridge and the medulla oblongata at the level of the pyramids. Further, the nerve is directed forward and through the fissure of the orbitalis superior (it leaves the cranial cavity into the orbit, where it innervates the only muscle - m. Rectus externus (lateralis - PNA), which turns the eyeball outward.

Nuclear lesions are usually accompanied by peripheral paralysis or paresis of the facial muscles and alternating (on the opposite side) central paralysis of the limbs - the so-called Fauville syndrome. In addition, nuclear or near nuclear foci entail not only paralysis of the rectal externa, but also gaze paralysis in the direction of the affected muscle or focus.

When a nerve or root is damaged, an isolated paralysis of the rectal externa occurs at its base, which causes a converging strabismus, the inability to turn this eyeball outward, double vision (diplopia), especially when looking towards the affected muscle, sometimes dizziness, forced position of the head.

| IV couple, n. trochlearis is a motor nerve. Nerve fibers emanate from the nucleus located in the bottom of the Sylvian aqueduct at the level of the posterior tubercles of the quadruple. The peculiarities of the exit of this nerve from the brain consist in the fact that the fibers from the cells of the nucleus are directed upward, bypass the sylvian aqueduct and exit not on the base, but dorsally, crossing in the anterior cerebral sail. Coming out behind the quadruple, the nerve goes around the cerebral peduncle and along the base of the skull passes to the fissure of the orbitalis superior, through which it throws the skull, innervating the only muscle in the orbit - M. appearanceus superior, which turns the eyeball outward and downward.

With an extremely rare isolated lesion of n. trochlearis is marked by converging squint and diplopia only when looking down. The patient's complaint of double vision is very characteristic only if he looks at his feet (for example, when descending a staircase); when looking straight ahead, up and to the sides, diplopia does not occur.

III pair, n. oculomotorius is a motor nerve. Core n. the oculomotor is located at the bottom of the Sylvian aqueduct, at the level of the anterior hillocks of the quadruple. Fibers from the cells of the nucleus go mainly to their (partly to the opposite) side, go down and go out to the base of the brain, at the border of the bridge and legs from the medial side of the latter. The nerve leaves the skull together with the IV and VI nerves and n. ophthalmicus n. trigemini through the fissure of the orbitalis superior, innervating 5 external (striated) and 2 internal (smooth) muscles.

Kernels n. Oculomotoria consist of five cell groups: two outer large-cell nuclei, two small-cell nuclei (Yakubovich) and one internal, unpaired, small-cell nucleus (Perlia).

From the paired outer large cell nucleus, fibers for the following outer mice emanate:

1) levator palpebre Superior - raises the upper eyelid;

2) rectus superior - turns the eyeball up and a little

inside;

3) rectus internus - moves the eyeball inward;

4) the appearance of an inferior - turns the eyeball upward and somewhat outward;

5) the rectus inferior moves the eyeball downward and somewhat inward.

From the paired small-cell (parasympathetic) nucleus of Yakubovich, fibers go to the smooth internal muscle of the eye - m. Sphincter pupillus, which constricts the pupil.

Parasympathetic fibers for the ciliary muscle (accommodation function) emerge from the unpaired inner small-cell (accommodative) nucleus.

Fibers from both paired and unpaired small-cell nuclei do not directly reach the sphincteris pupille and m. Ciliaris, but are interrupted in the ganglion ciliary, from where the fibers of the second neuron (non-fleshy) conduct impulses to the named muscles. With complete paralysis, the following is observed.

1. Ptosis - the eye is closed by the pubescent upper eyelid.

2. The eyeball is turned outward and slightly downward (as a result of the preserved m. Rectum externus and m. Superior appearance from the VI and IV nerves); therefore, there is a divergent squint.

3. When the upper eyelid is raised, diplopia is noted.

4. The pupil is dilated (antagonistic action of M. dilatoris pupille from N. sympathicus).

5. There is paralysis of accommodation (M. ciliaris is affected), why and vision at close distances deteriorates.

6. Convergence, upward movement of the affected eyeball and inwardly are impossible; its downward movements are significantly limited.

7. The eye is slightly out of the orbit (due to the loss of tone of a number of external muscles of the eye) - exophthalmus.

In processes inside the brain stem (in the brain stem), paralysis is usually accompanied by central paralysis of the opposite limbs (alternating Weber's syndrome) due to damage simultaneously with the fibers of the third nerve of the pyramidal pathways, making the cross below. With foci located at the same level, but more dorsally, with involvement of the Phase nucleus in the process, Benedict's alternating syndrome (111 nerve palsy and cerebellar ataxia in opposite limbs) is observed.

If a lesion is added to the complete paralysis of the 111 nerve. abducentis and n. trochlearis, then the movements of the eyeball are absent at all ophthalmoplegia, or a set.

With an isolated lesion of only small-cell nuclei, the so-called intern's ophthalmoplegia (loss of functions of only internal muscles) is observed, and with damage only to the outer large-cell nuclei, external ophthalmoplegia. With the defeat of N. oculomotoria, too, a picture of its complete paralysis is not always observed; perhaps a more limited loss of functions due to a violation of the conduction of only a part of the nerve fibers.

Paralysis of individual oculomotor nerves (III, IV and VI nerves) are always peripheral. Only with bilateral and, moreover, extensive supranuclear processes that turn off the central neurons going from both hemispheres to the nuclei, bilateral ophthalmoplegia of the central type can occur, since, by analogy with most motor nuclei of the cranial nerves, the nuclei of the III, IV and VI nerves have bilateral cortical innervation.

When examining the nerves of the eye muscles, some data can be obtained already with a simple external examination. So, when levatoris palpebre superioris (III nerve) is affected, ptosis is visible - drooping of the upper eyelid: divergent squint indicates insufficiency of the rectal medialis (III nerve), converging - to the insufficiency of the rectal lateralis (VI).

It is essential that the patient indicates that he has double vision (diplopia) - a sign that is sometimes more subtle than the objectively established insufficiency of one or another external muscle of the eye.

In case of complaints of diplopia, it is necessary to find out which muscle (or nerve) is affected by this disorder. Recall that diplopia occurs or worsens when looking towards the affected muscle. Insufficiency of the external and rectus internal muscles will cause double vision in the horizontal plane, and other muscles in the vertical or oblique planes. Even more clearly, diplopia is revealed if one of the patient's eyes is covered with colored glass. A noticeable sometimes, immediately, the difference in the size of the pupils (anisocoria), as well as their deformation, should alert the attention of the researcher, but by no means always prove the presence of a lesion of N. oculomotoria (possible congenital features, consequences of an eye injury or inflammation, asymmetry of sympathetic innervation, etc.).

In the future, the subject is invited, without moving his head, to follow his gaze moving up, down, right and left with a finger or a hammer, and paralysis or paresis of any external muscle or eye or gaze can be detected. When abducting the eyeballs to the sides, nystagmus is usually also examined. Convergence and the concomitant constriction of the pupils (the reaction of the pupils to accommodation by “convergence”) is investigated by transferring the gaze from a distant distance to an object placed close in front of the eyes (examining finger, hammer).

The study of the pupillary response to light is extremely important. Both direct and friendly reactions of everyone are tested.

pupil separately. The patient's face is turned towards the light source, the eyes are open; the investigator, first closing both eyes of the subject with his palms tightly, takes away quickly one of his hands, thus observing the direct reaction of the given pupil to light; the other eye is also examined. To detect a friendly reaction, one eye of the subject is closed with a palm; in the remaining open, there is an expansion of the pupil; when the hand is removed from the closed eye in both, there is a simultaneous sympathetic constriction of the pupils. The same is done for the other eye. An electric lamp with a switch or a pocket electric torch are very convenient for studying light reactions (especially in bedridden patients). The

complete loss of pupillary responses to both light and accommodation with convergence is called complete immobility of the pupil.

Loss of reaction to light with the preserved possibility of narrowing during convergence with accommodation is called Argyll Robertson's symptom and is observed almost exclusively in dorsal tabes and progressive paralysis. Much less often, the opposite is observed: the preservation of the reaction of the pupil to light in the absence of a reaction to accommodation with convergence.

VZOR INNERVATION, REAR LONGITUDINAL BEAM SYSTEM.

There is no isolated movement of one eyeball. Eye movements are always simultaneous and combined, which requires the joint movement of several external eye muscles, which are innervated by different nerves. When looking upward, four muscles innervated from the four cell groups of the nuclei of the third nerves contract simultaneously; when looking down - two muscles innervated by the third nerves, and two from the fourth nerves; when looking to the side, there is a simultaneous contraction of m. recti-lateralis (VI nerve) of one eye and m. recti-medialis (III nerve) of the other eye: when the eye axes converge, both mm are reduced. recti medialis from nuclei nn. oculomotor; finally, a number of other combined muscle contractions occur with "oblique" directions of gaze, for example, to the right and up, etc. If we also take into account that with the contraction of any oculomotor muscles, the tone of the corresponding antagonist muscles should simultaneously decrease, then the need for a very delicate and precise innervation system that regulates eye movements will become clear.

Both reflex and voluntary movements of the eyeballs are always associated and joint. This weight is due to the presence of a special connecting innervation system, which provides both internuclear (III, IV, VI nerves of both sides) connections, and connections of the nuclei of the eye of the mouse with other parts of the nervous system.

Such a system is the posterior longitudinal bundle. The beam nuclei, or Darkshevich nuclei, are located anterior to the NN nuclei. oculomotoria, near habenula and posterior commissure.

The fibers of both bundles are directed down the brain stem, located in the bottom of the Sylvian aqueduct and the rhomboid fossa dorsally, on the sides and close to the midline, and give collaterals to the cells of the nuclei of the III, IV and VI pairs of nerves, which ensures the compatibility and simultaneity of movements of the eye muscles in this or that combination.

Other fibers that make up the posterior longitudinal bundle are fibers from the cells of the vestibular nucleus, directed into the bundle of both its own and the opposite side. They branch into ascending and descending branches, heading upward, in contact with the cells of the nuclei of the eye of the mouse; descending - descend into the spinal cord, pass through it as part of the anterior pillars and end near the cells of the anterior horns - the vestibulospinalis tractus.

"Arbitrary" innervation of the gaze is carried out from the so-called center of arbitrary rotation of the eyes and head in the opposite direction, located in the

posterior part of the second frontal gyrus. Fibers from the bark, approaching the bridge in its anterior section, cross and end near the core n. abducentis of the opposite, therefore, side. From the nucleus of the VI nerve, the impulse simultaneously spreads along the nerve to the rectus externus (lateralis), and to the cell group of the 111 nerve, which gives fibers to the rectus internus (medialis) of the other eye, which causes a combined rotation of the eyeballs towards this nucleus (“ bridge gaze center "), but in the opposite hemisphere where the impulse arose. Consequently, when the second frontal gyrus is damaged, gaze paralysis is observed in the opposite direction, and when the bridge is damaged distally from the intersection of neutral fibers in it or the nucleus itself. abducentis observed paralysis of the gaze in the direction where the lesion is located. In both cases, due to the predominance of unaffected antagonists, a combined deviation of the eyeballs and the head may occur when the bridge is damaged in the direction opposite to the focus; with the defeat of the cortical sections - towards the focus. With irritation of the posterior part of the second frontal gyrus (Jacksonian epilepsy), tonic-clonic convulsions of the eye muscles and head are observed in the direction opposite to the focus of irritation.

The localization of the cortical projection (paths) of turning the eyes up and down is not well understood; apparently, it is located near the projection of the turn to the side, at the base of the same second frontal gyrus. Fibers from here enter the system of the posterior longitudinal bundle through the n nuclei. oculomotoria. Processes in the area of the anterior colliculus - nuclear (III nerves) and perinuclear - are often accompanied by paralysis of the gaze up and down, in the same way as the foci in the bridge or in the region of the nuclei of the VI nerves cause paralysis of the gaze to the side.

.In case of damage to the posterior longitudinal bundle, in addition, nystagmus is observed.

The connections just examined determine the innervation of the gaze from the cerebral cortex. Through the vestibular nucleus, the posterior longitudinal bundle establishes connections with the vestibular apparatus and the cerebellum. Connections with the extrapyramidal system are apparently carried out; through the Darkshevich kernels. Descending fibers of the posterior longitudinal ray determine connections with the spinal cord. Finally, there are connections between the nuclei of the eye muscles with the subcortical centers of vision and hearing (anterior and posterior tubercles of the quadruple), which causes an "involuntary" reflex turn of the eyes and head towards visual or auditory stimulation.

VISUAL AND OTHER NERVES.

II pair, n. opticus is a sensory nerve. The optic nerve (as well as the olfactory nerve analyzed below) differs significantly from the rest of the cranial nerves in its morphological features. Being, in essence, a reduced lobe of the brain, n. opticus in its structure is closer to the conductors of the central nervous system than to the spinal nerves.

The fibers of the optic nerves start from the ganglion cells of the retina (we do not dwell on the details of the structure and function of the retinal nervous apparatus); through the foramen opticum, the optic nerves enter the cranial cavity, go to the base of the brain, and here, anterior to the sella turcica, they cross, forming the chiasm nervorum opticorum. The intersection is partial, since only the fibers coming from the nasal (inner) halves of the retinas are exposed to it; the fibers from the outer or temporal halves do not cross the chiasm. After chiasma, the visual pathways are called the optic tracts. In the optic tracts (as well as in the optic nerves), fibers from individual fields of the retina are located in certain sections of the cross-section. So, fibers from the upper fields of the retina go to the upper parts of the nerve and tract; fibers from the lower fields of the retinas - in the lower sections. It matters

to clarify the "course" of the process, spreading to the optic tracts or nerves (for example, tumors). It should be borne in mind that in the event of a lesion, the fields of vision that are opposite to the dropped out field of the retina always fall out. As a result of those features of the intersection, which have just been mentioned, fibers pass in the optic tract not from one eye, as in the optic nerve, but from the same halves of the retinas of both eyes, for example: in the left optic tract from both left halves of the retinas. It should be recalled that the refractive media of the eye (lentils, vitreous body) "project onto the retina a reverse image of the visible, and, therefore, the left optic tract conducts irritations from the right, and the right tract from the left visual fields of both eyes.

The subcortical part of the visual analyzer includes: pulvinar (pillow) of the optic tubercle, the corpus of the geniculatum lateral with the optics (lateral geniculate body) and the anterior tubercles of the quadruple (reflex center). The next neurons, conducting visual stimuli into the cortex, begin only from the corpus geniculatum lateral to the optic cells. Fibers from its cells pass through the inner capsule, in the posterior part of the posterior thigh and as part of the Graziole beam, or radiation optics, and end in the cortical visual areas. The named paths are projected onto the inner surface of the occipital lobes, into the calcarine fissure region, and also into the depth of the groin groove - the nucleus of the visual analyzer. Thus, both the optic tracts, and the primary visual centers, and radiation optics, and the cortical territories in the area of the spur sulcus (sulzi calcarini) are associated with the same halves (their side) of the retinas of both eyes, but with opposite, therefore, halves of the visual fields.

In the area above the sulzi calcarini, i.e. in the cuneus, the upper quadrant of the retinas of the same side is presented; in the downward region, i.e. into the lingualis girus, - the lower one. The macular, or central, field of the retinas is apparently associated with the posterior parts of the spur sulcus (according to some authors, in its depth).

In the anterior tubercles of the quadruple, the so-called pupillary fibers of the optic nerves end, (representing the first link of the reflex arc of the pupil's reaction to light. From the anterior tubercles, the following neurons go to the Yakubovich nuclei (to the paired small-cell nuclei of the III nerve) of both their own and the opposite side, which determines a friendly or sympathetic reaction of the other

pupil when only one eye is illuminated; the further path is from the cells of Yakubovich's nuclei according to N. oculomotorius to the ganglion ciliary; the last neuron is from the cells of the ganglion to the M. sphincter pupilla. , establish, apparently, reflex connections with the interstitial and midbrain (somatic and visceral reflexes).

With a complete interruption in the conduction of the optic nerve, blindness occurs in the given eye (amaurosis) with the loss of the direct reaction of the given pupil to light (the pupil of the blind eye narrows to light only friendly when the second, healthy eye is illuminated). Decreased vision is called amblyopia. When only a part of the optic nerve fibers is damaged, the visual field is limited, the loss of sectors or islets in it (scotomas).

With the complete destruction of the chiasm, complete bilateral blindness occurs. But in a number of processes, the lesion of the optic chiasm can be limited. So with pituitary tumors, expansion as a result of hydrocephalus and stretching of the third ventricle, pressure can only act on the middle of the chiasm, on its crossing fibers from the inner nasal halves of the retinas of both eyes. In this case, the external or temporal fields of vision will be blind, i.e. the so-called temporal, or bitemporal, hemianopsia will occur, which is opposite (in one eye the right field of view falls out, in the other - the left field of vision. also dissimilar, but already binasal hemianopsia with loss of both internal visual fields.

Much more often there are so-called homonymous, or homonymous, hemianopsias, which occur when the visual pathways and centers are damaged posterior to the optic nerve intersection, i.e. with damage to the optic tracts, the optic tubercle, the inner capsule in its posterior part and the occipital lobe.

Starting from the optic tract, stimuli are conducted and perceived in the pathways and centers; in the right - from the right and in the left - from the left halves of the retinas of both eyes. With a break, there is a hemianopsia of the same name of opposite visual fields; for example, with a lesion on the left - right-sided hemianopsia of the same name, etc.

There are some strong points for distinguishing the seemingly identical hemianopsia in lesions: 1) the optic tract or subcortical visual center and 2) the capsule (radiation optics) or cortex (sulcus calcarinus). The hallmarks for these hemianopsias will be as follows:

Tractus hemianopsia: 1) simple atrophy of the optic nerves; 2) no reaction of the pupils when illuminating the switched off polonium of the retinas; 3) with partial homonymous hemianopsia, there is often a pronounced asymmetry of visual field defects.

Central hemianopsia: 1) no atrophy of the optic nerves;

2) the pupils react to light when both halves of the retinas are illuminated;

3) defects of the visual field, as a rule, are symmetrical. In the presence of asymmetry, it is not sharply expressed.

With incomplete damage to the cortical projection visual area or the visual pathways leading to them, only quadrant hemianopsia may occur. So, if, for example, the left cuneus is destroyed, only the left upper quadrants of the retina will be "blind" and only the right lower quadrants will fall out, respectively, in the

fields of view; with a focus in the area of the right gyrus lingualis, the left upper quadrants of the visual fields fall out, etc.

In rare cases, both cineus are involved in the process symmetrically, while the areas below the sulzicalcarini remain intact, i.e. giri linguales, or vice versa. In such cases, the so-called lower (when both cinerius are turned off) or upper hemianopsia are observed .

When the area of the furrow is irritated, visual hallucinations occur in opposite fields of vision, such as simple photomas, which are usually the aura of a seizure of cortical epilepsy that develops after them. With irritation, not the sulzi calcarini area, but the outer surface of the occipital lobes (i.e. closer to the area where the visual analyzer meets other analyzers), visual hallucinations are of a more complex type: figures, faces, cinematic pictures, etc.

For neurological diagnostics, it is necessary to study not only the fields of vision and its acuity, but also the fundus. Visual acuity is determined using Kryukov tables, visual acuity - by the perimeter. The presence of hemianopsia can be detected by other, simpler, although inaccurate techniques. So, when asked to show with a finger the middle of a stick or a towel or cord stretched in his hands, the patient does not halve the entire length, but only three quarters of it, since about a quarter of the stick falls out of the field of view from the edge where there is hemianopsia. Threatening finger movement from the "blind" side towards the eye does not cause protective blinking. From the defective side of the visual field of each eye, the patient does not notice the movements of the fingers made by the researcher.

Of the changes in the fundus that are important in neurological diagnostics, one should especially highlight: 1) optic neuritis (in inflammatory processes); 2) its atrophy (with dorsal tabes, pituitary tumors, etc.) and 3) congestive nipple (with an increase in “intracranial pressure).

I pair, n. olfaktorius, a sensory nerve. As in the visual tract system, the cells of the first olfactory neurons are located in the periphery, not in the ganglion. Olfactory bipolar cells are scattered in the mucous membrane of the upper portions of the superior concha and nasal septum. The axons of these cells in the form of thin filaments enter the cranial cavity through the cribrosa lamina and end with the olfactory bulbi, which are located at the base of the brain in the anterior cranial fossa. From the bulbus olfactorius, where the cells of the second neurons are laid, the fibers in the tractus olfactorius go further posteriorly and end in the so-called primary olfactory centers

The third neuron conducts olfactory stimuli from the aforementioned primary centers to the cortical projection territories of smell located in the hippocampal girus of the temporal lobe (mainly in its uncus), the ammonic horn, the dentatus girus, etc. olfactory function, but also to autonomic-visceral regulation, as well as to the emotional sphere.

These paths reach the cortex in various directions, mainly above and below the corpus callosum, and also through the fasciculus uncinatus. It is important to note that the primary olfactory centers are associated with the cortical areas

(temporal lobe) of both their own and the opposite side; the transition of a part of the fibers to the other side occurs through the anterior commissure.

This circumstance explains the absence of anosmia or hyposmia in unilateral lesions of the cortical centers.

Bilateral disorders of the sense of smell are not of great importance in neurological diagnostics, since they are too often the result of diseases of the nasal cavity and nasal passages or are congenital.

Pathological processes in the frontal lobe and at the base (anterior cranial fossa) lead to unilateral anosmia or hyposmia (loss or decrease in the sense of smell). Irritation of the temporal lobe sometimes causes olfactory hallucinations, which are usually the aura of a seizure of cortical or Jacksonian epilepsy that follows them.

The study of smell is carried out using a set of bottles with aromatic substances (clove oil, camphor, etc.); each nasal passage is examined separately. Harsh irritants, such as ammonia, acetic acid, should be avoided, since the resulting irritations are also perceived by the trigeminal nerve receptors.

Cerebellar lesions and movement coordination disorders

The purpose of the lesson is to study the structural and functional features of the cerebellum, its role in the motor analyzer system, lesion syndromes, methods of studying its function.

The student should know:

features of the development of the cerebellum in onto - and phylogeny, its structural and functional features; afferent and efferent connections, participation and role of the cerebellum in the motor analyzer system;

ensuring coordination of movements, plastics, proportionality of movements, muscle tone;

somatotopic projection in the cerebellum, syndrome of damage to the hemispheres and the cerebellar worm, ascending and descending its conduction systems;

features of cerebellar disorders in various forms of nosological pathology.

The student should be able to:

analyze the patient's complaints to assess the appearance, posture, gait, speech of the patient, handwriting, the ability to follow instructions;

to investigate the functions of the cerebellum using special tests;

to solve problems of topical diagnosis of lesions of the cerebellum and its afferent and efferent systems.

Control questions.

1. What structures of the brain are united by the extrapyramidal system?
2. What are the anatomical - physiological features of the cerebellum?
3. What are the pathways of the extrapyramidal system and the cerebellum?
4. How are the neural connections between the cerebellum and the striopallidal system ensured?
5. How is the neural connection between the cerebral cortex and the cerebellum ensured?
6. What symptoms are observed with damage to the worm and cerebellar hemisphere?
7. What symptoms can be observed with damage to the striatal system?
8. What are the methods for studying the function of the cerebellum and the strio-pallido-nitral system?
9. What symptoms are observed with damage to the pallid-nigral system?
10. What are the symptoms of parkinsonism?
11. What is the difference between cerebellar ataxia and sensitive, vestibular and frontal ataxia?

The cerebellum is located in the posterior cranial fossa above the medulla oblongata and the pons varoli. Above it are the occipital lobes of the large brain. The cerebellum consists of a middle section, or worm, and two hemispheres. Its surface layer is the bark (gray matter). In addition, in the white

thing there are also accumulations of gray matter - the nucleus of the cerebellum, of which the nuclei dentati and nuclei tecti are more important. The cerebellum is connected with other parts of the central nervous system by three pairs of legs: 1) lower legs, rope bodies; 2) medium legs; 3) upper, or front, legs.
The phylogenetically more ancient part of the cerebellum is the worm, which is closely associated with the vestibular apparatus.
The cerebellar hemispheres are a newer formation that developed in parallel with the development of the cerebral cortex and in connection with the complication and improvement of motor acts.
The inclusion of the cerebellum in the system of coordination of movements is provided by its afferent connections with numerous proprioceptors embedded in the organs of movement, and with the vestibular apparatus, which perceives changes in the position of the body (head) in space. The efferent influences of the cerebellum on the striated muscles are carried out through special pathways terminating in the cells of the peripheral motor neuron. Connections of the cerebral cortex with the cerebellum ensure the inclusion of the latter in the regulatory system that carries out "voluntary" movements. Lesions of the cerebellum or its connections are accompanied by disorders of coordination of movements, muscle tone and balance.

WAYS TO THE CEREBELLA FROM THE SPINAL AND OBLONVOUS CORD

Spinocerebellar pathways

Nerve endings are located in the muscles, joints, ligaments, tendons and periosteum, the conductors from which go, in particular, to the cerebellum. Cerebellar proprioception provides constant information about the state of the organs of movement. The impulses arising in these organs, along the centripetal fibers of the peripheral nerve, reach the cells located in the intervertebral spinal ganglion, from where, along the corresponding fibers of the posterior sensory root, they are conducted to the base of the posterior horn of the spinal cord. Here the first (peripheral) neuron of the cerebellar proprioceptors ends, and the impulse is transmitted to the cells of the second neurons.
Flexig's bundle starts from the cells located at the base of the posterior horn. The axons of these cells extend into the lateral column of their side and are directed up the spinal cord, located on the periphery of the posterior part of the lateral column. Reaching the medulla oblongata, Flexig's bundle in the form of fibrae arcuatae externae as part of the lower legs of the cerebellum or congpus restiforme enters the cerebellum and ends in its worm.
The Govers bundle begins from the cells of the middle part of the gray matter of the spinal cord; axons also go out into the lateral column and are located on the periphery of it, anteriorly (ventrally) of the Flexigus bundle. The fibers of the Govers bundle pass the dorsal, medulla oblongata, pons varoli; in the anterior part of the latter, they turn first upward, then posteriorly and, as part of the upper legs of the cerebellum, enter, like the Flexigus bundle, into the cerebellar worm, where

they end. With the defeat of the Flexig and Govers bundles and the congus restiforme, cerebellar disorders occur on the affected side.

Vestibulocerebellar pathways
The axons of the cells of the vestibular nucleus (mainly the ankylosing spondylitis) enter through the lower legs of the cerebellum, i.e. through congus restiforme into the cerebellar worm, in particular in its nucleus tecti.
There are also similar paths from the sensory nuclei of the trigeminal and vagus nerves, as well as from the reticular formation.

Paths from the cores of the rear pillars
From the nuclei of Gaulle and Burdach, most of the fibers, as is known, in the composition of the tractus bulbothalamicus (articular-muscular and tactile sense) are directed to the optic tubercle. Some of the axons from the cells of the nuclei of the posterior columns in the form of fibrae arcuatae exsternae posteriores also pass through the corpus restiforme into the cerebellar worm.

Olivecerebellar pathway
From the inferior olives through the corpus restiforme, the conductors of the extrapyramil system pass to the cerebellar cortex.

Pathways from the cerebral cortex
From various parts of the cerebral cortex, mainly from the frontal lobes, tractus corticocerebellares begins, consisting of two neurons: tractus corticopontinus and tractus pontocerebellaris
1. The frontal path of the bridge, originates from the anterior sections of the superior and middle (1 and II) frontal gyri, passes through the centrum semiovale, the anterior femur of the inner capsule, the inner section of the base of the pedicles and ends in the nuclei of the bridge of its side.
2. The occipital-temporal path of the pons, starts from the posterior sections of the II and III temporal gyri and the occipital region of the cortex; in the inner capsule, it is located in the posterior part of the hind thigh, in the outer part of the base of the legs of the brain; also ends in the cores of the bridge on its side.
The pons nucleus cells are second neurons; their axons cross at the base of the bridge and, under the name tractus pontocerebellares, enter the opposite hemisphere of the cerebellum through the middle legs of the cerebellum, where they end in its cortex. The next neuron connects the cortex of the cerebellar hemisphere with the nucleus dentatus.
Thus, the cerebral hemispheres are connected with the opposite cerebellar hemispheres. Cerebellar disorders arising from damage to the cortex (mainly the frontal and temporal lobes) are found on the side opposite to the focus.

Paths from the cerebellum

The lower and middle pedicles of the cerebellum consist mainly of conductors going to the cerebellum; the upper legs are the path along which its centrifugal fibers depart from the cerebellum.

The main conductors of the upper lobes are tractus dento-rubralis.

Paths from the nucleus dentatus, making a cross (Werneking) and ending in opposite red nuclei and partly heading further to the visual tubercle. From the red nuclei, the rubrospinalis tractus begins, coming from the cells of the red nucleus, making a cross (Trout) immediately after leaving the nuclei and descending along the brain stem and spinal cord. Located in its lateral column in front of the corticospinalis tractus, the oubrospinalis tractus ends in the anterior horns of the spinal cord, establishing contact with the cells of the peripheral motor neurons and, consequently, with the muscles. This is the main cerebellofugal pathway that establishes connections between the cerebellum and the musculature.

In addition, pathways go from the red nuclei through the optic tubercle to the extrapyramidal system (connecting it with the cerebellum) and to the cerebral cortex. The number of cerebellofugal conductors also includes the fibers coming from the nucleus of the cerebellum to the reticular substance of the medulla oblongata and to the vestibular nucleus.

From the nucleus of the vestibular nerve, the fibers of the tractus vestibulospinalis begin, descending along the periphery of the anterolateral column of the spinal cord downward and ending, like the fibers of the tractus rubrospinalis, in the cells of the anterior horns. Thus, the connection of the cerebellum with the spinal cord, its segments and muscles is carried out not only through the rubrospinal, but also through the vestibulospinal pathways, possibly through the reticulosninal pathways.

Finally, through the same vestibular (Deuter's) nucleus, the cerebellum is connected through the posterior longitudinal bundle with the nuclei of the oculomotor nerves and, consequently, with the oculomotor muscles.

Analyzing the listed connections, we can be convinced of the following:

1. The cerebellum receives continuously flowing impulses from the joints to the muscles of the whole body: trunk, limbs, head, eye muscles, etc., as well as from the vestibular apparatus. These impulses reach the cerebellum mainly through the lower legs and end in the more ancient parts of it - in the worm.

Reverse regulatory impulses go through the upper legs to the red nuclei and through the tractus rubrospinalis, vestibulospinalis and the posterior longitudinal bundle reach the cells of the anterior horns of the spinal cord (or motor nuclei of the cranial nerves) and through the peripheral motor neurons reach the musculature. The named closed system (worm) serves as a reflex system of balance of the body (trunk) and the "roots" of the lower extremities.

2. The cerebellum is included in the extrapyramidal system, sending its impulses through the upper legs to the red nuclei, and from there through the optic hillocks to the striatum and palidum. The return path to the musculature passes through the same rubrospinal, vestibulospinal pathways, the posterior longitudinal fasciculus and the tecto-spinal path.

3. The cerebellum is connected with the cerebral cortex: from the cerebellum, from the cortex
his hemispheres to the nucleus dentatus; from there through the upper legs - to the red nucleus, the optic tubercle and, finally, to the cortex. From the cerebral cortex, mainly from the frontal lobes, there are tractus corticocerebralis, reaching the cerebellar cortex through its middle legs. The further path goes to the nucleus dentatus and through the upper legs to the red nuclei. The path to musculature is the same rubro-spinal (Monakovsky) bundle, etc.

New systems of the cerebellum and its hemisphere are connected with the cerebral cortex. Here, apparently, the regulation of limb movements is presented.

In the considered cerebellar system, there are three main intersections:

1) upper legs - Werneking;
2) rubrospinal (Monakov) paths - Trout;
3) middle pedicles of the cerebellum - ponto-cerebellar fibers.

Due to the presence of these crossovers, it becomes clear that cerebellar disorders occur when: 1) the cerebellum itself - on the side of the focus; 2) the cerebral cortex and red nuclei - on the opposite.

There is a somatotopic projection in the cerebellum. It should be assumed that the musculature of the trunk is represented in the worm, in the hemispheres - of the limbs, especially their distal parts.

SYMPTOMOKOMPLEX OF CEREBELLUM DAMAGE

The function of the cerebellum is to reflexively maintain muscle tone, balance, coordination and synergy of movements. With damage to the cerebellum, a number of movement disorders of an atactic and asynergic nature arise.

1. **Gait disorder** . Atactically, the cerebellar, or so-called "drunken" gait is the result of not only imbalance, but also asynergy. The patient walks with his legs wide apart and staggering, which is especially pronounced when turning. Deviation to the side when walking, and in severe cases, the fall is observed more often in the direction of the cerebellar lesion.
2. **Intentional tremor** is observed during movement and is absent at rest. It is found most sharply at the end of the movement and is examined in the hands with the help of the finger-nose, and in the legs with the help of the calcaneal-knee test. The patient is given the task with closed eyes to hit the tip of his nose with his index finger; the closer to the target, the more clearly and sharply the tremor of the finger or the entire hand and arm is detected . The intentional tremor in the hands is revealed even better in a different way; the patient touches the hammer or the examiner's finger with open eyes with the index finger, and the position of the hammer changes several times. The calcaneal-knee test is performed in a lying patient, who is invited to first raise his leg high, then touch the heel of the other knee and wind the heel down along the front surface of the lower leg. It should be indicated that the subject should only touch the surface of the shin with the heel, and not lean on it.
3. **Nystagmus** (twitching of the eyeballs when abducting them), observed with damage to the cerebellum, is more often horizontal than vertical or rotatory; an

indication that it is more pronounced when looking at the diseased side is unreliable.
There are doubts whether nystagmus can in general be a symptom of a lesion of the cerebellum itself and whether it is exclusively a symptom of a stem lesion. However, it is permissible that "cerebellar nystagmus" is a particular manifestation of intentional tremor of the eye muscles.
4. **Adiadochokinesis is** detected when trying to quickly perform alternately opposite movements. So, patients cannot quickly change pronation to supination of the hand: awkward, incorrect movements are obtained.
5. **Dysmetria,** or rather hypermetry of movements, can be easily
discovered by the following method: the subject is asked to keep
hands extended forward, palms up, with fingers apart;
an order follows to quickly turn the hands, palms down; on the side,
where there are cerebellar disorders, this movement is performed
with excessive rotation of the hand.
When asked to touch the heel of one leg to the knee of the other (in a supine position), the patient lifts the leg above the knee and touches the thigh with the heel (the phenomenon of hyperflexia).
6. **Past - hit** , or miss, with the so-called test, the readings are detected as follows: the patient is asked to hit the examiner's finger 2-3 times with the index finger or into the hammer, after which the patient closes his eyes and continues the same task. In the hand, in which there are cerebellar disorders, there is a miss past the gap, more often outward.
7. **Speech disorder** is a frequent manifestation of cerebellar movement disorder; speech loses its fluidity, becomes chanted, explosive, slowed down.
eight. **Muscle hypotonia** , resulting in lethargy, muscle flabbiness,
in excessive excursion in the joints, found during examination
passive movements. It can be accompanied by a decrease in the tendon reflexes of the extremities.
Other symptoms may occur with lesions of the cerebellum.
Asynergy is expressed in a violation of the coordination of the work of a number of muscle groups necessary for the implementation of a particular movement. One of the manifestations of asynergy is the so-called combined flexion of the hip and trunk. The subject lies on his back, preferably on a hard couch without a pillow at the head; when trying to sit with arms crossed on the chest, flexion of one or both (in case of bilateral lesion) legs, combined with flexion of the trunk, occurs.
The symptom of the absence of a "reverse impulse" , also explained by hypotension and impaired antagonistic innervation; the patient holds his hand in front of him, bending it with force at the elbow joint, in which there is resistance, with a sudden cessation of resistance, the patient's hand hits the chest with force. In a healthy person, this does not happen, since the inclusion of antagonists (extensors of the forearm) into action - the "reverse push" - prevents the impact.
Disorder of handwriting is the result of a violation of the coordination of fine movements and trembling; handwriting becomes uneven, lines become zigzag, letters are too large (megalography).

Underestimation of the severity of an object held by a hand is a kind of symptom observed on the affected side.

Pendulum - shaped , or **"pendular"** reflexes are apparently due to the same hypotension. When examining the knee reflex in a sitting position, with the legs hanging freely from the couch, after hitting the lig with a hammer. In the patellae, there are several swinging movements of the lower leg.

Changes in postural reflexes are also one of the symptoms of cerebellar damage. Here you can cite Doinikov's digital phenomenon, if a seated patient is offered to hold the hands with sharply spread fingers on the knees in the supination position, then on the side of the cerebellar lesion (note that the same is observed with paresis), flexion of the fingers and pronation of the hand quickly occur ...

Dizziness is a fairly common symptom of acute cerebellar lesions.

When the connections between the nucleus dentatus and the nucleus guber are damaged, extrapyramidal hyperkinesis can occur; when the lower olive or its connections with the nucleus dentatus are damaged, myoclonus of the tongue, pharynx, and soft palate is sometimes observed.

When the worm is damaged, static and gait disorders prevail; with damage to the cerebellar hemispheres, the smoothness and accuracy of the movements of the homolateral limbs (intentional tremor) are especially affected.

SUBCORTAL DISEASES AND EXTRAPYRAMIDAL DISORDERS

The purpose of the lesson is to study the structural and functional features, lesion syndromes and research methods of the striopallidal system.

The student should know:

anatomy and physiology of subcortical formations;

participation of the extrapyramidal system in the implementation of all subcortical unconditioned reflexes;

the relationship between the pyramidal and extrapyramidal systems;

regulation of muscle tone;

the main pathological syndromes of lesions of the extrapyramidal system (hypotonic - hyperkinetic and hypokinetic - hypertonic);

clinical variants of pakinsonian syndrome and hyperkinesis.

The student should be able to:

analyze the patient's complaints with an emphasis on movement disorders, examine the appearance, gait, behavior, speech, handwriting, motor activity, speed of movement;

diagnose the type of hyperkinesis, decerebration, variants of the hormonal syndrome.

Control questions.

1. What structures of the brain are united by the extrapyramidal system?
2. What are the anatomical - physiological features of the cerebellum?
3. What are the pathways of the extrapyramidal system and the cerebellum?
4. How are the neural connections between the cerebellum and the striopallidal system ensured?

5. How is the neural connection between the cerebral cortex and the cerebellum ensured?
6. What symptoms are observed with damage to the worm and cerebellar hemisphere?
7. What symptoms can be observed with damage to the striatal system?
8. What are the methods for studying the function of the cerebellum and the strio-pallido-nitral system?
9. What symptoms are observed when the pallidoneigral system is affected?
10. What are the symptoms of parkinsonism?
11. What is the difference between cerebellar ataxia and sensitive, vestibular and frontal ataxia?

SUBCORTAL DISEASES AND EXTRAPYRAMIDAL DISORDERS

The subcortical regions of the brain include the optic hillocks, basal ganglia, the subcortical region, and the inner capsule.

VISUAL BUGS

The continuation of the brain stem anteriorly is the visual hillocks located on the sides of the third ventricle.

The visual hillock is a powerful accumulation of gray matter in which a number of nuclear formations can be distinguished.

There is a division of the visual hillock into actually thalamus, hypothalamus, metathalamus and epithalamus

Thalamus - the bulk of the optic tubercle - consists of the outer, inner, ventral and posterior nuclei.

Nurothalamus has a number of nuclei located in the walls of the third ventricle and its infundibulum. The latter is very closely related to the pituitary gland, both anatomically and functionally. This also includes the papillary bodies (sogrora mamillaria). Metathalamus includes the external and internal geniculate bodies (sogrog geniculata laterale et mediale).

Epithalamus includes the pineal gland, or pineal gland (glandula pinealis seu corpus puieale - PNA), and the posterior commissure (coimssura posterior).

The optic hillock is an important stage in the path of sensitivity. The following sensitive conductors (from the opposite side) are suitable for it.

1. The medial loop with its bulbothalamic fibers (touch, musculoskeletal sensation, vibration sensation, etc.) and the spinothalamic pathway (pain and temperature sensation).

2. Lemniscus trigeminalis - from the sensitive nucleus of the trigeminal nerve (sensitivity of the face) and fibers from the nuclei of the glossopharyngeal and vagus nerves (sensitivity of the pharynx, larynx, etc., as well as internal organs).

3. Optic tracts ending in the pulvinar of the optic tubercle
and in the congus geniculatum laterale (visual pathways).

4. Lateral loop, ending in the con us geniculatum mediale (auditory tract).

The olfactory pathways and fibers from the cerebellum (from the red nuclei) also end in the visual hillock.

Thus, impulses of exteroceptive sensitivity flow to the visual hillock, which perceives stimuli from the outside (pain, temperature, touch, light, etc.), proprioceptive (joint-muscular feeling, sense of position and movement), interoceptive (from internal organs).

Such a concentration of all types of sensitivity in the visual hillock will become understandable if we take into account that at certain stages of the evolution of the nervous system, the visual hillock was the main and final sensitive center that determines the general motor reactions of the body of a reflex order by transmitting irritation to the centrifugal motor apparatus.

With the appearance and development of the cerebral cortex, the sensory function becomes more complicated and improved; the ability to subtle analysis, differentiation and localization of stimuli appears.

However, the course of the sensitive pathways remains the same; there is only their continuation from the visual hillock to the cortex. The optic hillock basically becomes only a transmitting station on the path of impulses from the periphery to the cortex. Indeed, there are numerous thalamo-cortical pathways (tractus thalamocjrticales), those (mainly third) neurons of sensitivity, which have already been discussed in the chapter on sensitivity and which should only be briefly mentioned:

1) third neurons of skin and deep sensitivity (pain, temperature, tactile, joint-muscular feelings, etc.),

starting from the ventrolateral part of the optic tubercle, passing through the internal capsule, into the region of the posterior central gyrus and the parietal lobe;

2) visual pathways from the primary visual centers (congus geniculatum laterale - radiatio opticia) or Graciole's bundle to the sulci calcarini region of the occipital lobe.

3) auditory pathways from the primary auditory centers to the superior temporal gyrus and Heschl gyrus.

In addition to the connections already mentioned, the visual hillock has pathways connecting it with the strio-pallidary system. Just as the thalamus opticus is the highest sensory center at certain stages of the development of the nervous system, the strio-pallidary system was the final motor apparatus, carrying out a rather complex reflex activity. Therefore, the connections of the visual hillock with the named system are very intimate, and the entire apparatus as a whole can be called the thalamo-strio-pallidary system with a perceiving link in the form of thalamus opticus and a motor in the form of a strio-pachlidar apparatus.

It has already been said about the connections of the optic hillock with the cerebral cortex in the direction of the thalamus - cortex. In addition, there is a powerful system of conductors in the opposite direction, from the cerebral cortex to the visual hillocks. These pathways originate from various parts of the cortex, the most massive of them is the one that starts from the frontal lobe.

Finally, mention should be made of the connections of the optic hillock with the sub-hillock area, where the subcortical centers of autonomic-visceral innervation are concentrated.

The connections of nuclear formations in the thalamic region are very numerous, complex, and have not yet been sufficiently studied in detail. Recently, mainly on the basis of anatomophysiological studies, it has been proposed to divide the thalamo-cortical systems into specific (associated with certain projection zones of the cortex) and nonspecific, or diffuse. The latter begin from the medial group of nuclei of the optic tubercle (median center, intralaminar, reticular and other nuclei).

Some researchers (Penfield, Jasper) attribute to these “nonspecific nuclei” of the visual hillock, as well as the reticular formation of the trunk, the function of “substrate of consciousness” and “higher level of integration” of nervous activity. In the concept of the "central brain system," the cortex is viewed only as an intermediate stage on the path of sensory impulses flowing from the periphery to the "highest level of integration" in the interstitial and midbrain. The supporters of this hypothesis, therefore, come into conflict with the history of the developed nervous system, with numerous and obvious facts establishing that the subtlest analysis and most complex synthesis ("integration") of nervous activity is carried out by the cerebral cortex, which, of course, does not function in isolation. and in an inextricable connection with the underlying subcortical, stem and segmental formations.

Based on the above anatomical data, as well as existing clinical observations, the functional significance of the optic hillock can be determined mainly by the following provisions. The optic hillock is:

1) a transmission station for conducting into the cortex of all types of "general" sensitivity, visual, auditory and other stimuli;

2) the afferent link of the complex subcortical thalamo-strio-pallidary system, which carries out rather complex automated reflex acts;

3) through the visual hillock, which is also a subcortical center for visceroreception, automatic regulation of the internal processes of the body and the activity of internal organs is carried out due to connections with the hypothalamic region and the cerebral cortex.

Sensitive impulses received by the visual hillock can acquire one or another emotional color here. According to MI Astvatsaturov, the visual hillock is an organ of primitive affects and emotions closely related to the feeling of pain; at the same time, there are reactions from visceral devices (redness, paleness, changes in pulse and respiration, etc.) and affective, expressive motor reactions of laughter and crying.

Symptoms of the defeat of the optic tubercle

If the optic tubercle is affected, symptoms of loss of its functions or symptoms of irritation may occur.

In the first case, hemianesthesia is observed (on the opposite side), which concerns all types of sensitivity, both superficial and deep. Sensory disorders are more pronounced in the distal extremities; loss of joint-muscular sensation is

usually noted especially sharply. Therefore, in the anesthetized limbs, there is also a sensitive hemiataxia. Due to the defeat of the subcortical visual centers, hemianopsia also occurs - loss of vision in the fields of vision opposite to the lesion.

Finally, with a lesion of the visual hillock, paresis of the facial muscles can be observed, also on the opposite side, which affects only during emotive facial movements, for example, when smiling or laughing. When movements "on assignment" disturbances of innervation may not be noted.

With irritation of the visual hillock, severe, sometimes unbearable pains occur in the opposite side of the body. The nature of these "central" pains is hardly described by patients; more often it is a painful burning sensation, cold, unbearable pain. They are not clearly localized and are usually diffuse. Their intensity increases depending on external stimuli and especially emotions. Increased efficiency, violent laughing and crying are common. The addition of a number of autonomic disorders is possible. All these symptoms are easily explained by the role and significance of the visual hillock, as mentioned above.

With irritation of the visual hillock (possibly with partial damage to some of its nuclei), not only the described peculiar thalamic pains occur, but also sensory disorders on the opposite side of the body, which are in the nature of hyperpathy (a sharp feeling of unpleasantness, with inaccurate localization with an injection and temperature irritations, sometimes perverted perception of irritation, inaccuracy of its localization, irradiation, prolonged sensation of irritation, or the so-called aftereffect, etc.).

Impulses emanating from the irritated visual hillock in the direction of the strio-pallidary system, which is closely related to it, can sometimes cause involuntary violent movements, or hyperkinesis, such as chorea or athetosis, which are described below.

Finally, in some cases, cerebellar disorders can also join, since fibers from the cerebellum and red nuclei end in the optic tubercle.

SUBCORTAL NODES

The following anatomical structures belong to the basal ganglia: the caudatus nucleus and the lentiformis nucleus with its outer nucleus and two inner ones. They are located in front and outside of the optic hillocks. According to morphological features, phylogenetic age and functional significance, these formations can be more correctly divided into the striatum or neostriatum system, which includes the caudatus nucleus and the outer nucleus of the lentiformis-putamen, pallidum or paleostriatum nucleus, including the pallidus globe (inner nuclei of the nucleus lentiformis). The pallidary system includes the Lewis body, substantia nigra and red nuclei located in the legs of the brain.

Strio-pallidum is an important component of non-pyramidal (extrapyramidal) motor systems, starting from the cerebral cortex (mainly from field 6 in the premotor zone) and associated with a number of subcortical and brainstem formations.

The main pathways along which impulses are conducted to the striatum and pallidum are conductors from the optic tubercle. Through them, connections of the

extrapyramidal system are established not only with the thalamus opticus, but through the same visual hillock and with the cerebral cortex. In this way , the extrapyramidal apparatuses are included in the system of "voluntary", cortical movements. There are also independent connections of the strio-pallidary system with the cerebral cortex; known, for example, cortico-pallidary, corticonigral and other extrapyramidal motor conductors.

The striatum has a close relationship with the pallidum. The centrifugal paths are directed towards the reticular formation, the red nucleus, the Darkshevich nucleus, the quadruple, the olives. From the named formations, impulses from the extrapyramidal system follow to the segmental motor apparatus and muscles along the descending conductors

1) from red nuclei along the Monaco beam;

2) from the Darkshevich nucleus along the posterior longitudinal bundle to the nuclei of the III, IV, VI nerves

| and through it to the nucleus of the vestibular nerve;

3) from the nucleus of the vestibular nerve along the tractus of the vestibulospinalis;

4) from the quadruple to the tectospinalis tractus;

5) from the reticular formation along the tractus reticulospiralis (this path considered the main efferent extrapyramidal conductor).

Impulses from the strio-pallidal system, as well as from the cerebellum and the pyramidal system, flow, therefore, to the cells of the anterior horn, where all the conductors just listed end. The final route to the muscle is through a peripheral motor neuron.

Due to the presence of this system (receptors on the periphery of the thalamus-strio-pallidum - centrifugal extrapyramidal pathways the cell of the anterior horn - muscle), reflex activity is carried out concerning automated, sometimes quite skin movements. Thanks to the inclusion in the motor system of the cortex, the auxiliary participation of extrapyramidal apparatus of "voluntary" movements is provided.

In addition to the disassembled connections, we can once again mention the pathways to the hypothalamic region (subcortical centers of visceral innervation).

During the period when the cerebral cortex was not yet developed, the strio-pallidary system was the main motor center that determines the position of the animal. Sensory impulses flowing from the visual hillock were processed here into motor impulses, directed to the segmental apparatus and muscles. Due to strio-pallidary apparatuses, diffuse movements of a rather complex nature were carried out: movement, swimming, etc.

At the same time, support was provided for the general muscle tone, the "readiness" of the segmental apparatus for action, and the redistribution of muscle tone during movements.

With the further evolution of the nervous system, the leading role in movements passes to the cerebral cortex with its motor analyzer and pyramidal system. Finally, a person experiences the most complex actions that are purposeful, productive in nature with a subtle differentiation of individual

movements. Nevertheless, the strio-pallidal (extrapyramidal) system has not lost its importance in humans. It only passes into a subordinate, subordinate position, providing the "tuning" of the motor apparatus, their "readiness for action" and the muscle tone necessary for the rapid implementation of the movement.

The extrapyramidal system in humans automatically creates that background of "readiness", on which fast, precise, differentiated movements are carried out due to the activity of the cortex.

As noted above, the extrapyramidal system is divided into its more ancient division (palliostriatum) and a new, later one (neostriatum). The relationships between them are the same as those that exist in general between phylogenetically older and newer, more perfect apparatuses: the activity of the pallidary system is inhibited, regulated (subordinated) by the striatal system.

The anatomical connections and functional relationships of the brain regions involved in extrapyramidal innervation are very complex. Neural circles, along which impulses continuously circulate, unite individual nuclear formations into functional systems, for example: the optic tubercle - striatum - pallidum - visual tubercle, or cortex - substantia nigra - pallidum - Lewis body - red nucleus - visual tubercle - cortex. The presence of systems that functionally unite many parts of the brain explains the fact that similar symptoms can be observed at different localizations of the lesion of the extrapyramidal system.

Extrapyramidal Disorders

Extrapyramidal disorders are manifested mainly by changes in muscle tone (rigidity or hypotension) and changes in motor activity (hypokinesis or hyperkinesis). These disorders can be combined. So, hypokinesia is often combined with muscle rigidity, and some hyperkinesis (for example, choreic) - with muscle hypotonia.

Hypokinesis is observed with lesions of the frontal lobes, black

substance, reticular formation (but not the globus pallidus, as previously believed). Hyperkinesis can occur at various localizations of the process in the nervous system, in particular in the striatal nuclei, optic tubercle, red nuclei, cerebellar-thalamic pathways.

From extrapyramidal disorders, we will consider parkinsonism and the most common hyperkinesis.

Parkinsonism

This symptom complex occurs when the substantia nigra, located in the cerebral peduncles, is functionally associated with the reticular formation of the brain stem, subcortical ganglia and cerebral cortex. Parkinsonism is characterized by the following symptoms: muscle rigidity, hypokinesia and tremor at rest. Extrapyramidal hypertension, or muscle rigidity, differs significantly from that of pyramidal lesions. With extrapyramidal rigidity, the resistance experienced by the examiner during passive movements remains the same all the time from the beginning to the end of the movement, while with central paralysis or paresis, spasticity is especially great at the beginning of the movement and is noticeably weakened at the end ("folding knife" symptom). Extrapyramidal rigidity is called "waxy". With passive extension of the forearm, head, circular movements in the

wrist joint, you can sometimes feel a kind of discontinuity, graded stretching of the muscles, which is called the "cogwheel" symptom.

Hypokinesia, or oligokinesia, is by no means due to the presence of paralysis; the study reveals that voluntary movements are performed in a sufficient volume of unsatisfactory muscle strength. The main ones are the patient's inactivity, a sharp decrease in motor initiative, difficulty in the transition from rest to movement. The patient, having taken a certain position, retains it for a long time, even if it was uncomfortable, “freezes” in the accepted position, resembling a statue or a mannequin.

The patient's usual posture is quite characteristic: the back is bent, the head is tilted to the chest, the arms are bent at the elbows, the hands are at the wrist, the legs are at the knee joints (flexor posture).

The gait is slow, reminiscent of senile, small steps. It is not possible to move forward immediately, but in the future the patient can "disperse" and move faster. But he cannot stop quickly: if necessary or when ordered to stop, he still continues to "pull" forward.

The facial expressions are extremely poor, the face takes on a frozen, mask-like expression (hypomimia). A smile, a grimace of crying with emotions arise with a delay and just as slowly disappear.

The patients' speech is quiet, monotonous, deaf, without sufficient modulation and sonority.

Characteristic is the absence or decrease in physiological friendly or concomitant movements, synkinesis. existing in the norm and contributing to this or that main movement. So, patients do not have the usual waving of their hands in time with walking, there is no wrinkling of the forehead when looking up, there is no extension in the wrist joint when clenching into a fist, etc. Not only is the transition from rest to movement difficult, but all voluntary movements are usually sharply slowed down (bradykinesia). All actions of the patient resemble automatic.

In addition to the symptoms described, with parkinsonism, there is often a kind of tremor, which is observed at rest, is expressed in the distal parts of the limbs, sometimes in the lower jaw and is usually distinguished by a small amplitude, frequency and rhythm. In contrast to the cerebellar intentional tremor, which appears in motion and is absent at rest, it is typically present at rest and decreases or disappears during movements.

The described syndrome is also called the amiostatic symptom complex.

In the absence of changes in the tendon reflexes of the extremities for another reason, with parkinsonism, the so-called axial reflexes can come to life and, first of all, the group of oral automatism reflexes. Reflexes of position or posture (postural reflexes) are significantly impaired.

Extrapyramidal hyperkinesis

Excessive, involuntary, violent movements are called hyperkinesis. The main forms of extrapyramidal hyperkinesis are as follows.

Athetosis, which occurs more in the distal extremities, such as the hands and fingers. There are slow, writhing, worm-like movements at intervals, during which the limb assumes unnatural positions. Athetosis can be limited, it can be

widespread, sometimes involving the entire musculature of the body. There is reason to believe that athetosis occurs when the nucleus caudati is affected.

Torsion spasm (or torsion dystonia) is trunk athetosis. It is characterized by bending, sometimes corkscrew-like movements of the trunk that occur when walking, which is often significantly difficult.

Chorea differs from athetosis in the speed of twitching, the latter are observed in various muscle groups, often in the proximal extremities, in the face. A rapid change in the localization of the spasm is characteristic: either the mimic muscles twitch, then the muscles of the leg, at the same time the eye muscles and the arm, etc. In severe cases, the patient becomes like a clown. Grimacing, smacking is often observed; speech is upset. Movements become sweeping, redundant, gait - "dancing". It is possible that chorea occurs when the outer nucleus of the nucleus is affected with the simultaneous involvement of the dentorubral system in the process.

Myoclonus is similar in character to chorea. Here twitching is also very fast, observed in individual muscle groups or single muscles. Unlike chorea, myoclonus is often not accompanied by significant motor affect. Localized spasm and some forms of tics can also be a phenomenon of extrapyramidal hyperkinesis. Localized spasm, as the name suggests, is observed in isolation in one muscle group, most often in the muscles of the face or neck. This is especially often observed in the muscles innervated by the facial or accessory nerve (n. Accessorius). These rather rare forms should not be confused with the frequently observed neurotic tics, which are habitual obsessive movements caused by organic damage to the nervous system. The described hyperkinesis occurs with different localizations of lesions of the extrapyramidal systems. Recently, an opinion has been expressed about the possibility of classifying them according to the levels of brain damage. L.S. Petelin secretes hyperkinesis predominantly of the stem level (various types of organic tremor, myoclonus, torticollis, etc.), predominantly of the subcortical level (chorea, athetosis, torsion dystonia, ballism) and subcortical-cortical hyperkinesis (hyperkinesis of epilepsy).

Common features characteristic of all types of extrapyramidal hyperkinesis are that they usually disappear during sleep (as opposed to cortical seizures) and intensify with excitement and voluntary movements .

Lesions of the large hemispheres and disorders of the higher cortical functions

The purpose of the lesson is to study the structural and functional foundations of higher mental functions; master the methods of neuropsychological examination of neurological patients.

The student should know:

structural - functional foundations of higher mental functions for the formation of intellectual activity of the brain;

aphasia, apraxia, agnosia as the main forms of pathology of higher mental functions;

memory disorders with focal brain lesions.

The student should be able to:

conduct a neuropsychological examination of the patient;

identify syndromes of lesion of primary, secondary, tertiary fields of special analyzers;

evaluate thinking, attention, short - and long term memory.

Control questions:

1. What are the current ideas about the localization of higher cortical functions
2. Give a definition of the various forms of aphasia.
3. With the defeat of which areas of the cerebral cortex occurs motor, sensory, amnestic aphasia?
4. What are the symptoms of motor aphasia?
5. What are the methods for studying expressive speech?
6. What are the symptoms of sensory aphasia?
7. What are the methods of researching impressive speech?
8. What is amnestic aphasia?
9. What are paraphasia and perseveration?
10. What is alexia, and in what area of the brain is it affected?

11. What are the research methods of reading?
12. What is agraphia, and in which area of the brain is affected?
13. What are the methods of researching writing?
14. What is a shark, and in which area of the brain is it affected?
15. What are the methods of researching accounts?
16. What is apraxia, and when which area of the brain is affected, does it occur?
17. What are the methods of praxis research?
18. Give a definition of the term agnosia, and what types of agnosia do you know?
19. What is the research methodology for gnosis?
20. Give a definition of a violation of the body scheme, and in which area of the brain it is affected?
21. What is the difference between aphasia and dysarthria?
22. What are the functional differences between the left and right hemispheres?

In the large human brain, we distinguish between the subcortical ganglia of the base, the white matter of the hemispheres, and, finally, the cerebral cortex, which is the latest in development and the most perfect part of the central nervous system, which have already been considered. Anatomically, the cortex is a plate of gray matter that covers the outer surface of the hemispheres.

Distinguish between the outer (convex) surface of the hemispheres, their inner surface and the base. On the outer surface, a powerful Roland groove separates the frontal and parietal lobes. Below its located Sylvian groove separates the temporal lobe from the parietal and frontal. The occipital lobe is separated from the parietal and temporal line extending downward from the zulkus parietooccipitalis. Thus, on the convex surface of each hemisphere, four lobes of the cerebral cortex are outlined: frontal, parietal, temporal and occipital (according to another division, there is also a limbic and an insula). Two massive gyrus located "along the banks" of the Roland groove, one anterior to the latter (anterior central gyrus) and the other posterior to it (central posterior gyrus), often stand out in a special lobe called the region of the central gyrus.

On the outer, convex surface, there are: in the frontal lobe proper (in the area anterior to the anterior central gyrus), three gyrus located approximately horizontally: the first, or superior, second, or middle, and the third, or inferior, frontal gyrus.

The parietal lobe is divided into the superior and inferior parietal lobes by a horizontal groove running in the middle. In the lower parietal lobe, the gyrus supramarginalis is located more anteriorly and the gyrus angularis bordering the occipital lobe posteriorly.

In the temporal lobe, three horizontally located gyri are noticeable: the first, or superior, second, or middle, and the third, or inferior temporal gyri.

On the inner surface of the hemispheres, after the section of the brain along the sagittal line, the Zulus parietooccipitalis is well expressed, separating the occipital lobe from the parietal. In the occipital lobe, a deep fissure of calcarin is outlined, above which is located cuneus and below the gyrus lingualis seu occipitotempopalis medialis. The anterior temporal lobe contains the gyrus heppocamp. In the middle of the section, the intersected fibers of the main commissural adhesion of the hemispheres - corporis callosi (corpus callosum) are visible.

On the lower surface of the cerebral hemispheres (on the base), in the anterior section, there are the frontal lobes, posterior to them - the temporal lobes, separated by the sylvian groove, and even more posteriorly - the occipital lobes. At the base of the frontal lobes, bulbus and tractus olfactorii are visible, posterior to them is the intersection of the optic nerves. A rather massive formation is the brain stem: the legs of the brain, the pons varoli, the medulla oblongata and the cerebellum lying above the latter and under the occipital lobes.

The bark is a gray matter. The microscopic structure is rather complex; the cortex is made up of a series of layers of cells and their fibers. The main type of structure of the cerebral cortex is six-layered.

I. The molecular layers, the most superficial, lies directly under the pia mater, is bleached with cells, its fibers have a direction parallel to the surface of the cortex, which is why it is also called tangential.

P. The outer granular layer is located deeper than the first, includes a large number of small granular nerve cells.

III. A layer of small and medium pyramidal cells.

IV.Inner granular layer.

V Layer of large pyramidal cells.

Vi. The layer of polymorphic cells consists of cells of the most diverse shapes (triangular, fusiform, etc.).

The fibers of these cells have either a direction parallel to the surface of the cortex (association paths connecting different areas of the cortex), or are radial, perpendicular to the surface. The latter type of fibers are typical for projection pathways (connecting the cerebral cortex with its underlying formations).

The six-layer type of structure of the bark is far from homogeneous. There are sections of the cortex in which one of the layers appears to be especially powerful, while the other is very weakly expressed. In other areas of the cortex, some layers are subdivided into sublayers.

The founder of a detailed study of the structure of the cellular composition of the cortex was the Russian scientist V.A. Bets. Many years before Brodmann. Focht, Economo and others with the help of the new method of serial brain sections and carmine staining by V.A. Betsem, which he himself developed, the cytoarchigectonics of the cerebral cortex was carefully developed and a big step was taken in the study of the localization of the function in it. The very term "architectonics" of the cortex also belongs to V.A. Bets.

Numerous subsequent works, especially by domestic scientists (I.N. Filimonov, S.A. Sarkisov, E.T. Kononova, etc.), expanded our understanding of the fine

histological structure of the cerebral cortex. The results of cytoarchitectonic studies have played a known role in resolving controversial issues about the localization of functions in the cerebral cortex. It has been established that the areas associated with a certain function have their own, inherent structure; that sections of the cortex, which are close in their functional significance, have a certain similarity in structure, both in animals and in humans. The same areas, the lesions of which cause a disorder of complex, purely human functions (for example, speech), are found only in the human cortex, and in mammals, including even in anthropomorphic monkeys, they exist.

LOCALIZATION OF FUNCTIONS IN THE CORRUS

The idea of the localization of functions in the cerebral cortex is of great practical importance for solving the problems of tonics of lesions in the cerebral hemispheres. However, to this day, much in this section remains controversial and not fully resolved. The doctrine of the localization of functions in the cortex has a rather long history - from the denial of the localization of functions in it to the distribution in the cortex in strictly limited areas of all functions of human activity, in the flesh to the highest qualities of the latter (memory, will, etc.) and, on the end, before returning to the `` equipotentiality " of the cortex, i.e. again, essentially to the denial of the localization of the function (recently abroad).

The concept of the equivalence (equipotentiality) of various cortical fields contradicts the huge factual material accumulated by morphologists, physiologists, and clinicians. Everyday clinical experience shows that there are certain unshakable regular dependences of function disorders on the location of the pathological focus. Based on these basic provisions, the clinician solves the problems of topical diagnostics. However, this is the case as long as we operate with disorders related to relatively simple functions: movements, sensitivity, etc. periphery. The functions of the cortex are more complex, phylogenetically younger, and cannot be narrowly localized; very extensive areas of the cortex, and even the entire cortex as a whole, are involved in the implementation of complex functions. That is why , as clinical experience shows, the solution of problems of the topic of Lesions on the basis of speech disorders, apraxia, agnosia, and even more mental disorders, is more difficult and sometimes inaccurate.

At the same time, within the limits of the cerebral cortex there are areas, the lesion of which causes one or another character, one degree or another, for example, speech disorders, disorders of gnosia and praxia, the topodiagnostic value of which is also significant. From this, however, it does not follow that there are special, narrowly localized centers that "control" these most complex forms of human activity. It is necessary to clearly distinguish between the localization of functions and the localization of symptoms.

The foundations of a new and progressive doctrine of the localization of functions in the brain were created by I.P. Pavlov.

Instead of the idea of the cerebral cortex as in the well-known "Measure, an isolated superstructure over other levels of the nervous system, narrowly localized, connected along the surface (associative) and with the periphery (projection) areas, I.P. Pavlov created the doctrine of the functional unity of neurons belonging to

different departments of the nervous system, from receptors in the periphery to the cerebral cortex - the doctrine of analyzers.What we call mtrom, is the highest, cortical section of the analyzer.Each analyzer is associated with specific areas of the cerebral cortex.

I.P. Pavlov makes significant adjustments to the previous ideas about the limited territories of cortical centers, to the doctrine of the narrow localization of functions. The cortical representation of the analyzers is not limited to the projection area of the corresponding conductors, but extends to wider areas; the cortical fields of different analyzers overlap each other.

This conclusion, based on extensive experimental and physiological studies, is in full agreement with the latest morphological data on the impossibility of precise delimitation of cortical cytoarchitectonic fields.

Consequently, the functions of the analyzers (or, in other words, the operation of the first signaling system) cannot be associated only with the cortical projection zones (the nuclei of the analyzers). Moreover, it is impossible to narrowly localize the most complex, purely human functions - the functions of the second signaling system.

The operation of the second signaling system is inextricably linked with the functions of all analyzers; therefore, it is impossible to imagine the localization of the complex functions of the second signaling system in any limited cortical fields.

The significance of the inheritance left to us by the great physiologist for the correct development of the theory of the localization of functions in the cerebral cortex is extremely great. I.P. Pavlov laid the foundations of the doctrine of the dynamic localization of functions in the cortex. The concept of dynamic localization suggests the possibility of using the same cortical structures in various combinations to serve various complex cortical functions.

Keeping a number of definitions and interpretations established in the clinic, we will try to make our presentation some corrections in the light of the teachings of I.P. Pavlov about the nervous system and its pathology.

First of all, you need to consider the issue of the so-called projection and association centers. The usual idea of motor, sensory and other projection centers (anterior and posterior central gyri, visual, auditory centers, etc.) is associated with the concept of a rather limited localization in a given area of the cortex of a particular function, and this center is directly related to the underlying nervous devices , and subsequently with the periphery, with its own conductors (hence the definition - "projection"). An example of such a center and its vehicle is, for example, the anterior central gyrus and the pyramidal path; etc. The projection centers are associated with other centers, with the surface of the cortex by association paths. These broad and powerful associative pathways determine the possibility of the combined activity of various cortical areas, the establishment of new connections, the formation, therefore, of conditioned reflexes.

"Association centers", in contrast to projection centers, do not have a direct connection with the underlying parts of the nervous system by the periphery; they are connected only with other parts of the cortex, including the "projection centers". An example of an "association center" is the so-called "stereognosy

center" in the parietal lobe, located posterior to the posterior central gyrus. The division of centers into projection and associative centers seems to us incorrect. The large hemispheres are a collection of analyzers. The upper layers of the cerebral cortex, in fact, are entirely occupied by the perceiving centers or, in the terminology of I.P. Pavlov, "the brain ends of the analyzers" 1. At the same time, from all the lobes, from the lower layers of the cortex, there are already efferent conductors connecting the cortical ends of the analyzers with the executive organs through the subcortical, stem and spinal apparatuses. An example of such an afferent conductor is the pyramidal pathway - this intercalary neuron between the kinesthetic (motor) analyzer and the peripheral motor neuron. The anterior central gyrus is only a motor projection area for the pyramidal pathways, and not a "projection motor center".

There is no doubt that "voluntary" movements are conditioned motor reflexes, i.e. movements, established, "beaten", in the process of individual life experience; but in the development, organization and already established activity of skeletal muscles, everything depends on the afferent device - the skin and motor analyzer (clinically - skin and joint-muscular sensitivity, more broadly - kinesthetic feeling), without which fine and precise coordination of the motor act is impossible.

The motor analyzer (whose task is to analyze and synthesize "voluntary" movements) does not at all correspond to the ideas about cortical motor "projection" centers with certain boundaries of the latter and a clear somatotopic distribution. The motor analyzer, like all analyzers, is associated with very wide areas of the cortex, and the motor function (in relation to "voluntary" movements) is extremely complex. This complex function is by no means provided only by the projection zone for pyramidal paths.

The same is the case with other "projection" centers (skin sensitivity, vision, hearing, taste, smell). Consequently, in each analyzer (its cortical section), including the motor, there is an area or zone "projecting" on the periphery (motor area) or into which the periphery is "projected" (sensitive areas, including kinesthetic receptors for the motor analyzer).

It is possible that the “projection core of the analyzer” can be identified with the concept of a motor or sensitive projection zone. The maximum of violations, wrote I.P. Pavlov, of analysis in synthesis occurs when just such a "projection nucleus" is damaged: if we take the maximum of functional impairment as the real maximum "breakdown" of the analyzer, which is objectively absolutely correct, then the greatest manifestation of damage to the motor analyzer is neutral paralysis, and the sensitive - anesthesia. From this point of view, it would be correct to identify the concept of "analyzer core" with the concept of "projection area of the analyzer".

Based on the above, we consider it correct to replace the concept of the projection center with the concept of the projection area in the analyzer zone. Then the division of cortical "centers" into projection and associative ones is unreasonable: there are analyzers (cortical and their departments) and within them there are projection areas.

Cortex projection areas

The motor projection areas for the muscles of the opposite side of the body are located in the anterior central gyrus.
The projection for individual muscle groups is presented here in the reverse order of their location in the body: the upper sections of the anterior central gyrus correspond to the leg, the middle sections correspond to the hand, and the lower sections of the anterior central gyrus correspond to the face, tongue, larynx and pharynx.
The projection of trunk movements, apparently, is presented in the posterior part of the superior frontal gyrus.
The projection of turning the eyes and head in the opposite direction corresponds to the posterior part of the second frontal gyrus. The paths from here are directed downward in close proximity to the pyramidal bundles and establish connections with the posterior longitudinal bundle in the brain stem, carrying out voluntary innervation of the gaze (either alone or in combination with a turn of the head).
Sensitive projection areas are located in the posterior central gyrus. The projection of cutaneous receptors is similar to somatotopic representation in the anterior central gyrus: in the upper gyrus, the sensitivity of the lower extremity is presented, on the average - of the arms and in the lower - of the head.
The visual projection area is located in the occipital lobes, on the inner surface of the hemispheres, along the edges and in the depths of the sulci calcarini. Opposite fields of view of both eyes are represented in each hemispheres, and the area located above the sulco calcarino corresponds to the lower, and the area below it corresponds to the upper quadrants of the visual fields.
The auditory projection area of the cortex is located in the temporal lobes, in the first (superior) temporal gyrus and in the gyrus of Heschl (on the inner surface of the temporal lobe).
The olfactory projection area is also located in the temporal lobes, mainly in the gyrys parahippocampalis, especially in its anterior region (hook).
Also in the temporal lobe, gustatory areas are located close to the olfactory areas.
All projection areas of the cortex are bilateral, symmetrically located in each hemisphere. Some of them are associated only with the opposite side (the anterior and posterior central gyri, the zone of rotation of the eyes and head, the visual area). The cortical auditory, olfactory and gustatory territories of each hemisphere are associated with the corresponding receptor fields on the periphery on both sides (opposite and own).
The impulses of voluntary movements (the result of cortical, conditioned reflex activity) pass through the system of descending conductors to the skeletal muscles. From the periphery - a wide field of receptors for skin, muscle, special sensory organs, visceral and others - centripetal impulses through the system of afferent pathways enter the corresponding projection sensitive territories, to special cells of a certain layer of the cortex, and from here - into the wide territories of the analyzer, where it occurs analysis and synthesis of perception.
It would be correct to limit the concept of a projection area to only a group of special nerve cells located in a certain area, only in a certain cellular layer of the cortex. The projection area is determined only by the area of this cell group.

Otherwise, the entire crust of this territory is undoubtedly part of this analyzer. Thus, the anterior, say central, gyrus completely enters the wide fields of the motor analyzer, the posterior central gyrus - the cutaneous gyrus, etc.

Disorders of praxia and gnosia

On the basis of physiological and anatomical (cytoarchitectonic) studies and clinical experience, it is known that the territories of the cortex associated with the implementation of praxical and gnostic functions are not strictly delimited; this differs from projection territories.

Proceeding to the definition of praxia, let us recall that only primitive movements are innate in a person (in a child). A combination of a number of movements, which is already a certain complex and combination, which is strengthened in a skill through repetitions and exercises. creates great complexity of movements with the transition later into special, purposeful actions, or praxia. The latter are created by imitation, learning and constant practice throughout the individual life.

Praxia is inherent in humans, it is a product of long-term development, both biological and social.

Vast cortical areas related to praxia enter the motor analyzer system.

"Praxia cannot be considered separately from gnosia, since" gnosticism is not limited to a combination of the sensory organs ... E.K. Sepp).

Even a simple movement cannot be accomplished without constant reception of the movement itself. Continuous information from the proprioceptors is all the more necessary when a complex and finely differentiated movement is carried out. The highest analysis and synthesis of kinesthetic stimuli is carried out by the motor analyzer, the work of which provides an extraordinary variety, accuracy and coordination of complex motor acts developed in the practice of individual experience. There is an accumulation of "sensory images" of actions, inextricably and continuously associated with ideas about their own body and about the working (performing) organ (tongue, hand), and about the spatial relationships of the latter with the surrounding world.

Equally, gnosia cannot be understood in isolation from praxia. Indeed, in the process of cognizing the external world, the development of the human hand as an organ of labor and a means of communication was of great importance.

Praxia is the result of the combined activity of wide areas of the cerebral cortex, both receptor-gnostic and praxical. The final motor effect occurs through descending, efferent conductors. It is therefore clear that, as clinical experience shows, distinct and significantly pronounced praxical disorders occur rather rarely in focal lesions of the cerebral hemispheres; it is clear that apraxia can occur with localizations of the process, both in the frontal lobes and in the parietal (receptor-gnostic), finally, it is also clear that for the occurrence of apraxia, as a rule, it is necessary to damage large areas of the left hemisphere, and sometimes bilateral defeat. The most distinct forms of apraxia nevertheless arise when the left parietal lobe is affected in right-handers (inferior parietal polka). Peculiar apraxic disorders are possible with foci in the frontal lobes.

There are ileatory, motor and constructive forms of apraxia. We do not dwell on the description of these options, which are often difficult to differentiate. The

indication that more pronounced types of praxia are observed with foci in the left inferior parietal lobe of the marginal gyrus, (in right-handers), requires, as we believe, an amendment or, more correctly, clarification.

We are accustomed to referring the functions of higher analysis and synthesis, the second signaling system, higher nervous activity to the higher parts of the analyzers, which are the nerve cells of the cerebral cortex. This is undoubtedly so, but no less important for the implementation of these functions is also the preservation of the structure of wide connections between the cortical fields (their nerve cells), providing an associative process, "beaten paths", temporary connections.

The complex functions of the human brain, the second signaling system, are associated, as already mentioned, with very wide areas of the cortex, and it would be wrong to be satisfied, for example, with the old, narrowly localistic concepts of limiting such a complex function as praxia to localization only in the region

The anatomical substrate of these countless connections is the association and commissural fibers of the white matter of the hemispheres and, apparently, not only and not so much short (Y-shaped) - between adjacent gyri, as long association paths that connect whole lobes of the brain. These pathways pass under the cerebral cortex, in the white matter of the hemispheres, often at a considerable depth, and constitute, as it were, the highways necessary to ensure the joint, combined activity of vast territories occupied by the nerve cells of the cortex.

Therefore, the greatest damage to complex functions is caused by the separation of such connections, especially where they are concentrated and intersected. Such areas are, in particular, those that are located in the depth of the **junction of the** frontal, parietal and temporal lobes or the occipital, temporal and parietal. The most severe losses and disorders of the higher cortical functions - apraxia, agnosia, speech disorders - occur precisely with focal lesions of these parts of the brain.

The experience of comparing clinical disorders with the location of the pathological focus detected at the operation or on the section shows that these syndromes are usually caused by deep ones. subcortical location of the focus. The defeat of the superficial cortical layers, the outer gray plate with its nerve cells, at least, for example, within the entire surface of the hemispheres, does not give persistent and deep apraxic disorders. It can also be mentioned that diffuse meningoencephalitis, if they are not accompanied by focal encephalitic softening, creating a vivid picture of general cerebral disorders - changes in consciousness, agitation, delirium - rarely determine the development of disorders such as apraxia or aphasia.

From this point of view, one can explain the fact that the concept of localization of a function, which is extremely widely represented in the cortex of a normal brain, does not coincide with the concept of a symptom in case of focal lesion of the latter. Then the true topical meaning of such disorders as aphasia or apraxia is understandable, which, however, has invariably been confirmed and confirmed by clinical experience, although speech and praxia as a function cannot, of course, be narrowly localized.

Apraxia is the result of damage to the motor analyzer without the phenomena of paralysis or coordination of movements. As a result of a violation of synthesis and analysis, the skills of complex, purposeful, production actions are lost . Correct use of household items, professional work processes, semantic gestures, playing a musical instrument, etc. in a patient - are violated.

Moving on to the consideration of agnosia, i.e. disorders of the ability to recognize objects by one or another of their properties, we will again make a reservation that we do not mean the defeat of special "gnostic centers". We are talking about complex disorders of functions associated with the work of any analyzer.

Agnosia cutaneous and kinesthetic occurs with lesions of the parietal lobe. For the normal function of recognition, it is necessary that the afferent systems be preserved, projecting stimuli from the opposite side of the body into special cells of the sensitive area, which is part of the system of skin and kinesthetic analyzers.

Further subtle analysis and synthesis of skin and kinesthetic irritations is carried out in the vast areas of both analyzers, the cortical sections of which are mutually overlapping, especially in the anterior sections of the parietal lobe.

An example of cutaneous and kinesthetic agnosia is stereognostic feeling disorder. Stereognosy - recognizing objects by touch - is a common type of complex sensitivity. Disorder of stereognosy occurs with foci in the parietal lobe, posterior to the posterior central gyrus, while close functional and anatomical connections with the projection area of hand movements, especially the hand and fingers, are disrupted. In such cases, the patient with closed eyes cannot recognize objects when feeling them with the hand opposite to the focus.

Auditory agnosia can occur when the temporal lobes are affected, where the afferent auditory conductors fit. Auditory gnosia is the ability, acquired during life, to distinguish objects by their characteristic sounds. So, a car is recognized by a whistle, engine noise, a locomotive by a whistle, a clock by a tick, a person by a voice. Differentiation (discrimination) in humans by ear reaches high perfection. Auditory agnosia is extremely rare; apparently, for its occurrence, an extensive and, moreover, bilateral lesion of the temporal lobes is necessary.

Visual agnosia is associated with damage to the occipital lobes and their outer surfaces. Visual agnosia means recognition, differentiation of objects by their appearance. Partial visual gnosia refers to the ability to recognize color.

Visual agnosia is rare in the clinic; more often it is temporary, transient or partial; apparently, for its occurrence it is necessary to turn off the functions of both occipital lobes (outer surfaces of the hemispheres).

Olfactory and gustatory agnosia are disorders of complex analysis and synthesis of the corresponding stimuli; in practice, even with bilateral lesions, they are not installed.

Speech disorders

Speech is one of the late (phylogenetically new) functions of the cerebral hemispheres. Speech is only a human function: cytoarchitectonic fields that are most related to speech function exist only in humans.

Human thinking is always verbal. Verbal thinking and speech belong to the second signaling system, which is peculiar only to humans.

The second signaling system and speech are of tremendous importance in the history of the development of human relations and society, as well as in the individual development of man. On the other hand, the emergence of the second signaling system and speech is due to the very course of the historical process of the development of man and his social relations.

It is clear that in parallel with this in the cerebral cortex, a complex function of a combination of movements was formed that produced speech sounds, syllables, words, etc. Through long-term experience, "beating", skills of constructing certain series of sounds - motor speech stereotype - were associated with certain ideas. Part of the kinesthetic (motor) analyzer was specialized for this function. It is clear that it was formed near the projection area for the movements of the lips, tongue, and larynx - in the posterior part of the inferior frontal gyrus (Broca's area), in one hemisphere (in right-handers, in the left).

Inseparably with this was the process of distinguishing, recognizing conditional sound series - signals - by their sound and order of arrangement - gnosia of speech. In the auditory analyzer system, a specialized area for the analysis and synthesis of speech sounds was formed - in the posterior part of the superior temporal gyrus (Wernicke's area).

Consequently, the motor component of speech (expressive) is a special kind of praxia; sensory - a special type of auditory gnosia. Both speech functions are closely related.

The function of speech is extremely complex. As a manifestation of higher nervous activity and an integral part of human thinking, it, of course, cannot be narrowly localized in the cerebral cortex. Clinical experience shows, firstly, that the diversity of the nature of speech disorders is very large: secondly, the determination of the location of the lesion focus on the basis of only one speech disorders is rather difficult and often erroneous.

In the implementation of the most complex speech functions, not separate isolated cortical areas are involved, but complex functional systems covering vast areas of the cortex. This position does not at all negate the fact that different parts of the cortex are by no means equivalent in terms of speech functions. Clinical experience teaches that lesions of different parts of the cortex cause qualitatively different speech impairments. Therefore, we distinguish between individual areas, the defeat of which causes one or another, to one degree or another, speech disorders. But this does not mean that such areas are normally “speech centers” that control certain speech functions.

Wernicke's area is located in the temporal lobe, in the posterior section of the superior temporal gyrus. Through this zone, the analysis and synthesis of sound speech occurs in the cortex, the comparison of its elements with certain ideas, objects, concepts (recognition of oral speech).

Understanding of speech in a child develops earlier than other speech functions, therefore, the defeat of the Wernicke area entails the most serious damage, upsetting the function of other parts of the cortex associated with it in the activity of the cortex. The loss of the ability to understand human speech that occurs when Wernicke's area is affected is called sensory aphasia (verbal agnosia).

Broca's area is located in the frontal lobe, in the posterior part of the inferior frontal gyrus. A person builds his speech on the basis of his existing experience, pronouncing the syllables in such a sequence and in such a combination to create a word; words - to put together a phrase that expresses the desired thought. The motor function of speech acquired by experience during childhood (according to the principle of the development of conditioned reflexes) is associated with the indicated Broca's territory. When speaking, a person controls his own speech through a specialized part of the auditory analyzer - Wernicke's area and the analysis of kinesthetic stimuli from the speech muscles. The functions of Broca's domain are, of course, practical. The implementation of the movements of the tongue, lips, and vocal cords necessary for speech occurs through the region of the anterior central gyrus located next to Broca's zone , its lower section, where the motor paths to the muscles that participate in the formation of sound speech begin . When Broca's area is affected, so-called motor aphasia (verbal apraxia) occurs.

In isolation, the reading function, or vocabulary, is impaired when the focus is located in the parietal lobe, in the angular gyrus. Through this area, the letters are recognized, which are conventional symbols of sounds, and in a certain combination of them - words and phrases. Alexia is a type of visual gnosia. Written comprehension disorder (alexia) occurs with lesions at the junction of the temporal lobe (verbal gnosia) and the occipital lobe (visual gnosia).

The function of writing or graphing in isolation is impaired when the posterior part of the middle (second) frontal gyrus is affected next to the projection area of turning the eyes and head and movements of the hand in the left hemisphere in right-handers. This arrangement is understandable, since the process is highly associated with eye movement and is carried out with the right hand. Graphy is one of the types of complex praxia - written speech consists in drawing conventional, corresponding to the sounds of signs (letters), constituting words and phrases in certain combinations. The loss of the ability to write is called agraphia.

Features of the neurological examination of the child.

The purpose of the lesson : to study the features of examination and assessment of the state of motor functions and neuropsychic development of a child.

The student should know : the peculiarities of the development of the child's motor and neuropsychic spheres.

The student should be able to :
1) examine the child's reflex, motor and neuropsychic spheres;
2) assess the state of the motor sphere;
3) evaluate the neuropsychic development of the child.

Control questions:

1. What are the criteria for assessing the consciousness of infants?
2. What are the options for the size and configuration of the skull of newborns?
3. What sutures and fontanelles are palpated in a newborn?
4. What are the characteristics of the study of neurological status in infants versus adults?
5. What samples and tests are used to assess the motor sphere of newborns?
6. What seizures are observed in children of early life?
7. What meningeal symptoms are investigated in children in the neonatal period?

The newborn periodically makes spontaneous movements, alternately unbends and bends the legs, crosses them, pushes off the support. Hands move mainly in the elbow and wrist joints, movements are performed at chest level. The legs are more mobile than the arms. Spontaneous movements of the newborn are sharp, massive, impulsive, jerky, and suddenly follow each other. Along with a certain posture and spontaneous motor activity in a newborn, congenital reflex reactions are determined, the time of appearance of which and the degree of severity characterize his state and dynamics of development. The formation and extinction of these reflexes is of great diagnostic value. Assessment of the reflex background

of a child in the first year of life makes it possible to suspect, and in some cases, to diagnose cerebral palsy in the early stages of its development. This dynamic assessment and timely diagnosis are of decisive importance in the adequate correction of movement, speech, and psyche disorders.
Congenital reflexes are divided into two groups: segmental motor and suprasegmental posotonic automatisms. The first group of automatisms is divided into oral and spinal.

Oral reflexes

The search reflex is triggered by stroking a finger in the corner of the mouth without touching the lips, while the corner of the mouth is lowered and the head turns towards the stimulus. Disappears by 4.5 months.
The proboscis reflex of Kussmaul is **examined in the supine position. A quick blow to the lips causes the circular muscle of the mouth to contract, the lips are pulled into the proboscis. Disappears by 4.5 months.**
The sucking reflex is tested with the baby supine. When nipples are inserted into the mouth, rhythmic sucking movements occur. Disappears by 4.5 months.
Babkin's palmar-oral reflex is studied in the supine position. When you press on the palm closer to the eminence of the thumb, the mouth opens, the neck, shoulders and forearms are bent, the child seems to be pulled forward. The reflex disappears by 4.5 months.
Midface - Babkin's reflex is triggered by hitting the hammer on the midline of the face. As a result, the head is extended , followed by a return to its original position. The reflex disappears by 4.5 months.
Babkin's cervical - rotational reflex is caused by a jerky blow of the hammer in the cheek area. The response is to rotate the head towards the stimulus, followed by a return to the starting position. Disappears at 4.5 months.
Astvatsaturov's nasolabial reflex is caused by tapping on the dorsum of the nose. In response, there is a contraction of the circular muscle of the mouth. The reflex is reduced at 3 months.

Spinal automatisms

Robinson's grasp reflex is tested in the supine position. When a finger or any object touches the palms of a newborn, he firmly grabs it, sometimes so strongly that the child can be lifted in this position. The reflex disappears by 5.5 months.
The Moro reflex is examined with the child supine. The child is pulled up by the arms, without lifting the head from the diaper, then suddenly the arms are released. The child takes his hands to the sides and unclenches his hands, after a few seconds the hands return to their original position. The reflex disappears by 4 months.
The stance and automatic walking reflex is triggered in the vertical suspension position. The child is slightly tilted forward, placed on a support. At the same time, he straightens the torso, leaning on a full foot, makes step movements, without

accompanying them with the movement of his hands. The reflex disappears by 2.5 months.

The crawling reflex is examined in the **prone** position. The child makes crawling movements - spontaneous crawling. If you put your palm to the soles, the child pushes off from it with his feet and crawling increases. The reflex disappears by 4.5 months.

The Galant reflex is triggered in a child in a prone position . In case of paravertebral irritation of the skin of the back, the newborn bends the body in an arc open towards the stimulus, turns the head in the same direction, sometimes unbends and abducts the leg. The reflex disappears by 4 months.

The Perez reflex is tested with the child in the prone position. They are carried with fingers, lightly pressing along the spinous processes of the spine from the coccyx to the neck, lordosis appears, the child raises his head, raises the pelvis, bends his arms and legs. The reflex disappears by 3 months.

As the child grows, unconditioned reflexes disappear. On their basis, numerous conditioned reflex reactions are formed. However, in the case of the development of cerebral palsy, the extinction of these reflexes is delayed. With a pronounced grasping reflex and with a delay in the hand of the child, they acquire a pathological setting. They are clenched into a fist, thumbs are brought in, hands are pronated to varying degrees. This attitude prevents the development of fine motor skills and, as a result, psycho-pre-speech development. A delay in the reaction of support and automatic walking when the feet and support come into contact causes a sharp increase in the extensor tone in the legs and trunk. The child walks on his toes, crosses his legs, throws his head back. In this case, the development of the reaction of equilibrium during standing and walking is delayed. A delay in spinal automatisms leads to a delay in the formation of straightening and balance reflexes.

The tonic labyrinth reflex , caused by changes in the head in space, which lead to stimulation of the otolith apparatus of the labyrinths, as a result, the tone of the extensors is maximally expressed in the supine position, and the flexor activity is relatively inhibited. In the prone position, the ratios are reversed.

Asymmetric tonic **cervical reflex is a** proprioceptive reflex resulting from stretching of the neck muscles . Turning the head to the side to shoulder level is accompanied by the extension of the limbs to which the face is facing and the flexion of the opposite ones. The reaction of the hands is more pronounced than from the legs.

Symmetrical tonic cervical reflex is a proprioceptive reflex from the musculo-articular formations of the neck when bending the head, flexor tone in the arms and extensor tone in the legs increases, extension leads to the opposite effect.

The normal development of the child's movements after the neonatal period is characterized by two interrelated and interdependent processes of the development of posture reflexes (postural reflexes), which include straightening reactions, protective and other reactions integrated at the midbrain level, and inhibition of congenital automatisms of the spinal stem level.

Straightening and equilibrium reactions .

The labyrinthine straightening positioning reflex is clearly formed by the age of two months. The child is freely held and moved in the air, the child's head is set so that the line of the mouth is parallel to the plane of support. This reflex stimulates the development of symmetrical chain reflexes aimed at adapting to an upright position. It provides the installation of the child's neck, torso, arms, pelvis, legs.
The cervical straightening reaction is finally formed by the age of four months; passive or active turning of the head to the side is followed by rotation of the entire trunk. With the maximum severity of the reflex, there is a "turn by the block" now the straightening reflex of the body helps to straighten the head relative to other parts of the body in space.
The straightening reflex becomes pronounced at the age of 6 - 8 months, contributes to the fact that the body no longer follows the head as a whole, it rotates between the shoulder girdle and the pelvis when turning to the side, and later on to the stomach, further rotation within the axis body enables the child to roll over from belly to back, from back to belly, get on all fours and take an upright position.
The defense reaction of the hands and the Landau reflex do not belong to the straightening reflexes, but at certain stages it contributes to motor development. A protective extensor reaction of the arms occurs in response to a sudden movement of the trunk forward (at 4 months), to the side (at 6 months), back (at 9 months). These reactions contribute to the fact that the child in a sitting position can maintain his body weight with his arms extended forward (at 6 months), to the side (at 8 months), back (at 10-12 months). At first, the hands are clenched into fists, but then they create support on the palm. The Landau reflex is combined with straightening reflexes and is a part of them. The child is held free in the air, face down. At first, he raises his head, so that the face is in a vertical position, and the mouth is in a horizontal position, then tonic extension of the back occurs, but the reflex appears at 5-6 months, in the second year of life it begins to fade.
Equilibrium reactions are provided by the interaction of the vestibular nuclei of the basal ganglia, the nuclei of the subthalamic region, the cerebellum and the cerebral cortex. These reactions make it possible to maintain and restore balance in the process of physical activity. Like straightening reactions, equilibrium reactions develop over a long time in a specific sequence. They appear and fade away when the straightening reactions are already fully established. In the supine position, they become pronounced when the child is already sitting without support (8 months), in a sitting position they appear when the child can stand (at 9-10 months), and in a standing position, when the child is already walking (at 10 - 12 months). Equilibrium responses are not improved until the child has advanced to a higher stage in the development of straightening responses. By 18-24 months All equilibrium reactions are already formed, but not yet perfect. They develop and improve up to 5-6 years.
When the brain is damaged during its formation, intensive growth and differentiation, the ontogenetic sequence of motor development is disrupted. Along

with a slowdown in the formation of normal postural mechanisms, tonic reflexes are activated, which aggravates the motor defect. In children with cerebral palsy, their effect can be detected in various positions. A child in a supine position cannot raise his head or does it with great difficulty, cannot stretch his arms, turn, he has no prerequisites to sit down. ***The tonic asymmetric cervical reflex*** is manifested by the asymmetry of the body ("fencer's pose") and voluntary movements. ***The tonic symmetrical cervical reflex is*** most pronounced when the head is included in the movement. Flexion of the head leads to an increase in flexion in the arms, extension in the legs, extension causes the opposite effect. Changes in posture, muscle tone, spontaneous movements under the influence of this reflex are equally pronounced on both sides. In this case, contractures can develop. In the prone position, the child cannot raise his head, turn to the side, free his arms and lean on them, bend his legs and kneel, while maintaining balance.

Symmetrical tonic cervical reflex has a symmetrical effect on the muscles of the arms and legs. For the position on the stomach with the head down, pronounced flexion in all joints of the arms, extension of the legs, which leads to kyphotic curvature of the spine, are typical. This prevents support on the forearms and hands.

On all fours: the labyrinthine tonic reflex interferes with standing on all fours due to flexor spasticity in the arms and legs.

Asymmetric cervical tonic reflex, causing asymmetry in muscle tone, creates obstacles to support on the hands and knees. In a position on all fours, the child cannot set his head in the midline, which creates a constant threat of loss of the supporting function of the hands.

Symmetrical tonic cervical reflex is manifested by impaired support function of the limbs. The constantly changing distribution of the muscle tone of the arms and legs, depending on the position of the head, even in mild cases, creates great difficulties for crawling on all fours.

In a sitting position, the labyrinth tonic reflex sharply disrupts motor activity. With a prolonged stay of the child in the described position, flexor contractures develop in the hip and knee joints, persistent kyphosis of the spine.

The asymmetric cervical tonic reflex in this position is expressed in turning, tilting the head to the side and asymmetric position of the limbs, which makes it difficult to maintain a free sitting posture and manipulate the hands.

Symmetrical tonic cervical reflex makes it difficult for the child to maintain a sitting posture. When the head is extended, the child falls backward, and when flexed, the child falls forward.

The pronounced labyrinth tonic reflex prevents the vertical position of the body and the support of the legs, since when standing up, at the moment the body moves forward, a total flexion posture occurs. Standing and moving the patient is impossible.

Under the influence of an asymmetric cervical tonic reflex, the position of the head and trunk is asymmetric at rest and when walking. Stabilization of the standing position is achieved through excessive flexion of the head and kyphosis of the

spine. To maintain balance when moving forward, the child takes his arms to the sides, walks with small steps.

A positive supportive response leads to a simultaneous increase in muscle tone in the extensors and flexors of the legs, as a result of which the legs become rigid and become "columns" for support. This reaction is stimulated by tactile irritation of the soles upon contact with the support, and the transition from a sitting position to a standing position, and vice versa, is also disturbed. A pronounced supportive reaction sharply violates the balance reaction, since the rigidity of the legs does not allow them to move quickly when the center of gravity of the body is shifted.

Friendly movement (sinkenezii) - a tonic reaction, causing a widespread increase in spasticity in the parts of the body that do not have direct relation to the movement performed at the moment. Pathological synkinesias inhibit the development of trunk straightening reflexes, balance reactions, and purposeful movements.

When the nervous system is damaged, the mechanism of equilibrium reactions is disturbed, the posture of the patients is unstable. To maintain balance, the child compensatory flexes the knee and hip joints and the vertical projection of the center of gravity falls on the foot.

Thus, contractures and deformities are formed under the influence of an increase in muscle tone, the distribution of which depends on reflexes, synkinesias, pathological reactions of balance, a long stay of the child in the same position.

Contractures and deformations.

In the *lower extremities* , adduction, intrarotator contracture of the hip joint is more characteristic. The development of contracture is associated with spasticity of the adductor muscles of the thighs. Its combination with flexion contracture is often observed. Due to an imbalance in muscle tone, dislocation of the hip joint is possible, usually observed in severe patients who are unable to move independently. Flexion contractures of the knee joints are often combined with flexion-adduction contractures of the hips and equinovarus deformities of the feet. Recurvation of the knee joints is more typical for the forms of cerebral palsy, accompanied by low muscle tone. Hallux valgus is usually associated with flexor - adductive intrarotator hip contracture and knee flexion. Horse foot is the most common leg deformity. Spasmodic contraction of the calf muscles and relative weakness of the peroneal muscles lead to plantar flexion of the feet and support on the toes. The calcaneal foot develops as a result of an incorrectly performed achillotenotomy in order to correct the equine foot. In rare cases, it is primary and is formed due to the predominance of the tone of the extensors of the foot and fingers over the tone of the flexors. The plano-valgus foot is caused by weakness of the muscles that lift the medial edge of the foot, and hypertonicity of the peroneal muscles.

In the *upper extremities* , adductor and intrarotator contractures of the shoulder are more often noted, due to spasticity and shortening of the pectoralis major muscle, latissimus dorsi muscle, shoulder pronators. Flexion contracture of the elbow joint

is formed as a result of shortening of the biceps brachii and brachioradialis muscles. Flexion contracture of the hand is almost always combined with abduction to the ulnar side, flexion contracture of the fingers and adduction of the thumb.

In the area of the trunk, deformities are manifested by scoliosis or kyphosis of the spinal column, intersection of the pelvis, and asymmetry of the chest. These deformities are caused by various violations of the position of the trunk due to tonic activity, pathological postures aimed at stabilizing the trunk and maintaining balance.

Contractures and deformations fix the child in pathological positions, impede the development of static and locomotor skills, and limit the possibilities of social adaptation.

Examination of the motor sphere usually begins with the identification of muscle weakness, which can accompany paralysis and paresis of both peripheral and central character. Attention is drawn to bone deformities, which may be secondary. For example, various injuries to the spine, chest, ribs, limbs and joints. More often this is due to muscle weakness with flaccid paresis. Examples are rickets, rickets-like diseases, mucopolysaccharidoses, etc.

Limitation of active movement and decreased muscle strength are important characteristics of paralysis and paresis. Active and passive movements are studied both in the proximal and in the distal parts of the limbs. Muscle strength, tone, tendon, periosteal, abdominal and plantar reflexes are tested.

A complete study of the range of active movements and testing of muscle strength are possible in children with normal intelligence, usually after 3 years. If you have to examine a child of an earlier age or a patient with mental retardation, then you should resort to various methods. Observing the child's walking, it is possible to reveal the features of motor activity in the form of a paretic, atactic or spastic-paretic gait. A pretentious gait is caused by hyperkinesis, flaccid paresis, etc. Observing the child's play, an attempt to take him in the upper limbs, about which of them is paretic.

Assessment of the state of motor skills of a child of the first year of life must be carried out taking into account the age norms of the rate of motor development.

Muscle strength is examined in individual muscle groups of flexors, extensors, abductors, adductors, etc. The state of muscle strength is carried out according to a 5-point system:

- complete paralysis - 0 points;
- active movements are absent, but muscle tension is determined by palpation when trying to move - 1 point;
- movements are possible with the exclusion of the gravity of the limb - 2 points;
- muscles overcome the gravity forces of the limb, but do not overcome additional resistance - 3 points;
- muscles do not fully overcome resistance - 4 points;
- muscle overcomes resistance - 5 points.

In children under 1.5 - 2 years old, to determine muscle strength, the child's resistance to examination, withdrawal of the limbs can be used. The same technique can be used in older children with mental retardation. Such an examination allows you to identify the affected limb and decide which departments (distal or proximal) suffered more.

In order to determine the weakness of a particular muscle group, you can use

Some special tricks. With weakness of the flexors of the neck, an attempt by a lying child to lift the child by the arms leads to a sharp throwing back of the head. Lack of function of the proximal arms and shoulder girdle can be identified by lifting the child "under the armpits". In this case, there is a symptom of "free shoulder girdle". The symptom of a "tripod" is indicative, indicating weakness of the back muscles. In this case, in order to support himself in a sitting position, the patient leans on his hands. The "frog" abdomen, revealed in the supine position and indicating the weakness of the abdominal muscles, has a certain diagnostic value.

In assessing the state of the movement system, an important place is occupied by the study of muscle trophism, the presence of atrophy and pseudohypertrophy, fibrillar twitching. Atrophy is evidenced by a decrease in the circumference of the limb, a decrease in muscle mass detected by palpation, and muscle laxity.

The state of muscle tone, the presence of retractions and contractures is determined by passive movements. The position of the patient in bed, the posture of the limbs already indicate the predominance of tone in one or another muscle group. Muscle tone refers to the minimum tension of a relaxed muscle. The presence of muscle hypotension indicates peripheral muscle paresis or insufficient function of the cerebellum and striatal system. An increase in muscle tone is observed with damage to the pyramidal tract (spasticity) or the pallidal system (rigidity).

Checking deep and superficial reflexes is carried out only with minimal contact with the patient, therefore it does not present any particular difficulties. On the upper extremities, the metacarpal-ray reflexes (C5-C8), from the biceps muscles (C5-C6) and triceps muscles (C7-C8) are examined. On the lower extremities, knee (L7-L8) and Achilles (S1-S2) reflexes are determined. An increase in deep reflexes is observed with damage to the pyramidal pathway and accompanies central paralysis. A decrease or complete absence of deep reflexes is characteristic of flaccid or peripheral paralysis.

Abdominal, plantar, cremasteric reflexes are important for diagnosis. Their decrease is a consequence of insufficient functioning of the corresponding reflex arcs. Abdominal reflexes correspond to D - D, cremasteric L - L, plantar L - L segments of the spinal cord.

Identification of plantar reflexes, excluding the presence of pathological foot reflexes (Babinsky, Rossolimo, Bekhterev, Oppenheim, etc.), which are found when the pyramidal pathway is affected.

Coordination of movements is carried out due to the activity of the cerebral cortex, strio-pallidary system, brainstem, spinal cord, sensory system and cerebellum. Defeat of - Latter leads to ***ataxia*** . Distinguish between static and locomotor ataxia. Static ataxia, leading to disruption of faiths - tikalnogo posture,

due to lack of function of the cerebellar vermis. Locomotor ataxia, detected during active movements, occurs when the cerebellar hemispheres and their connections are damaged.

Identification systems koordinatornyh disorders in infants WHO - Rasta presents certain difficulties. Children born with invisible - Lost or damage to the cerebellum, as a rule, detained in the engine development. Their ataxia is not immediately apparent due to muscle hypotonia. Cerebellar hypotonia is not accompanied by a decrease in su - hozhilnyh and periosteal reflexes. Check cerebellar function - tions using paltsenosovoy, kolennopyatochnoy and other samples not all - GDA manages to hold in young children and are lagging behind in psihoreche - tion development. On examination of such children should pay attention to the child's ability to retain the upright position, unstable - ness and staggering when walking, frequent falls. Keep in mind ha - rakter and precise movements when eating, trying to take the subject, show the finger of his body, or a toy. In this case, you can detect not only ataxia, but also intentional tremors.

Movement disorders may be caused by insufficient - the accuracy of the extrapyramidal system. It is expressed either the excess - GOVERNMENTAL ***violent movement (hyperkinesias)*** or ***hypokinesia,*** ie, poverty of movement, their slowness, in solidifying defined.. - hydrochloric posture amimia. Identify violations in the form of hyperkinesis, which more often than hypokinesia, common in sick children can be both in on - observations of the movements of the child, and by using special - GOVERNMENTAL receptions. Violent movement of its Clinical manifestations - leniyam very diverse and always indicate an organic lesion of the subcortical brain structures. Children bowl meets ***ate toidny hyperkinesia*** , is a slow tonic su - the road at the same time including the effect muscles agonists and anta - gonisty. Limb movements become pretentious, redundant, worm-like. Athetoid hyperkinesis extends more to the distal extremities, facial muscles. Muscle tone during movement increases, becomes rigid, after the end of the movement it decreases to hypotension. Athetoid hyperkinesis, like other violent movements, disappears in sleep.

Torsion spasm differs in that the spasm is rotational in nature. Violent muscle movements are characterized by the rotation - Niemi around the axis of the limb, torso. Often there is a combination of athetoid hyperkinesis with torsion ones. When infantile cerebral pa - ralichah, metabolic diseases such giperkinezy brain occur frequently. There are choreic, myoclonic hyperkinesis, which differ from the previous ones in a fast pace, sweeping movements.

To assess hyperkinesis, it is necessary to characterize the amplitude and formula of movement, rhythm and localization. In order to detect hyperkinetic necessary to observe the child at ease and perform - Research Institute of a motor act (when trying to take a toy odes - Noah or with the other hand, walking or driving with the help of parents, game). Under emotional stress giperkinezy amplified and on - has also been observed with greater ease.

An important place in assessing the extent of damage for motor function - Nima ***study gait characteristics*** of the child. This method of definition - Nia movement disorders is available from any, on their own or with the help of others-moving child,

regardless of its level of intellectual development. When bilateral pyramidal violated - niyah (cerebral or spinal) is typical ***spastic-paretic gait*** . The baby rests on your toes, heel raised and rotated outwards, legs bent at the knee and hip joints, hip while - vedeny. These children wear shoes on the inside of their socks.

With the defeat of the cerebellum or its connections appears ***ataxic on - Walker*** . The child walks uncertainly, spreading his legs wide apart, deviating to one side or another, trying to stick to the surrounding objects.

In a patient with gait giperkinezami different redundant netse - expediency, artsy limb movements. Equilibrium is affected to a lesser extent.

Hemiparetic gait , caused by lesions of the pyramidal system of one of the hemispheres of the brain, typical of tightening or reducible - lakivaniem lower limb, which is dominated by the tone of the extensor muscle groups. The upper limb on the same side on - hoditsya flexion and driving (posture Wernicke-Mann).

Children with sluggish paresis of distal lower extremities ho - DYT highly lifting knees, with feet dangling, "prishlepyvayut". This gait is called ***"steppage".***

The peculiarity is characterized by the walking of children who have weakened pelvic muscles and proximal parts of the legs. This can be observed in various muscle diseases and traumatic injuries on - yasnichnogo thickening of the spinal cord. Patients walk swaying from side to side, with difficulty climbing the stairs. This gait is called a ***"duck"*** gait **.**

SENSITIVITY STUDY TECHNIQUE

Sensitivity study in young children gives Minor - battening information on the state of the nervous system. This is due to the fact that only superficial sensitivity is developed in a newborn, and a deep one is formed only by 2 years. In addition, children of early vozras - she could not localize the irritation. Rough surfaces disorders - hydrochloric sensitivity can reveal the general anxiety, reflexive - the secondary protective response, crying, etc...

METHODS FOR STUDYING NERVO-MENTAL DEVELOPMENT

Assessment of the level of neuropsychic development of infants and young children presents significant difficulties. Therefore, you must not - a one-time observation of the child at the time of communication with adults, reactions to the toy, visual and auditory reactions, etc. In those.. - chenie first months of life the baby's position depends on the particular - stey muscle tone (flexor position of the limbs). In this case, the arms are bent at all joints, brought to the body, the fingers are clenched into fists. The legs are bent at the hip and knee joints, apart at the hips. Already ***in the first month of the*** child reacts to bright light, pripod - Nima and holds the head of the prone position. The movements of the eyeballs are jerky. An undifferentiated reaction to sound appears , children emit guttural sounds.

In the second month, saved flexion of the extremities, increases the amount of active movements, the child holds the head of the longer polo - zheniya on his belly for a few seconds, holding his head in ver - tikalnom position longer captures glance, a smile appears gu - Lenie. Reflexes of support and automatic gait fade away, physiological astasia - abasia occurs. Symmetrical and asymmetric reflexes begin to appear , contributing to the preparation of the formation of an upright body position.

In the third month of life increases the amount of active movements, CCA - cially in the hands. A child holds a well-head in an upright polo - zhenii, turns his back on his side, visual reaction Stano - vyatsya duration, turns his head to sounds, there sochetannyj turning the head and eyes to the side. Humming is pronounced, vowel sounds appear . Labyrinth cervical tonic reflexes are weakened. During this period weakened grasp reflex, there are first arbitrary - motions. The child actively picks up toys.

In the fourth month, the child holds his head well, picks up toys and pulls them into his mouth. When pulling up by the arms, he sits down, in a horizontal position on his stomach he lifts and holds not only the head , but also the shoulder girdle. Unconditioned reflexes and hypertonicity in the flexor muscles fade away . The child distinguishes sounds, voices, makes loud sounds.

At 5-6 months, the child sits with support, turns from back to stomach and from stomach to back, distinguishes familiar faces. Emotions become brighter, the first syllables "ba", "pa", "ma", "dya" appear.

At 7-8 months, the child sits steadily, gets on all fours. Deeds flushes attempts to sit, and occasionally sits. He gets up on his feet and stands for a short time at the support. Shifts toys from one hand to another. Claps his hands, pronounces the syllables "ba-ba", "ma-ma". Expresses surprise, interest, recognizes strangers.

At 9-10 months, the child kneels down, holds onto a support, stands, makes attempts to walk, waves his hand "goodbye", eats from a spoon, knows the meaning of some words, shows objects, toys, says separate words "mother" , "woman", "uncle", "aunt", performs some pro - sby adults understand taboos.

At 11-12 months, the child begins to walk on his own, although he still often falls, manipulates toys freely, knows the names of many objects, eats on his own with a spoon, the vocabulary increases to 10-12 words.

An important point in assessing the neuropsychiatric development of a child is the ***study of pre-speech and speech development.*** Delay in pre-speech and speech development, as a rule, is combined with intellectual disability. Violations of the development of speech can range from mild types of speech disorders (dysarthria, rhinolalia) to total alalia or aphasia.

It is necessary to bear in mind the connection between speech development and the state of the child's auditory function. In children with deafness, speech development is delayed or completely absent.

If deafness is combined with intellectual disability, then speech function suffers significantly. The reasons leading to speech disorders are varied. These include intrauterine lesions of the speech cortical zones as a result of the effects of infections, intoxication , endocrine and metabolic disorders, and pathological childbirth.

Alania is characterized by a pronounced congenital speech underdevelopment in a child. Such a child had never spoken before. This defect is caused by dysfunction of the cortex and is characterized by either misunderstood - Manius reversed speech or inability to play it. In the first case, alalia is called ***sensory*** , in the second, ***motor*** . In most cases there is a combination of one kind and another alalia, t. E. ***Total ala lija*** . As a rule, general speech

underdevelopment is combined with mental incompleteness of the child. Speech disorders in patients with palia range from mild, easily corrected, to pronounced, difficult to correct.

Another type of speech disorders caused by the violation of certain zones of the cortex as a result of postnatal diseases is called

aphasia. **Aphasia is** characterized by the loss of previously existing speech functions. Aphasia cause infectious diseases can be ner - implicit systems, such as meningoencephalitis, leykoentsefality, grass - matic brain injury. Aphasia, like alalia, mo - Jette be ***total*** or ***partial, motor, sensory*** and other insurgents. - vit lost it is sometimes less difficult than re-train never tell a child. However, this is possible if the disease - s, not prone to progression.

These speech disorders caused by lesions of the definition - PARTICULAR structures cortex of the dominant hemisphere of the brain, the races - laid mainly in the frontal and temporal lobes. They can be combined with such violations of the cortex functions as disfafiya, dis - Lex. There are other types of speech disorders called dysarthria, which are caused by lesions of systems such as pi - ramidnaya, striae-pallidarnaya and cerebellar. The defeat of each of them has its own clinical features. Dysarthria, associated with damage - it stri-pallidarnoy system, such as hyperkinetic odds - IU cerebral palsy, because giperkinezy cover articulation muscles. At the same time, speech becomes slurred, jerky, explosive, stretched.

A somewhat different nature is it a child suffering mozzhech - kovoy failure. In this case, speech bears signs of atactic violations and is called chanted. The patient says, layer - in not together, and said, highlighting the "stamping" each syllable.

Dysarthria, due to inadequate functioning of the feast - Midna system associated with impaired cortico-nuclear pathways and as a rule, two-way involvement in the cerebral process. Muscle - language travel and other muscle groups involved in the articulation, locat - ditsya thus able to spastic paralysis. Arises dizart - dence, which is accompanied by drooling, patients are not able to swallow saliva. This dysarthria is one of the manifestations of pseudobulbar paralysis.

The extent and depth speech disorders within each species dis - arthritis may vary from light to full anarthria disorders where the patient can not utter a word or a syllable.

In addition to the listed speech disorders, which have an organic basis, its functional disorders (stuttering, mutism) are distinguished.

Used Books:

1. Barker R., Barazi S., Neil M. Visual neurology. - M., 2006 .-- 136s.
2. Gusev E.I., Skvortsova V.I., Konovalova A.N. and other Neurology. National leadership. –M., 2008. - 850 p.
3. Nikiforov A.S., Gusev E.I. Private neurology. - M., - 768 p.
4. Skvortsova V.I. Guide to Practical Exercises in Topical Diagnostics. - M., "Litter", 2006. - 240 p.
5. Yakhno M.N., Parfyonova V.A. Propedeutics of Nervous Diseases. Situational tasks and tests. - M., Medical Information Agency, 2009 .-- 172 p.
6. Aminoff MJ, Geenberg DA, Simon RP Clinical Neurology. - Medical Publishing Bivision, New York, 2009 .-- 480 p.
7. Jose Biller. Practical Neurology Lippincott - Reven, Philadelphia. New York, 2005 .-- 416 p.