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NEUROLOGY: TOPICAL DIAGNOSIS

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The study guide "Neurology: Topical Diagnosis" outlines the principles of topical diagnosis in neurology based on the analysis of symptoms and syndromes identified during clinical neurological examination of patients. The paper describes anatomical data of the nervous system structures, examination methods, syndromes of functional and organic disorders, and the relationship of the revealed symptoms to the lesions of specific neurological structures. The study guide is intended for students pursuing a bachelor's degree in international medical faculty and intern doctors in «General Medicine».

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LIST OF ABBREVIATIONS

AVM	-	arteriovenous malformation
BP	-	blood pressure
VA	-	vestibular ataxia
AI	-	autonomic index
ANS	-	autonomic nervous system
WHO	-	World Health Organization
GABA	-	gamma-aminobutyric acid.
HLRC	-	hypothalamic-limbic-reticular complex
B	-	brain
Hz	-	hertz
BPV	-	benign positional vertigo
PCG	-	postcentral gyrus
HG	-	Heschl's gyrus
MRI	-	magnetic resonance imaging
NS	-	nervous system
ACVA	-	acute cerebrovascular accident
ARVI	-	acute respiratory viral infection
UN	-	United Nations
PNS	-	peripheral nervous system
MS	-	multiple sclerosis
SNS	-	sympathetic nervous system
s/c	-	spinal cord
USDG	-	ultrasound dopplerography
USE	-	ultrasound examination
CNS	-	central nervous system
CN	-	cranial nerves
HR	-	heart rate
EMG	-	electromyography
ENMG	-	electroneuromyography
EEG	-	electroencephalography
EchoEG	-	echoencephalography
D=S	-	dextra = sinistra

INTRODUCTION

Neurology is one of the most complex branches of medical science, studying the nervous system in normal conditions and pathology. Neurology includes neuroanatomy, neurophysiology, neurohistology, and neuropathology. The first components are part of general neurology, whereas neuropathology is categorized as particular neurology. Particular neurology deals with the study of etiology, pathogenesis, and clinical manifestations of nervous system diseases, including their diagnosis, treatment, and prevention. Neurosurgery is the branch of surgery that studies operative methods of treating nervous system diseases. However, knowing the basics of neurology is necessary before starting to study a particular neurology. Clinical and topical diagnoses are impossible without understanding the functional significance of the significant neuroanatomical structures.

The importance of the fundamentals of topical diagnosis in neurology can be compared to the alphabet in the educational system, just as in clinical neurology. Without knowledge of the necessary primordial fundamentals, further development is significantly stagnant.

Topical diagnosis is the key to clinical neurology.

It is not by chance that the nervous system is called the "eloquent organ" since any pathology at any level of nerve structures is manifested either by loss of function of these structures when affected or by hyperactivity when irritated. What makes the nervous system unique is its high degree of specialization. Identification of characteristic signs allows the localization of the damage with millimeter accuracy.

The relevance of neurology is exceptionally high. The prevalence of diseases of the nervous system is a testament to this.

The growth in the number of patients with neurological pathology is steady and relentless. The severe consequences in the form of profound disability and mortality also emphasize the importance of knowledge of neurology [1,2].

Knowledge of the basics of neurology for doctors of any specialty will significantly expand the opportunities of clinical reasoning, help to orient in time, provide emergency care, and save patients from disability and lethality.

This study guide explains the basics of topical diagnosis in clinical neurology. It addresses the sensory and motor areas, the extrapyramidal system, the cranial nerves, the cerebral cortex, and the autonomic nervous system. Separate chapters are devoted to each of these systems, describing critical summary anatomical data, examination techniques, symptoms, and syndromes of affection of these systems, and topical diagnosis. The consistency of the methodology for studying the above systems allows for the development of the students' clinical reasoning. At the end of each chapter, figures are provided to facilitate learning and retaining this material. Multiple-choice tests with answer keys for self-checking follow the conclusion.

1. SENSORY AREA

Sensitivity is the ability of a living organism to feel and perceive the effects of stimuli of the external and internal environment and respond to them with adequate forms of reaction. The sensory area is comprehensible once the analyzer system has been studied.

I.P. Pavlov introduced the concept of analyzers and analyzer systems; he grouped all nerve elements involved in transforming, conducting, and processing the same type of nerve impulses into a functional system. Analyzers consist of receptors, conductive tracts, and cortical areas.

Analyzers ensure that all information entering the brain from the external and internal environment is received, stored, synthesized, and analyzed.

Part of all incoming information is perceived in the form of sensations recognized by a person; however, there is a part that is not consciously perceived, yet the brain controls both.

Excitable formations, known as receptors, transform various forms of energy into a bioelectrical potential - into a nervous impulse - when acted upon by stimuli. This process is called transduction. The transmission of a nerve impulse is called transmission. Perception and analysis together constitute perception, which primarily occurs in the brain's cerebral cortex.

Receptors are classified as follows:

- Exteroceptors are divided into contact (pain, temperature, tactile, mechanical) and distant (color, smell, sound, light).
- Proprioceptors (deep sensitivity: pressure, weight, vibration, and others)
- Interoceptors (found in internal organs and vessel walls) [3,4].

1.1. Sensitivity classification

1. **General sensitivity**
   - ***Exteroceptive (superficial)***: pain, temperature, and light tactile
   - ***Proprioceptive (deep)***: muscle-joint, kinesthetic, vibrational, sense of weight and pressure, deep tactile
   - ***Interoceptive***: associated with internal organs and vessels
2. **Complex sensitivity:** discrimination, stereognostic, two-dimensional spatial, localization.

1.2. Anatomical data of the sensory area

The sensory analyzer for both superficial and deep sensitivity consists of three neurons.

Superficial sensitivity tract

The first neuron is located in the spinal intervertebral ganglia. The dendrites of the first neurons begin with receptors located in the superficial layers of skin and mucous membranes (Ruffini's corpuscles react to heat, end-bulbs of Krause - to cold, Meissner and Merkel's corpuscles - to touch, free nerve endings - to pain). The first neurons' axons enter the spinal cord's posterior horns through the posterior roots.

The second neuron is located in the posterior horns. The axons of the second neurons pass obliquely across the anterior gray commissure 2-3 segments higher to the opposite side. Then, they ascend through the spinothalamic tract (tr. spinothalamicus lateralis).

The third neuron is located in the thalamus. The axons of the third neurons in the thalamocortical tract pass through the posterior femur of the internal capsule, the white matter of the cerebral hemispheres, and reach the postcentral gyrus of the parietal lobe.

In the postcentral gyrus, receptor fields of the opposite half of the body are projected in reverse sequence: in its upper section, receptors of the skin of the leg are represented; in its middle section - of the trunk and arm, in its lower section - of the neck and head [4, p 9].

Deep sensitivity tract

The first neuron is located in the spinal intervertebral ganglia. Dendrites of the first neurons begin in the receptors of synovial membranes of joints and articular ligaments of muscles and muscles and go as part of peripheral nerves, nerve plexuses, spinal nerves, and spinal nodes. The axons of the first neurons enter the posterior columns through the posterior roots, forming the medial thin bundle of Goll at the lumbar and thoracic level and the Burdach lateral wedge-shaped bundle at the level of the cervical thickening. These bundles ascend their side to the medulla oblongata.

The **second neuron** is located in the gracilis and cuneate nuclei (nucl. gracilis et cuneatus), located in the medulla oblongata. The axons of the second neurons make a decussation over to the opposite side, going upward.

The third neuron is located in the thalamus, whose axons pass through the posterior third of the posterior pedicle of the internal capsule and reach the postcentral gyrus as part of the radial vein.

The projection of body parts in the postcentral gyrus is inverted. Thus, facial sensitivity is at the bottom, trunk, arm sensitivity is in the middle, and leg sensitivity is in the upper part of the postcentral gyrus of the opposite side [5,6].

1.3. Methodology for the examination of the sensory area

(performed with the patient's eyes closed)

Superficial sensitivity examination

Pain sensitivity

A sharp item such as a needle or pin is used, or - some neurological hammers have special tips with a sharp end. A mild tingling irritation is applied to symmetrical areas of the skin. The patient is asked at each touch whether this tingling touch feels "sharp" or "blunt" and whether he/she feels the same everywhere.

Temperature sensitivity

Two test tubes are used, each filled with water of different temperatures: one with warm and the other with cold water, or items of different temperatures with which a doctor touches the patient's skin and asks him/her what he/she feels: whether the item is warm or cold, whether he/she feels the same temperature on different parts of the body. A temperature difference of 1-2 °C usually is perceived.

Light tactile sensitivity

Light tactile sensitivity is checked by lightly touching symmetrical areas of the skin with a cotton wool piece, a soft brush, or a piece of paper. At each touch, the patient is asked to respond whether he/she feels the touches. However, tactile sensitivity is different everywhere. Tender areas, such as lips, fingertips, inguinal folds, and medial surfaces of hands and feet, are more pronounced than on the dorsal and proximal parts of the extremities or trunk.

Deep sensitivity examination

The ***articular-muscular sensation*** is examined in the following way: the patient is asked to lie relaxed; these movements are passive for the patient, as the doctor himself, with the patient's eyes closed, makes movements in the patient's arms and legs (distal or proximal) in different joints (interphalangeal, wrist, ankle, and others) in different directions (up, down, sideways) with different scopes of movement. The examination starts from the distal parts of the extremities (fingers) and proceeds proximally, including the shoulder and hip joints. The patient is asked which joint or finger the doctor takes, what the doctor is doing, bending or extending, and in what direction he/she is moving. Typically, a person is making an unmistakable determination and answering all questions easily.

Vibration sensitivity is examined with a sounding tuning fork (128 or 256 Hz), the stem of which is placed on bony surfaces (mastoid, spinous processes, ankles). In doing so, the patient is asked what they feel and how they feel the vibration. When the patient stops feeling it, the tuning fork is moved to a symmetrical place of the opposite extremity or another obviously healthy part of the body, and the duration of perception of vibration of the sounding tuning fork by the examined and healthy parts is compared.

Kinesthetic sensitivity is also examined with the patient's eyes closed; the doctor takes the first two fingers on any area of skin in a fold and moves it in different directions while asking the patient which direction he moves the skin fold. Typically, the examinee freely and easily answers these questions correctly.

The sense of pressure is tested tentatively with the patient's eyes closed by pressing on different body parts with the fingers. In doing so, the examinee is asked what he or she feels and where.

The ***sense of weight*** is checked with weights or items of different weights, and the patient should be able to feel the difference in weight [4, p.11].

Complex sensitivity examination

(also performed with the patient's eyes closed)

Two-dimensional-spatial sense is checked in the following way: the doctor figuratively draws different elementary figures, most often a circle or a cross, with a hammer or a finger on the skin of the patient and finds out what the patient feels and whether he can say what object the doctor has drawn.

Discrimination sensitivity

On the skin area of the examinee, the doctor applies two irritations simultaneously, distant from each other, while asking the patient how many irritations the patient feels: two or one. A Weber instrument or two sharp items may be used for this purpose. With the legs of the instrument spread apart, two touches are applied. The legs are then shifted until their touch is perceived as one. The obtained data is evaluated in millimeters and compared with the figures obtained when examining symmetrical healthy skin areas or with regular rates. The ability to discriminate is most developed (up to 1 millimeter) on the tip of the tongue and on the skin of the "pads" of the fingers, the terminal phalanges of the fingers of the hands. At the same time, it is less developed (up to 4-6 centimeters) on the skin of the back

Stereognosis

The examinee closes their eyes, and some familiar item is placed in their hands. They are asked to feel the item. The patient should identify and answer what is in their hand and what properties the item has (density, softness, and other characteristics).

Sense of localization - the ability to locate the site of irritation when the eyes are closed. The examinee is irritated (pricked or touched) on some skin area, then asked to point to the place of touch with a finger. Typically, healthy people have no problems with this (but errors of up to 1cm are possible) [7, p.16].

1.4. Kinds and types of sensory disorders

Kinds of sensory disorders

Anesthesia is the absence of all or certain types of sensitivity.

Depending on what type of sensitivity is lost, anesthesia can be tactile, pain (analgesia), temperature (thermoanesthesia), when the sense of localization is lost (topanesthesia), stereognostic sense (astereognosis). Loss of all types of sensitivity is called total anesthesia.

Hypesthesia is a decrease in sensitivity and the intensity of sensations due to an increase in perception threshold. There is a decrease in all or certain types of sensitivity.

Hyperesthesia is a condition with increased sensitivity to various types of irritation. Hyperesthesia is associated with lowering the excitability threshold when pain, temperature, and other receptors are irritated. In such cases, even minor irritations cause excessively strong sensations. Hyperesthesia is also possible in diseases of internal organs and can be reflected in the Zakharyin-Head's zones located in the back in the corresponding areas.

Hyperpathy is an increased perverted sensitivity (most often pain, temperature, less often tactile) with a change in the quality of sensation. Unpleasant sensations at high excitability threshold, impaired localization, and differentiation of sensation with prolonged aftereffects are characteristic. Even point stimuli are "scattered" or "blurred," and the qualitative distinction between stimuli is obliterated. Any sensation is unpleasantly marked with a painful tone, often accompanied by motor-affective reactions.

Paresthesia is a sense of "crawling goosebumps," numbness, burning, and tingling, occurring spontaneously without an irritant.

Polyesthesia is a disorder of perception of a single stimulus; it is perceived as multiple stimuli.

Dysesthesia is a perverted sensation of various stimuli, where pain is perceived as warmth, touch as cold, and others.

Synesthesia is when a stimulus in one sensory area leads to a response in another.

Allochaeria is a sensation of irritation applied in a symmetrical area on the other side.

Thermalgia is a perception of real cold or heat stimuli but with a painful component.

Dissociated disorder is the loss of one type of sensitivity while others are preserved (preferential dissociation between superficial and deep sensitivity).

Pain is "an unpleasant (sensory or emotional) sensation caused by irritation of sensitive neurons or pathologic changes due to existing or possible damage to body tissues or described in terms of such damage."

During neurological examination, it is necessary to assess the following characteristics of pain: localization, type, duration, intensity, character of pain (aching, pulling, tugging, and others), triggering factors, and concomitant autonomic, motor, and emotional disorders [8].

Kinds of pain sensations

Local pain - the sensation of pain in this type is localized in places corresponding to the localization of the pathological process. This type of pain appears in the area of an existing painful irritation.

Projection pain - localization of pain is located in the dermatome area corresponding to the radicular-segmental zone. For instance, when the L4 nerve root is compressed due to a herniated intervertebral disc, pain projects to the periphery along the outer surface of the shin in a "stripe" pattern, and when the S1 nerve root is compressed, it radiates to the back of the thigh and shin.

Irradiating pain may spread from the affected nerve branch to others not directly affected by the pathologic process. An example would be the irradiation of pain from one branch of the trigeminal nerve to other branches.

Reflected (reflex) pain occurs with diseases of internal organs. Pathologic input enters the posterior horn of the spinal cord from the receptors of internal organs, exciting the pain sensitivity conductors of the corresponding dermatomes. This is called the viscerosensory phenomenon, and the area of pain projection is called the Zakharyin-Head zone. It is not uncommon to observe hyperesthesia or hyperpathy in these areas. Examples are left shoulder blade and arm pain with angina.

Phantom pain - postamputation pain in the missing extremity (such pain occurs after amputation of an extremity as a result of neurinoma formation in the stump).

Causalgia is a burning attack-like pain, intensified by light touch or blowing air, extremely intense, excruciating, usually developing in partial damage to nerves rich in autonomic fibers (median, tibial).

Reactive pain occurs in response to compression or tension of nerves or roots. Such pain symptoms are called tension symptoms.

The ***Neri sign*** is a pain in the lower back upon forced head bending.

The ***Dejerine's sign*** is a pain in the lower back that occurs during coughing or sneezing and straining.

The ***Lasegue sign*** is a pain along the path of the sciatic nerve or in the lumbosacral area upon leg flexion at the hip joint. (In this test, the doctor raises the patient's leg straight up while the patient is lying on their back, and pain may occur at any level, even at an angle of 30 degrees. Then, the doctor bends the same leg at the knee joint, and the pain disappears.)

The ***Wasserman sign*** is a pain that occurs on the anterior surface of the thigh in a patient lying on their stomach when the doctor raises their extended leg.

The ***Matskevich sign*** is a pain that occurs on the anterior surface of the thigh or in the inguinal fold during passive or active leg flexion at the knee joint in a patient lying on their stomach.

Types of sensory disorders

Disorders or disturbances of sensitivity are associated with damage to the sensory tracts at any level, peripheral or central. The central level is divided into

spinal and cerebral, depending on the localization of the lesion in the spinal cord or brain.

There are three types of sensory disorders:

1). The peripheral type means that the sensory disorder occurred due to damage to peripheral nervous structures. This type of sensory disorder is defined as a variant of disorders arising from lesions of the sensory tracts within the peripheral nervous system (peripheral nerves, ganglia, roots, and plexuses). The peripheral type includes mononeuritic, polyneuritic, plexus, ganglion, symphatthalgic, and radicular syndromes.

Mononeuritic syndrome of sensory disorders occurs when a single sensitive or mixed nerve (the main trunk or its sensitive portion) is affected. It is manifested by sensory disturbances in the innervation area of one nerve, with the area of sensory disturbances less than the zone of its anatomical innervation due to cross-innervation of the skin by neighboring nerves. The initial signs of the affection are more likely to be symptoms of irritation (pain, paresthesias, hyperpathy, nerve trunk tenderness on palpation, positive tension symptoms, painful nerve exit points) and/or symptoms of prolapse (anesthesia, hypoesthesia, and others).

Polyneuritic syndrome of sensory disorders occurs due to lesions mainly in the distal parts of peripheral nerves and, accordingly, is characterized by localization of sensory disorders in the distal parts of the extremities in the "gloves and socks" or "high gloves and high golf" type. The degree of sensory impairment is predominant in the fingers of the extremities and attenuates proximally [9,10].

The ***radicular syndrome*** of sensory disorders occurs when the roots are affected, characterized by a violation of all types of sensitivity and radicular pain in the corresponding zones in the form of bands (transverse on the trunk, longitudinal on the extremities). The clinical picture of a spinal root affection is characterized by pain and paresthesia in the area of the corresponding dermatome. Loss of sensitivity in the form of hypoesthesia (less often anesthesia) is often found not in the entire area innervated by the affected root but only in the distal part of the extremity due to cross-innervation of the skin by neighboring roots.

The ***ganglionic syndrome*** occurs when the spinal node is affected. It is characterized by pain, hypoesthesia, or hyperesthesia in the zone of innervation of nerves emanating from the affected root, often accompanied by herpetic skin rashes in the corresponding dermatome.

Sympathalgia syndrome occurs due to the sympathetic ganglia affection. It is characterized by sharp irradiating pain, causalgia, vasomotor, and trophic disorders in the zone of disturbed innervation.

2). The spinal type of sensory disorders is caused by damage to sensory structures in the spinal cord. This type includes posterior root segmental, commissural-segmental, conductor, posterior column, and Brown-Séquard syndrome.

The ***posterior horn segmental*** (dissociated) syndrome manifests as a loss of superficial sensitivity in the area corresponding to the dermatomes in a "jacket" distribution (in two-sided involvement) or a "half-jacket" distribution (in one-sided involvement on the affected side). This type indicates damage to the posterior horns

or anterior gray matter at the level of corresponding segments of the spinal cord. The affection of the posterior horn is characterized by pain and temperature sensitivity disorders in the area of the corresponding dermatomes with preservation of deep and essentially tactile sensitivity - dissociated sensitivity disorder. Segmental types of sensory disorders typically have upper and lower boundaries. This variant of sensory disorders is seen in syringomyelia or other etiologies.

The ***commissural-segmental syndrome*** is observed when the anterior gray commissure, where the crossroads of the path of superficial sensitivity are located, is affected, so only superficial sensitivity is lost. In contrast, deep sensitivity is preserved (dissociation). The sensory loss area is two-sided, depending on which segments are affected. So-called "jacket" or "belt" syndrome may be observed.

Conductor spinal syndrome is manifested by sensory impairment distal to the horizontal level of the spinal cord lesion. The conductor part appears blocked at some level, and impulses are not transmitted upwards from that level.

Loss of all types of sensitivity below the lesion focus indicates a complete cross-sectional spinal cord affection. Thus, the detection of loss of all types of sensitivity below the umbilicus on the trunk and in the legs means a transverse affection of the spinal cord at the level of the Th10 segment. The presumed affection level reference points are the trunk's conditional lines. Thus, if all types of sensitivity are lost below this line, it is possible to assume tentatively the level of the spinal cord that has been affected by one or another cause.

The chin line corresponds to level C1, the collarbone line - C5, the nipple line - Th4, the navel line - Th10, the inguinal fold line - L1.

If only the posterior column on one side is affected, there is a loss of deep sensitivity on the affected side below the affection level.

Posterior column syndrome - loss of deep sensitivity on one side indicates a lesion of the posterior column on the same side. In affections of the posterior canaliculi, there are disorders of deep sensitivity below the level of the lesion with preservation of superficial sensitivity, as well as sensory ataxia.

In this type of sensory disorder, movements become disproportionate and inaccurate, and when walking, the patient excessively extends and throws his legs forward ("stamping gait").

The ***spinothalamic syndrome*** is formed if the lesion covers only the lateral spinothalamic tract on one side. There is a loss of superficial sensitivity below the level of the lesion 1-2 segments lower on the opposite side because of decussation in the anterior gray commissure.

Affection of half of the spinal cord (Brown-Séquard syndrome) is characterized by loss of deep sensitivity on the affected side and disruption of superficial sensitivity on the opposite side below the affection level (motor disorders are also observed on the affected side).

The ***law of the eccentric location*** of long conductors is essential for the topical diagnosis of spinal tumors. Thus, in extramedullary tumors, conductor disorders of superficial sensitivity start from the distal parts of the legs in an ascending type,

increasing from bottom to top, and in intramedullary tumors - from top to bottom in a descending variant.

3). The cerebral type of sensory disorders occurs when the sensory tracts in the brain are affected. The pathological process may involve the nuclei of sensory-related cranial nerves located in the brain stem (altering stem syndrome), the optic tubercle - the collector of all types of sensitivity (thalamic syndrome), the internal capsule (capsular syndrome), and the cerebral cortex (cortical syndrome).

A distinctive characteristic of conductor cerebral sensory disorders is their localization on the side of the body opposite to the lesion [11].

Alternating stem syndrome develops in brain stem lesions, characterized by alternating hemianesthesia, which implies loss of pain and temperature sensitivity on the face on one side and half of the trunk and extremities - on the side opposite to the pathological focus. This variant of sensory disorder indicates an affection in the caudal parts of the brainstem, where the long sensitive nucleus of the trigeminal spinal tract of the trigeminal nerve is localized, and the spinothalamic tract runs there.

Thalamic syndrome develops when the thalamus is affected, characterized by hemianesthesia on the opposite side of the lesion with loss of all types of sensitivity, hyperpathy, and hemianopsia.

The ***capsular syndrome*** is observed when there is a pathologic process in the internal capsule, manifested by opposite hemianesthesia, hemiparesis, and hemianopsia (three hemi- syndrome).

The ***cortical syndrome*** develops when the posterior central gyrus is affected, manifested by contralateral hemianesthesia.

However, in partial affections of the posterior central gyrus, sensitivity is lost in the corresponding area of the lesion, so if the upper part of the posterior central gyrus is affected - then hypesthesia is in the opposite leg, the middle part - in the arm, the lower part - in the face. That is, in an inverted form.

The ***sensitive variant of Jacksonian epilepsy*** develops with irritation of the postcentral gyrus. It is characterized by attacks of paresthesias on the opposite half of the body and the area corresponding to the irritated cortical centers [12].

(Diagram 1).

Questions on the topic “Sensory area”:

1. Structure of the deep and superficial sensitivity pathways.
2. What kinds of sensory disorders do you know?
3. What types of sensory disorders exist?

Anatomy	Research technique	Lesion symptoms	Topical diagnosis
Superficial sensitivity 1st order neuron ➢ Cerebrospinal node 2d order neuron ➢ Dorsal horn ➢ a cross in the anterior gray commissure ➢ spinothalamic pathway 3 neuron: ➢ optic tubercle Cortical end posterior central gyrus	- **Examine the surface sensitivity:** - pain - temperature - tactile	hypoesthesia hyperesthesia anesthesia paraesthesia pains synaesthesia	Defeat on any level
		herpetic rashes pains	Cerebrospinal node
		dissociative disorder with loss of superficial sensation	Dorsal horn or the anterior gray commissure
Deep sensitivity 1st order neuron ➢ Cerebrospinal node Through the dorsal collumn: Gol's bundle from the legs, Burdach's bundle from the arms 2d order neuron: ➢ In the Medulla Oblangata 3 neuron: ➢ optic tubercle Cortical end posterior central gyrus	➢ **Examine the deep sensitivity:** - kinesthetic - musculo-articular - pressure - weights - vibrations	dissociative disorder with loss of deep sensation	Dorsal column
		hypoesthesia hyperpathy dysesthesia	thalamus capsule posterior central gyrus
	➢ **Examine the complex sensitivity:** - discriminative - two-dimensional - stereognostic - localizations	alternative stem syndrome	Medulla Oblangata
		paresthesias attacks	irritation of the posterior central gyrus

Diagram 1. Sensory area
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

2. MOTOR AREA

Movement is a phenomenon that ensures the quality of life; it is a product of the organism's vital activity, and through movement, the interaction with the environment is ensured. Generally, the body can respond to various stimuli with a motor act. There are involuntary and voluntary movements. Voluntary movements are distinguished by the ability to control the motor process; in fact, these are purposeful acts of motor behavior of a human being. Voluntary movements are realized due to the presence of the pyramidal system, which is a collection of motor neurons [13].

2.1. Anatomical data of the motor area

The pyramidal tract consists of two neurons: central and peripheral.

The first (central) motor neuron.

The bodies of the first neurons are located in layer 5 of the cerebral cortex, in the anterior central gyrus, in the posterior parts of the superior and middle frontal gyrus, and in the paracentral lobule. Two components are distinguished in the pyramidal tract: the corticomuscular and corticonuclear tract.

The corticomuscular or corticospinal tract (tr. Corticospinalis) is a tract from the cerebral cortex to the spinal cord and the executing muscles located peripherally on the trunk and extremities.

The corticonuclear tract (tr. Corticonuclearis) is much shorter in duration; it goes from the cerebral cortex to the nuclei of the cranial nerves and, accordingly, to the muscles innervated by the cranial nerves.

The bodies of the first neurons of the corticomuscular tract are located mainly in the upper and middle parts of the anterior central gyrus. The axons of the first neurons pass through the knee and posterior femur of the internal capsule, then pass through the entire brainstem and terminate at the motoneurons of the anterior horns of the spinal cord.

The bodies of the first neurons of the corticonuclear tract are located mainly in the lower part of the anterior central gyrus; their axons pass through the internal capsule and terminate in the brainstem at the motor nuclei of the cranial nerves. Above the nuclei, axons of the first neurons of the corticonuclear tract make a partial decussation (50% cross to the other side, 50% remain on their side); the only exceptions are the fibers above the lower part of the nucleus of the facial nerve and above the nucleus of the hyoid nerve, where a complete 100% of fibers cross.

As for the corticomuscular tract, between the medulla oblongata and the spinal cord, most (about 90%) of the fibers of the first neurons pass to the opposite side, forming a pyramidal decussation (decussatio pyramidorum). Further, the crossed fibers go to the lateral column of the spinal cord, located in its posteromedial part in the form of the lateral corticospinal tract (tr. Corticispinalis lateralis). This tract provides voluntary movements of both the extremities and trunk. The uncrossed part of the fibers (about 10%) remains on its side. It goes to the anterior tubules, forming

the anterior cortical-spinal tract (tr. Corticispinalis anterior) and providing voluntary movements mainly in the trunk and neck muscles.

The second peripheral motor neuron is located in the spinal cord, namely in the anterior horns of the spinal cord (corticospinal tract) and in the brainstem in the motor nuclei of the cranial nerves (corticonuclear tract). Axons of the second neurons reach the executor muscles via anterior roots, spinal nerves, plexuses, and peripheral or cranial nerves. [14].

2.2. Methodology for the examination of the motor area

The examination of the motor area involves many aspects related to movement. This includes external examination, peculiarities of posture and gait, and musculature; it is necessary to pay attention to the presence of atrophy or hypertrophy of muscles, measure the scope of muscles, palpation to determine their tension, configuration, and the presence of fascial jerks.

Scopes of active and passive movements

Active movements are tested in all movable joints, with the patient asked to produce the movements themselves. The patient may present with self-reported complaints of lack of or restricted movement or decreased scope of movement.

The doctor examines ***passive movements*** when the patient's muscles are completely relaxed. The scopes of passive hand movements are checked in the shoulder, elbow, and wrist joints (flexion, extension, pronation, supination), finger movements (flexion, extension, withdrawal, adduction), and opposition of the thumb to the little finger. Passive movements in the hip, knee, and ankle joints of the lower extremities (flexion and extension, inward and outward rotation), flexion and extension of the toes of the foot are examined. Attention is paid to contractures in the joints, swelling, and others

Muscle strength is tested in all muscle groups, with the patient actively resisting. For example, in a shoulder girdle muscle strength test, the examinee is asked to raise both arms above a horizontal line and hold them up as hard as he/she can while resisting the actions of a doctor trying to lower his/her arms.

Shoulder muscle strength: The patient is asked to bend the arm at the elbow, and the doctor tries to straighten it.

Forearm muscle strength: counteracting supinator-pronator actions.

Finger muscle strength: The patient is asked to make a "ring" of the thumb with each of the other fingers of the hand and hold firmly. The examiner attempts to open the examinee's fingers. Alternatively, the examinee holds the fingers in a closed "fist," preventing the doctor from opening or unclenching the "fist."

Hand strength can be tested with a dynamometer; the patient may be asked to resist bending and extending the hands.

The *strength of the pelvic girdle and thigh muscles* is tested by lifting, lowering, adduction, and abduction of the thigh, with the patient resisting the doctor.

The *strength of the lower leg muscles* is tested by asking to bend the foot while the doctor holds it unbent and vice versa.

The *strength of the toes of the foot* is tested by flexing and extending them, overcoming resistance.

Reduced strength and restricted movement are called paresis, and a complete lack of strength and movement is called plegia or muscle paralysis.

Muscle strength is rated on a five-point system:

5 points – excellent, 4 points – mild paresis, 3 points – moderate paresis, 2 points – marked paresis, 1 point – deep paresis, 0 points – total plegia or paralysis, complete lack of strength and active movements.

The **Barré's test** detects latent paresis, where the patient may not complain or feel a decreased strength.

The ***upper Barré's test*** is designed to detect paresis in the upper extremities; the patient is asked to raise their hands with closed eyes and, in the supination position, hold their hands in a horizontal plane in front of them for 30-60 seconds.

The ***lower Barré's test*** helps verify paresis in the lower extremities. The patient in the supine position raises the legs and keeps them bent at the knee and hip joints for 30-60 seconds. If there is paresis in one extremity, it begins to tremble with weakness, droop, or deviate from the set position compared to the healthy side.

Muscle tone is an involuntary, reflexive muscle tension that provides muscle preparation for performing a motor act. Examination of muscle tone is performed with the patient in a fully relaxed state using two methods:

- by palpating the muscles;
- by rhythmic passive movements in the joints of the extremities (flexion and extension, extension and abduction, supination and pronation).

Reflexes:

The functional unit of the activity of the nervous system is the ***reflex.***

A ***reflex*** is a response to the irritation of receptors in a particular reflexogenic zone, where the receptors are located, whether in the tendons of muscles, in the skin of a particular area of the body, or the mucous membrane. The body responds to stimuli with many simple and complex reflexes. Reflexes, according to the location of receptors, are divided into *superficial* (exteroreceptive) – skin and mucous membranes and *deep* (proprioceptive), such as reflexes to muscle stretching, tendon, periosteal, and joint reflexes.

Superficial reflexes are elicited by mild mucous membranes or skin irritation, such as the cornea, conjunctiva, mucosa of the soft palate, and pharynx. We will list the most important of these for diagnosis.

The ***conjunctival reflex*** – eyelid closure when medical cotton wool is gently touched to the conjunctiva.

The ***soft palate reflex*** – when the soft palate is touched with a spatula or teaspoon, the soft palate and uvula are raised.

The ***pharyngeal reflex*** – touching the pharyngeal wall with a spatula or teaspoon causes coughing or gagging.

Cutaneous reflexes include abdominal, plantar, cremasteric, and anal reflexes.

***Abdominal reflexes* (*upper, middle, lower*)** are elicited by stroke irritation with the handle of a neurological hammer in the corresponding cutaneous area in the supine position.

The upper reflex is caused by irritation of the abdominal skin below the edge of the rib arch, the middle reflex – at the level of the umbilicus, and the lower reflex – above the inguinal fold. This involves the contraction of the abdominal muscles at the corresponding level.

The ***plantar reflex*** is a flexion of the foot and toes upon stroke irritation of the outer edge of the sole.

The ***cremasteric*** *reflex* is a pulling of the testicle upward with a stroke irritation of something or a slight tingling of the inner surface of the thigh.

The ***anal reflex*** is a contraction of the external sphincter of the anus at a slight tingling of the skin near it.

Deep reflexes include periosteal and tendon reflexes, triggered by striking an area of periosteum or tendon muscle with a hammer.

The ***mandibular reflex*** is a masseter muscle contraction when a hammer is struck with a finger applied to the chin of a slightly ajar mouth.

The ***carpal-radial (carporadial) reflex*** is a slight flexion and pronation of the forearm, flexion of the fingers of the hand when hitting the styloid process of the radius with a hammer.

The ***flexor-elbow (bicipital) reflex*** is a flexion of the forearm in the elbow joint when hitting the tendon of the biceps brachii muscle with a hammer.

The ***extensor-elbow (tricipital) reflex*** is an extension of the forearm half-bent in the elbow joint when hitting the tendon of the triceps muscle of the shoulder with a hammer.

The ***knee reflex*** is an extension of the tibia when the hammer is struck on the patella ligament. It is examined in the position of the examinee, either supine or sitting. This involves striking the patella ligament with a hammer. In sitting, the patient is asked to spread and extend the legs slightly at the knee joints. Suppose the quadriceps femoris muscle contracts weakly or does not contract when the hammer is struck on the patella ligament. In that case, the patient is suggested to interlock the fingers of the hands in a “lock” and stretch them firmly to the sides (Jendrassik’s technique).

The ***plantar reflex*** is a flexion of all toes of the foot upon stroke irritation of the sole with a hammer handle or needle.

The ***Achilles reflex*** is a plantar flexion or dorsal extension of the foot when the Achilles tendon is struck with a hammer. The patient is asked to kneel on a chair with the feet dangling and the hands holding onto the back of the chair. When inducing the reflex in the supine position, the doctor takes the foot with the left hand, bends it at a right angle while slightly bending the leg at the knee joint, and strikes the Achilles tendon with a hammer. Examining reflexes helps assess the state of various parts of the nervous system, determining the nature, uniformity, asymmetry, and degree of expansion of reflexogenic zones [4, p.23].

2.3. Symptoms and syndromes of motor impairment

Types of muscle strength impairment:

Paresis is a decreased muscle strength and reduced scope of active movements.

Plegia or paralysis is a complete loss of muscle strength and active movement.

Depending on the localization of the development and spread of motor disorders, the following variants of paralysis and paresis are distinguished:

- ***Monoplegia/monoparesis*** is in one extremity;
- ***Hemiplegia/hemiparesis*** is on one side of the body, arm and leg (left or right);
- ***Paraplegia/paraparesis*** – only in the muscles of both arms or legs (respectively upper in both arms or lower in both legs);
- ***Tetraplegia/tetraparesis –*** in all four extremities;
- ***Triplegia/triparesis*** – in three extremities.

The degree of paresis is usually quantified using a five-point system:

5 points — movement in full force;
4 points — a slight decrease in strength;
3 points — a moderate decrease in strength;
2 points — a significant decrease in strength;
1 point — a marked decrease in strength and minimal movements or only visible muscle contraction without extremity movement;
0 points — complete lack of movement (paralysis or plegia)

A 4-point paresis is mild, a 3-point paresis is moderate, and a 1-2-point paresis is deep.

Types of muscle tone impairment:

Hypotonia is a decreased muscle tone when the muscle feels flabby, sluggish, and soft when palpated.

Atonia is a complete lack of muscle tone.

Hypertonia is an increase in muscle tone, where the muscle has a thicker consistency when palpated.

In passive movements, if the tone is reduced, the examiner notes lightness, absence, or reduction of muscle resistance; the extremity moves like “fluff” or “rag.”

With increased tone, passive movements are difficult to make, and strong resistance is felt, although the patient does not actively resist himself, being asked to be relaxed.

Hypertonia can be spastic and plastic.

Spastic hypertonia is an increased tone due to lesions of the pyramidal tract, characterized by a substantial increase in tone – rigidity in the initial stage of passive movements, and at the end of these movements – muscle resistance is sharply reduced; this phenomenon has been called “the symptom of a penknife or folding pocketknife.”

Plastic hypertonia increases muscle tone when the extrapyramidal (pallidonigral) system is affected. With repeated passive movements during the study, muscle tone increases, and the “cogwheel symptom” is noted (resistance is felt in the

form of jerky, intermittent movements resembling circular movements of a "cogwheel").

Types of reflex impairment:

Areflexia is the complete absence of reflexes.

Anisoreflexia – different reflexes in symmetrical areas.

Hyporeflexia is a decrease in reflexes.

Hyperreflexia – increase in reflexes with the expansion of reflexogenic zones.

Pathologic reflexes are reactions of certain muscle groups to a stimulus that should not usually be present.

Pathologic reflexes include *oral automatism* reflexes:

Proboscis reflex – contraction of the circular muscles of the mouth with stretching of the lips forward and as if "in a tube" at a light blow with a hammer on the upper or lower lip.

Nasolabial reflex – contraction of lip muscles when tapping the back of the nose with a hammer, accompanied by pulling the lips forward.

Sucking reflex – sucking movements of the lips when the lips are lightly stroked.

Marinescu-Radovici palm-chin reflex – contraction of the chin muscle at stroke irritation of the skin in the area of the elevation of the I finger on the palm surface.

Pathologic reflexes also include plantar and hand pathologic reflexes:

Plantar pathologic reflexes:

Depending on the response, they are categorized into extensor and flexor.

Extensor plantar reflexes:

The ***Babinski's sign*** is an extension of the I toe of the foot in response to irritation of the sole with a hammer handle, with the fan-shaped divergence of the other toes (in children up to 1-2 years old – this reflex is considered a physiological reflex).

The ***Oppenheim's sign*** is an extension of the I toe of the foot with sliding and pressing downward movement of the I and II fingers of the examiner along the anterior edge of the tibial crest to the ankle joint;

The ***Gordon's sign*** is an extension of the I toe of the foot and fan-shaped divergence of the other toes with compression of the calf muscle

The ***Schaeffer's sign*** is an extension of the first toe of the foot with Achilles tendon compression.

Flexor plantar reflexes:

The ***Rossolimo's sign*** is a flexion of the toes in response to short and quick strokes on the pads of the patient's toenail phalanges.

The ***Bekhterev sign*** is a flexion of the toes of the foot when hitting the dorsal surface of the foot with a hammer in the area of III-IV metatarsal bones;

The ***Zhukovsky's sign*** is plantar flexion of the toes of the foot when striking the plantar surface of the foot with a hammer in the area of the metatarsophalangeal joints.

Carpal reflexes:

The ***Rossolimo's carpal sign*** is a flexion of the fingers of the hand, which is in the pronated state in response to rapid and rhythmic tapping on the pads of the patient's fingers with the fingers of the doctor;

The ***Jacobson-Lask sign*** is a palm flexion of the fingers of the hand in response to a hammer blow on the styloid process of the radius;

The ***Zhukovsky's carpal sign*** is a flexion of the fingers of the hand when hitting the palm surface of the hand with a hammer in the area of III-V metacarpal bones.

Pathological reflexes also include **protective reflexes**, which can be elicited by pinching, pricking, or sharp flexion of the toes of the foot, occurring in paralyzed extremities.

An example of a protective reflex is the ***Marie-Foix-Bechterev sign***, which consists of involuntary flexion of the leg in the knee and hip joints in response to a sharp passive plantar flexion of the toes of the foot.

Synkinesias are involuntary co-occurring movements that accompany the performance of active movements. They are divided into physiologic (waving arms when walking) and pathologic.

Pathologic synkinesias:

Global synkinesias – involuntary movements on the paralyzed side that occur when performing any movements with healthy extremities (clenching the hand into a fist, lifting the hand, and others). The paralyzed arm is flexed at the elbow joint and brought to the trunk, the forearm is pronated, the hand is flexed, and the leg is extended and brought to the midline.

Coordinator synkinesias – when a paretic extremity attempts to make a movement, another movement involuntarily appears. For example, extending both arms forward results in extension, spreading the fingers of the paretic extremity. When trying to bend the lower leg, overcoming the doctor's resistance to pressing the knee joint with his hand, the foot and toe of the paretic leg involuntarily extend.

Imitation synkinesias – involuntary repetition in a paretic extremity of movements performed in a healthy extremity of the other side of the body. Another manifestation of imitation synkinesias may be involuntary movements in the paretic arm that occur when the arm is extended forward and when the healthy arm is pronated. When the healthy leg is withdrawn and brought in, overcoming the doctor's resistance causes the same involuntary movements in the paralyzed leg.

Central and peripheral paralysis/paresis

Depending on the localization of the pathological process in the central or peripheral motor neurons, two primary syndromes of pyramidal tract lesions are distinguished: central and peripheral paralysis or paresis.

Central paralysis/paresis occurs when a central motor neuron is affected at any of its sites or levels: the motor area of the cerebral cortex – anterior central gyrus, the internal capsule, the brainstem, and the spinal cord.

Symptoms of central paralysis:

- spastic hypertonia
- hyperreflexia and allodynia
- decreased cutaneous reflexes;
- pathologic reflexes with clonus;
- protective reflexes;
- pathologic synkinesias.

Peripheral (flaccid) paralysis or paresis occurs when peripheral motor neurons (cells of the anterior horns of the spinal cord, motor nuclei of the cranial nerves, motor branches of the cranial nerves, anterior roots of the spinal cord, plexuses, peripheral nerve) are affected.

Symptoms of peripheral paresis or paralysis:

- hypo- or atonia of the muscles;
- hypo- or areflexia;
- hypo- or atrophy of the muscles;
- Fasciculations or fibrillations – involuntary contractions of individual parts of a muscle in the absence of its overall contraction.
- EMG excitability changes [11, p.36]

Topical diagnosis in motor disorders

Depending on the affection level, affection of the pyramidal tract is manifested by pareses and paralyzes of different characters and muscle groups.

The **peripheral level** gives paresis on the affected side:

- When ***one nerve*** is affected – peripheral paresis and paralysis are observed in the muscles innervated by this nerve.
- When ***multiple nerves*** in the distal arms and legs are affected, peripheral paresis or plegia of muscles in the distal arms and legs – peripheral tetraparesis occurs.
- If ***one plexus*** is affected, peripheral paresis of the muscles innervated by that plexus will occur (for example, muscles in one extremity – peripheral mono paresis).
- In the case of ***anterior root*** affection, peripheral paresis of muscles is innervated by this root.

At the level of the spinal cord:

- Affection of the ***anterior horns*** in the spinal cord, also manifested by peripheral paresis of those muscles, which are provided by this anterior horn, on the side of the lesion respectively segments.
- Affection of the ***lateral columns*** of the spinal cord causes central paresis on the side of the affection below the level of the affection.
- A transverse spinal cord lesion at the level of the ***lumbar thickening*** will result in peripheral inferior paraparesis.
- Transverse affection in the spinal cord at the ***thoracic level*** – lower central paraparesis.

- Transverse affection of the spinal cord at the level of the ***cervical thickening*** mixed tetraparesis: peripheral paraparesis in the arms, central paraparesis in the legs
- Transverse affection of the spinal cord at the level of the ***upper cervical region*** – central tetraparesis.

At the level of the brain:

- A unilateral ***brainstem*** affection leads to alternating syndromes, in which on the side of the lesion develops a picture of peripheral paresis of cranial nerves, and on the opposite side is central hemiparesis.
- ***Internal capsule*** – central hemiparesis, hemihypesthesia, and hemianopsia (syndrome of 3 hemi-), central paresis of the 7^{th} and 12^{th} pairs of cranial nerves develop on the opposite side from the lesion.
- ***Anterior central gyrus*** – on the opposite side – central hemiparesis, in case of affections of the upper part – lower central monoparesis, middle part – upper central monoparesis, lower part – central paresis of the 7^{th} and 12^{th} pairs of cranial nerves.
- ***When the anterior central gyrus is irritated*** – clonic seizures on the opposite side, which are called Gagarin-Jackson cortical epilepsy, can start either in the leg and go upward or in the face and go downward, depending on the direction of irritation along the anterior central gyrus: in the first case – from the top downward, in the second – from the bottom upward [14,15].
  (Diagram 2).

Questions on the topic "Motor area":

1. Cortico-muscular and cortico-nuclear pathways.
2. Methodology for studying the motor system.
3. What types of paralysis and paresis exist? Describe them.

Anatomy	Research technique	Lesion symptoms	Topical diagnosis
Corticomuscular tract **1st order neuron** ➢ Betz cells in the anterior central gyrus ➢ Crossing (90%) between the medulla oblongata and the spinal cord **2d order neuron** ➢ Anterior horns of the spinal cord. ➢ For arm muscles in the cervical thickening C5-Th1 ➢ For leg muscles in the lumbar thickening L1-L5	➢ **Volume of active movement** ➢ **Patient's posture and gait** ➢ **Test reflexes:** ➢ **Deep reflexes:** - **Tendon:** flexor-elbow, extensor-elbow, knee and Achilles - **Periosteal:** carpo- radial, costo- abdominal - **Superficial:** abdominal, plantar, cremasteric.	Decreased strength (1 – 4) paresis No strength (0 points) plegia or paralysis **Central paresis:** Hyperreflexia Hypertension Hypertrophy Pathologic extensor reflexes: Babinski, Gordon, Grossman, Shephard, Oppenheim. Flexor reflexes: Rossolimo, Zhukovsky, Bekhterev. Pathologic synkinesias, clonus of the feet and patellae Peripheral paresis Hypo-areflexia Hypo-atonia Hypo-atrophy Fibrillary twitches ENMG changes	lesion of the 1st central neuron 2nd peripheral neuron
Cortico-nuclear tract 1st order neuron ➢ Lower section of the anterior central gyrus of the cerebral cortex in the brainstem makes a partial crossing over the nuclei; besides the VIIth and XIIth pairs of cranial nerves 2d order neuron: ➢ the brainstem nuclei innervate the cranial nerves	➢ **To examine muscle tone:** -passive movements - to the touch ➢ **To examine muscle strength:** -on a 5-point system -Barre's upper and lower extremities ➢ **To examine pathologic reflexes** ➢ **To examine foot and kneecap clonus** ➢ **To examine muscle trophics** ➢ **Electroneuromyography** ➢ **Examine the function of cranial nerves**	Central hemiparesis - 1st neuron up to the crossover On the other side Alternating syndrome-- brain stem on one side. Central tetraparesis - upper cervical spinal cord Mixed tetraparesis - cervical spinal cord thickening Central inferior paraparesis - thoracic spinal cord Peripheral inferior paraparesis - lumbar thickening Peripheral tetraparesis - Nerves in the extremities	

Diagram 2. Pyramidal system
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

3. EXTRAPYRAMIDAL SYSTEM AND CEREBELLUM

3.1. Anatomical data of the extrapyramidal system

The **extrapyramidal system** consists of extrapyramidal subcortical and stem formations that, along with the pyramidal system, participate in the motor act.

The motor act requires the participation of all motor systems, all structures of the nervous system involved in movement, between which there are collateral connections, and each has its immediate task.

The extrapyramidal system ensures the readiness of muscles for a motor act, the sequence of activation of certain muscle groups, muscle tone, auxiliary movements, speed, rhythmicity, smoothness and flexibility, and others.

The extrapyramidal system includes the so-called basal ganglia or subcortical nuclei. The basal ganglia are a group of nuclei located in the deep parts of the brain and the upper part of the brain stem. They include the **striatum**, consisting of the caudate nucleus and shell, the **pallidum** - the globus pallidus, the substantia nigra, and the subthalamic nucleus. The shell and globus pallidus form the lenticular nucleus.

The extrapyramidal system also includes the reticular formation, thalamus, hypothalamic nuclei, nuclei of the covering of the stem, vestibular nuclei, inferior olive, red nucleus, and cerebellum. Of the cortical areas, the premotor area (field 6) can be referred to.

The extrapyramidal system participates in regulating a complex motor act due to the inclusion of individual formations of the extrapyramidal system in a system of closed neuronal circles, which operate on the principle of neural feedback.

There is also a multistage motor tract from the cerebral cortex to the red nuclei and nuclei of the reticular formation and then into the spinal cord to the motoneurons of the anterior horns (Monakov's rubrospinal tract and reticulospinal tract).

Within the basal ganglia, three levels are distinguished. The first includes two main tracts from the striatum: an indirect tract (striatopallidal) to the outer segment of the globus pallidus and a direct tract (striatonigral) to the substantia nigra and the inner segment of the globus pallidus. The second level includes tracts from the cortex (layer 5) to the striosomes and the striatum matrix. The third level is cortical projections to other sections of the striatum.

Dopamine deficiency with neuronal death in the substantia nigra results in impaired function of the extrapyramidal system, which consists of decreased activity of the striatonigral tract and increased activity of the striatopallidal tract, which eventually leads to impoverished and slowed movements - hypokinesia.

Increased movements- hyperkinetic syndrome- develop with excessive activation of dopamine receptors in the extrapyramidal system [16,17].

3.2. Methods of examination of the extrapyramidal system

The diagnostic algorithm for detecting extrapyramidal disorders involves observing the patient at rest and performing various motor activities, including walking.

During observation, involuntary forced movements or involuntary pathological posture at rest may be detected, and on analyzing movements, excessive movements or slowing down and impoverishment of motor acts may be found.

Attention is paid to the localization of motor disorders, character, symmetry, and manifestation at rest and when performing various movements.

In addition, the expressiveness of speech and facial expressions, writing patterns, tonic postural reflexes, and muscle tone are examined.

The **statics and gait examination** is performed by asking the patient to stand up and stand still for 15-20 seconds, then walk around the room with eyes open.

Writing examination - asking the patient to write something.

Examination of tonic postural reflexes:

Foot (Westphal's) phenomenon - when the doctor extends the foot of a patient lying on the back, the foot does not return to the initial position; it seems to freeze and remains in a "frozen" state;

Tibia (Foix-Thevenard) phenomenon - when a patient lying on the stomach bends the leg in the knee joint, bends the leg to the thigh, and releases, the leg remains in this position. It does not return to the original position.

Stewart-Holmes, **lack of pushback sign** - the patient is asked to resist the doctor's actions when attempting to extend the patient's arm in a flexed position and suddenly stopping the attempt, the arm may forcefully strike the patient in the chest (whereas usually, the strike is prevented by the action of antagonists).

Eye and tongue sign - the patient has difficulty holding a prolonged position with eyes closed and tongue out of the mouth at the same time

Examination of muscle tone using passive movements in the extremities: flexion and extension in large joints [18].

3.3. Symptoms and syndromes of extrapyramidal system affections

The primary syndromes of the extrapyramidal system are two syndromes: akinetic-rigid and hyperkinetic syndromes.

1). Akinetic-rigid syndrome

The **hypokinetic-hypertensive or Parkinsonian syndrome** is seen in affections of the pallidonigral complex, primarily the substantia nigra, with dopamine deficiency and consists of the following symptoms:

Plastic hypertonus is an increase in muscle tone of the plastic type (extrapyramidal rigidity, plastic muscle hypertension) - an increase in muscle tone detected simultaneously in the flexor and extensor muscles.

The **"cogwheel" symptom** is characterized by the sensation of uniform intermittent resistance in the muscles during passive movements of the extremities (flexion or extension at the joints) against a background of plastic hypertonia.

Hypokinesia or akinesia is manifested as poor movement (**oligokinesia**), absent movement, or slowing (**bradykinesia**) of voluntary movements.

The **"flexor posture"** is a characteristic posture of the patient - half-bent and slightly bent forward trunk, half-bent arms, and legs ("petitioner's position").

Difficulties in overcoming the inertia of rest and inertia of movement lead to difficulties in the transition from rest to movement and vice versa: a tendency to freeze in a given pose ("wax doll pose"), the ability to lie in bed with the head raised above the pillow (a symptom of "air pillow"), it is difficult to start the motor act of "stomping in place."

Asynkinesia - weakening or disappearance of joint movements); when walking, the patient does not wave their hands (achairokinesis).

Gait disorders - the patient moves in small, slow shuffling steps ("shuffling" gait);

Postural instability - manifested by difficulty maintaining a static posture when trying to change the direction of movement; when turning, the patient loses balance.

Propulsion - if a walking patient is lightly pushed forward in the back, they start moving forward faster and faster, cannot stop, and even fall.

Retropulsion - if a walking patient is pushed backward in the chest, they begin to move backward faster and may fall.

Lateropulsion - if a patient is pushed to the side, a non-stop involuntary sideways movement is observed.

Paradoxical kinesias - patients who are immobile during the day but may climb stairs, jump, dance, and others, during affective outbursts and emotional stress.

Bradyllalia - the speech is slow, monotonous, quiet, and tends to fade; repetition of same words.

Micrographia - handwriting is small, with irregular lines.

Hypomimia and amimia - depleted facial expressions or absence of facial expressions

Resting tremor - shaking of the distal extremities, especially the hands, resembling "coin counting" or "pill-rolling," which disappears in sleep and increases after emotional stress.

Autonomic disorders - oily face, flaking skin, increased salivation (hypersalivation), increased sweating (hyperhidrosis), orthostatic hypotension, constipation.

Changes in the psycho-emotional sphere - slowing of the pace of thought processes (bradyphrenia), depression, apathy, anxiety, dysphoria, lack of initiative, lethargy.

2). Hyperkinetic-hypotonic syndrome

Clinical manifestations of hyperkinetic-hypotonic syndrome arising from the lesion of the striatal system are characterized by **hypotension of muscles** with various involuntary movements and the appearance of **hyperkineses** - involuntary violent movements that increase with excitement and stress and disappear in sleep.

In examining hyperkinesis, attention is paid to the side, rhythm, character, shape, symmetry, and localization of movements.

The most typical of these are the following types of hyperkineses:

Chorea occurs in the genetic disorder Huntington's chorea, in rheumatic brain disease in children more often (rheumatic chorea), in encephalitis, brain trauma, cerebral atherosclerosis, degenerative changes of the basal nuclei, and in pregnancy (chorea of pregnancy).

Chorea presents as involuntary, continuously occurring rapid, jerky, irregular arrhythmic movements in different body parts, which may flow from one part to another. They have a large amplitude and may resemble voluntary movements, gestures, and grimaces. Patients buckle up, laugh at themselves, and their tongue sometimes pops out of their mouths. They cannot keep their mouths closed for short periods. Patients can only partially and briefly suppress the hyperkinesias; sometimes, they try to incorporate these movements into their voluntary actions (parakinesia). The gait in these patients resembles a dulcet, a "dancing gait."

Athetosis is characterized by slow tonic muscle contractions, pretension, and worm-like symptoms, predominantly in the distal extremities and less frequently in the muscles of the face and trunk. Straightened fingers bend alternately slowly and quickly in the metacarpophalangeal joints. The hand takes on a fancy shape at this time [11, p.44].

Torsion dystonia is characterized by persistent simultaneous contraction of agonist and antagonist muscles and repetitive twisting movements with the formation of pathological postures. When walking, the trunk and extremities make abrupt corkscrew-shaped movements resembling a body rotation around a long axis.

Hemiballism is characterized by rapid large-scale hyperkinesis resembling throwing or pushing a ball on one side.

Paraballism is characterized by rapid large-scale hyperkinesis resembling throwing or pushing a ball from both sides.

Myoclonus is characterized by jerky, sudden, brief muscle twitches resembling the twitching of an electric shock.

Tics are rapid, monotonous, stereotypically repeated twitches of certain muscle groups that mimic arbitrary movements, giving the impression of being "deliberate." Facial tics are accompanied by rapid forehead furrowing, blinking, raising eyebrows, twitching the corner of the mouth, and others

Facial hemispasm - periodic tonic-clonic twitches of mimic muscles with a predominance of tonic phase (spasms of eyes, mouth, circular muscles of frontal muscles). Facial hemispasm is characterized by muscle spasms that narrow the eye fissure and shift the corner of the mouth on one side.e [18,19]. (Diagram 3)

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
Pallidonigral system ➢ Substantia nigra ➢ Pale globe **Striatum** ➢ The caudate nucleus ➢ Putamen Thalamus The nucleus subthalamicus (corpus Luysii) The red nucleus The reticular formation The vestibular nuclear Cerebellum The inferior olivary nucleus Cerebral cortex The closed-loop system in neuroscience	➢ Evaluate posture, stance and gait ➢ Muscle tone test ➢ Tonic postural reflexes: - The Westphalian phenomenon - The phenomenon of Foix Thevenard ➢ To examine the handwriting ➢ speech analyze ➢ The "eye and tongue" symptom	**Akinetic - rigid syndrome:** A-hypo-kinesia Bradykinesia Amimia Bradylalia Plastic tone of the "cogwheel" type Resting tremor Pro lateroretropulsion Postural instability Micrographia **Hyperkinetic syndrome:** Hyperkinesia Hypertension Chorea Torsion dystonia Hemiballism Paraballism Athetosis, tics Myoclonia Localized spasm	Pallido-nigral complex Striar system

Diagram 3. Extrapyramidal system
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

3.4. Anatomical data of the cerebellum

Cerebellum - meaning "small brain" in translation, is the part of the brain located in the posterior cranial fossa between the rhomboid brain and the occipital lobes of the cerebral hemispheres. In the middle of the cerebellum is a prominent structure called the vermis, and on either side are the cerebellar hemispheres. The cerebellum consists of gray and white matter; the gray matter comprises the cerebellar cortex and subcortical paired nuclei: dentate, emboliform, globular, and roof nucleus. The cortex consists of three layers: a layer of molecular cells, a second layer of Purkinje ganglion cells, and a third layer of granular cells.

Transverse sulci divide the cerebellum into three principal lobes. The main transverse sulcus is located on the upper surface of the cerebellum and divides it into anterior and posterior lobes. The posterolateral sulcus, located on the inferior surface of the cerebellum, separates the small flocculonodular lobe from the large posterior lobe.

The cerebellum presents a definite somatotopic localization: the arm in the anterior hemispheres and the leg in the posterior hemispheres. In the medial part of the hemispheres are the proximal extremities, and in the labral part are the distal extremities. The upper part of the vermis is for the head and neck, while the lower part is for the trunk.

The cerebellum has three pairs of pedicles: upper, middle, and lower. These pedicles go to different structures: the upper ones to the quadrigeminal plate, the middle to the pons, and the lower ones to the medulla oblongata. The cerebellum communicates via the pedicles with the cerebral cortex, brain stem, and spinal cord.

Two spinal-cerebellar afferent pathways begin with the first neurons in the spinal ganglia and the second in the posterior horn. Further, these pathways are divided into the dorsal Flechsig's and ventral Gowers' tracts. The Flechsig's tract does not make decussations; it runs along its side along the dorsal portions of the lateral columns and ascends upward, reaches the medulla oblongata, and terminates in the cerebellar vermis. The Gowers' tract has two decussations: from the posterior horn, it passes through the commissure to the other side and ascends to the brainstem in the anterior aspect of the lateral columns, returns to the original side in the anterior cerebral sulcus in the midbrain, and with the upper cerebellar pedicles it reaches the cerebellar vermis. Because of this, the cerebellum's connection to the periphery is direct, not crossed, unlike the pyramidal tract.

The main efferent tract of the cerebellum is the dentorubrospinal tract. It starts from the dentate nuclei, reaches the red nuclei through the upper cerebellar pedicles, makes a Werneking decussation in the anterior cerebral pars, from the red nuclei, descends Monakov's rubrospinal tract with a Forel's decussation, and ends in the anterior horns of the spinal cord.

The cerebellar cortex and cerebellum connections are made via bi-neuronal pathways. The frontal-pontine-cerebellar pathway begins in the anterior pole of the frontal lobe, where the first neuron is located, the axon of which passes through the anterior femur of the internal capsule, ends in the pons, where the second neuron is located, which makes a decussation and reaches the cortex of the cerebellar hemispheres through the middle pedicles of the cerebellum. The connections to the temporal and occipital lobes of the large brain with the cerebellum have a similar structure.

The cerebellum regulates the automatic coordination of movements. It provides muscle tone, body balance, stabilization of the center of gravity, coordination of agonist and antagonist muscles, and accuracy and proportionality of movements.

The cerebellar vermis provides static (standing), and the hemispheres provide dynamic (extremity movements, walking) coordination [6, p.143].

3.5. Methodology for the examination of cerebellar functions

Finger-nose test. The patient is asked, with eyes open first and then closed, to hit the tip of their nose with their index finger and then with the other. In cerebellar affections, there is a slippage, sometimes combined with intentional tremors of the hand and index finger, the severity of which increases as the finger approaches the nose.

Heel-knee test. The patient is asked to lie down and, with eyes open first and then closed, lift one leg, and touch the knee of the other leg with the heel of the raised leg, and then run this heel along the front surface of the shin (as if along the crest of

the tibia) to the ankle joint and back up to the knee. The active leg has excessive movement on the side of the affected cerebellar hemisphere, and the heel shifts from side to side.

The past-pointing test. The patient is asked to hit the neurological hammer with the index finger with eyes open, and the position of the hammer is changed. On the side of the cerebellar affection, there is "past-pointing."

Stewart-Holmes no pushback test - the patient is asked to bend their arm at the elbow joint, suggesting they resist the doctor trying to straighten their arm. If the doctor suddenly stops their action and releases the patient's hand, the patient's hand hits his chest with force, in cerebellar lesions on the focal side.

Babinski asynergy test. A patient lying on their back is asked to sit with their arms crossed over their chest. If the cerebellum is affected, the patient has difficulty performing this movement; it becomes incongruous, and both legs may be lifted first (if the worm is affected), or one leg may be lifted first (if the cerebellar hemisphere on the affected side is affected).

Romberg test (simple). The patient is asked to stand up, feet tightly together, eyes closed, arms stretched forward. When the vermis is affected, the patient staggers and falls forward or backward; when the hemispheres are affected, the patient falls toward the affected hemisphere.

The **complicated Romberg test** is used when the simple test is in doubt to detect mild statistical ataxia. The patient is asked to stand with one foot in front of the other, the toe of one foot touching the heel of the other, with the feet in a line one behind the other. Standing on toes or one foot can also be suggested, tilting the head in different directions. The stability assessment is the same as for the simple test.

Diadochokinesis test. The patient is asked to quickly extend their arms to pronate and supinate simultaneously on both sides. If the cerebellar hemispheres are affected, the rotation of these movements will be slower on the affected side (adiadochokinesis).

Gait examination. The patient is asked to walk forward and backward (in a single line) or sideways ("flank" side-to-side gait) with eyes open and closed. When the cerebellum is affected, the patient walks wobbly, with legs spread wide ("drunken gait"). The patient staggers or leans to the affected side when the cerebellar hemisphere is affected.

Nystagmus examination. The examinee is asked to follow the hammer sideways, up and down with their eyes (without turning their head, the head should be in a straight fixed position). When the cerebellar hemisphere is affected, nystagmus is determined, which is more pronounced when looking in the direction of the affection.

Speech examination. The patient is asked to repeat a few words or phrases challenging to pronounce; in cerebellar affections, speech becomes scanty, divided into syllables, and slowed down, as in a drunken person.

Handwriting examination. The patient is asked to write something, and a change in handwriting in the form of megalography, large letters, sprawling, uneven, sloppy, is noted.

Dysmetria test. The patient is asked to take an item from the table and return it to its place; in cerebellar affections, this is difficult.

When examining the functional status of the cerebellum, it should be remembered that cerebellar tracts are either not crossed or crossed twice. Consequently, lesions of the cerebellum and cerebellar tracts almost always manifest cerebellar abnormalities on the affected side (except affections of the red nucleus).

On the other hand, the cerebellar hemispheres are connected to the contralateral cerebellar hemispheres so that when the cerebellar hemispheres of the large brain are affected, functional disability of the cerebellar hemispheres begins with cerebellar symptoms on the opposite side [7, p.20].

3.6. Symptoms of cerebellar affection

The symptoms of cerebellar damage can be grouped into a syndrome called cerebellar ataxia. Ataxia is a disturbance of coordination and balance.

Cerebellar ataxia is manifested by impaired smoothness and proportionality of upper and lower extremities movements, impaired gait, and imbalance in standing and sitting.

Static ataxia means impaired ability to stand.

Dynamic (locomotor) ataxia is a disorder of coordination of extremities movements when performing actions requiring precision, coordination tests when walking.

Astasia (inability to stand) is manifested by the patient's inability to stand, falling backward or on their side.

Abasia (inability to walk) manifests as a wobbly, unsteady ("drunken gait"), with legs wide apart (wobble increases toward the affected hemisphere) with zigzag deviations from a straight line.

Dysmetria is a disorder of the amplitude of a motor act.

Hypermetria is an increase in the amplitude of the motor act.

Hypometria is a decrease in the amplitude of the motor act.

Babinski asynergy makes it challenging to change body position from horizontal to upright [6, p. 145].

Adiadochokinesis is the slow and unsynchronized execution of rapid alternating movements.

Muscle hypotension is a frequently observed phenomenon in cerebellar hemispheric lesions that occurs on the side of the affected hemisphere and is usually more pronounced in the upper extremities. Muscle hypotension can be detected with passive movements in the extremities. An excess of passive movements in the joints manifests it.

Intention tremor manifests as excessive deviations of the extremities from the programmed ideal trajectory of the target movement, increasing in severity as the target approaches.

Scanty speech - loss of fluency in speech, slowness, monotony, and separation of words by syllables.

Nystagmus - rhythmic twitching of the eyeballs, more pronounced when looking toward the affection. Depending on the plane in which the twitch is observed, a distinction is made between horizontal, vertical, multiple nystagmus, and others

Past-pointing - when performing the past-pointing coordination test, there is past-pointing.

Past-pointing during coordination tests, finger-nose and heel-knee tests.

3.7. Types of ataxia

Ataxia can be observed not only in cerebellar affections; there are other types of ataxia: sensory, vestibular, and cortical frontal.

Sensory ataxia occurs when structures related to deep sensitivity are affected. Suppose these structures are affected (for example, polyneuritis, lesions of the posterior roots, posterior columns of the spinal cord, thalamus). In that case, there is instability, which increases when the patient closes their eyes or in the dark. The loss of deep sensitivity results in the patient's need to control movements with vision. When walking, the examinee strongly bends the legs in the knee and hip joints and brings them sharply down to the floor ("stamping gait"). Such a patient constantly looks under his feet to compensate for the defect in joint-muscle sense by vision.

Vestibular ataxia - develops with affections of the vestibular nerve or its nuclei in the trunk and affections of the labyrinth. Ataxia is accompanied by systemic dizziness, nausea, vomiting, horizontal nystagmus, and bradycardia.

Frontal cortical ataxia is observed when the frontal lobe cortex is affected, in which there may be marked astasia, abasia, mental disorders, decreased criticism, flat jokes, and others[20] (Diagram 4)

Questions on the topic "Extrapyramidal system and cerebellum":

1. What structures are included in the extrapyramidal system?
2. What are the main syndromes of damage to the extrapyramidal system?
3. What types of ataxia do you know? Describe them.

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
Afferent pathways **1st neuron:** ➢ spinal node **2d neuron:** ➢ posterior horn of the spinal cord Flexig's tract is dorsal along the lateral column to the medulla oblongata and into the cerebellar cortex without crossings. Gowers' tract ventral makes a crossing in the anterior gray commissure, reaches the midbrain, then descends, makes a crossing, and terminates in the cerebellar cortex **Efferent pathways** The frontal-brain-bridge tract 1st neuron: ➢ Anterior pole of the frontal lobe 2d neuron: ➢ pons varolii ➢ crossing in the pons ➢ ends in the cerebellum Dento-rubro-spinal tract -1 neuron - the dentate nucleus - 2 neuron - red nucleus 2 crossing: Vernekinga, Forelya Hemispheres of the cerebellum Cerebellar worm Nuclei: Nucleus dentatus, Nucleus emboliformis, Nucleus globosus, Nucleus fastigii	➢ To examine gait ➢ To examine muscle tone ➢ To examine nystagmus ➢ To examine coordinator tests: - Palpation test - The heel-knee test - Miss test ➢ Romberg test simple and complicated ➢ Dysmetria test ➢ Diadochokinesis test ➢ To examine handwriting ➢ To examine speech ➢ The Stuart Holmes Test ➢ Babinski's assynergia test	Cerebellar ataxia: Dynamic } Lesion of the cerebellar hemisphere Static } Worm lesion Symptoms of ataxia: Unsteadiness in the Romberg pose Coordinator disturbances Dysmetria Adiadochokinesis Babinski's asynergy Megalography Scanty speech Nystagmus Intention tremor Types of ataxia: Sensory ataxia - posterior columns of the spinal cord Cortical frontal ataxia - frontal lobe Vestibular ataxia - 8th pair of cranial nerves	

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Diagram 4. Cerebellum

(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

4. CRANIAL NERVES: ANATOMICAL DATA, EXAMINATION TECHNIQUES, SYMPTOMS AND SYNDROMES OF AFFECTION

The anatomical structure of cranial nerves is very versatile and variable, has both afferent and efferent tracts, and levels of localization of both central and peripheral nature, so unambiguously attributing the pathology of cranial nerves to diseases of the peripheral nervous system is not always possible, as we often deal with the clinic of lesions of cranial nerves also in diseases of the central nervous system. The functional significance of cranial nerves is quite substantial, and it is practically impossible to overestimate their importance. Loss or hyperfunction of their function often results in significant disability for the individual. However, despite the complexity of the study of this section, with careful parsing of anatomical data, proper clinical examination, and the analysis of the identified symptoms and syndromes of the lesion, a clear connection of these syndromes with the topical localization of the lesion, this whole process becomes bright and exciting.

The numbering of the nerves is not arbitrary. If one observes the basal surface of the brain from front to back, starting from the frontal lobe to the end of the occipital lobe, one can see the sequential localization of the cranial nerves based on the brainstem.

Depending on the functional significance, a distinction is made between sensitive (I, II, VIII pairs), motor (III, IV, VI, XI, and XII), and mixed (V, VI, IX, X) cranial nerves.

Clinical examination of the cranial nerves may reveal symptoms and syndromes of affection of one nerve, two nerves, or even several nerves simultaneously. Thus, some nerves are located together in some localized sites. As a result of the affection of these sites of different origins, a clinical picture of the simultaneous involvement of these nerves in the pathological process develops [21].

4.1. Olfactory nerve

The **first pair** of cranial nerves is the olfactory nerve.
This nerve is afferent and sensitive and goes by the name of olfactory analyzer. This analyzer consists of three neurons.

The **first neuron** is the receptor bipolar neuron cells, which are located in the mucous membrane of the upper nasal shell and nasal septum; their axons are directed in the form of olfactory filaments through the pierced lamina of the lattice bone into the cranial cavity to the olfactory bulb.

The **second neuron** is the mitral and bundle cells in the olfactory bulbs, which form the olfactory tract that terminates in the primary olfactory centers (anterior perforating substance, olfactory triangle, amygdala complex, and transparent septum).

The **third neuron** is located in those primary olfactory centers mentioned above; axons partially decussate, circle the corpus callosum above and below, and end in the entorhinal cortex of the temporal lobe (anterior part of the parahippocampal gyrus and uncinate gyrus), as well as a part - in the dorsolateral nucleus of the thalamus and hypothalamus [22].

Methods of examining the functions of the olfactory nerve:

The olfactory analyzer is tested using aromatic substances that emit certain odors familiar to everyone. It can be aromatic oils, coffee, camphor, perfume, peppermint, vanilla, and others. Irritants such as vinegar or ammonia should be avoided. During the examination, the patient is asked to close their eyes and close one nostril; then, the doctor offers a vial with a scented substance near the open nostril, asking if the patient can smell and identify the substance. Then, similarly, the other nostril is tested.

Symptoms of olfactory nerve affection:

Hyposmia is a decrease in the sense of smell.
Anosmia is a complete *l*oss of the sense of smell.
Hyperosmia is hypersensitivity to odors.
Dysosmia is a perversion of the sense of smell.
Olfactory agnosia is a disorder in the identification of odors.
Olfactory hallucinations or olfactory aura before an epileptic seizure is the sensation of unreal odors that do not exist.

In the presence of the first two symptoms, the pathologic focus is usually localized in the nasal mucosa, olfactory bulbs, and olfactory trigone. Hyperosmia may occur in hysteria or drug and substance abuse. Mild to moderate dysosmia may be in the olfactory bulbs' lesions, nasopharynx processes, and paranasal sinuses' empyema. In contrast, marked dysosmia may be in hysteria, schizophrenia, depression, psychosis, or lesions of the parahippocampal gyrus. Olfactory agnosia occurs when the cortical end of the olfactory analyzer, the parahippocampal gyrus, and the uncinate gyrus are affected. Olfactory hallucinations or aura before an epileptic seizure indicate a high central level of affection of the olfactory analyzer. When the temporal lobe is irritated by any pathologic process of an organic nature. Olfactory hallucinations may also occur in mental illness. [23]
(Diagram 5)

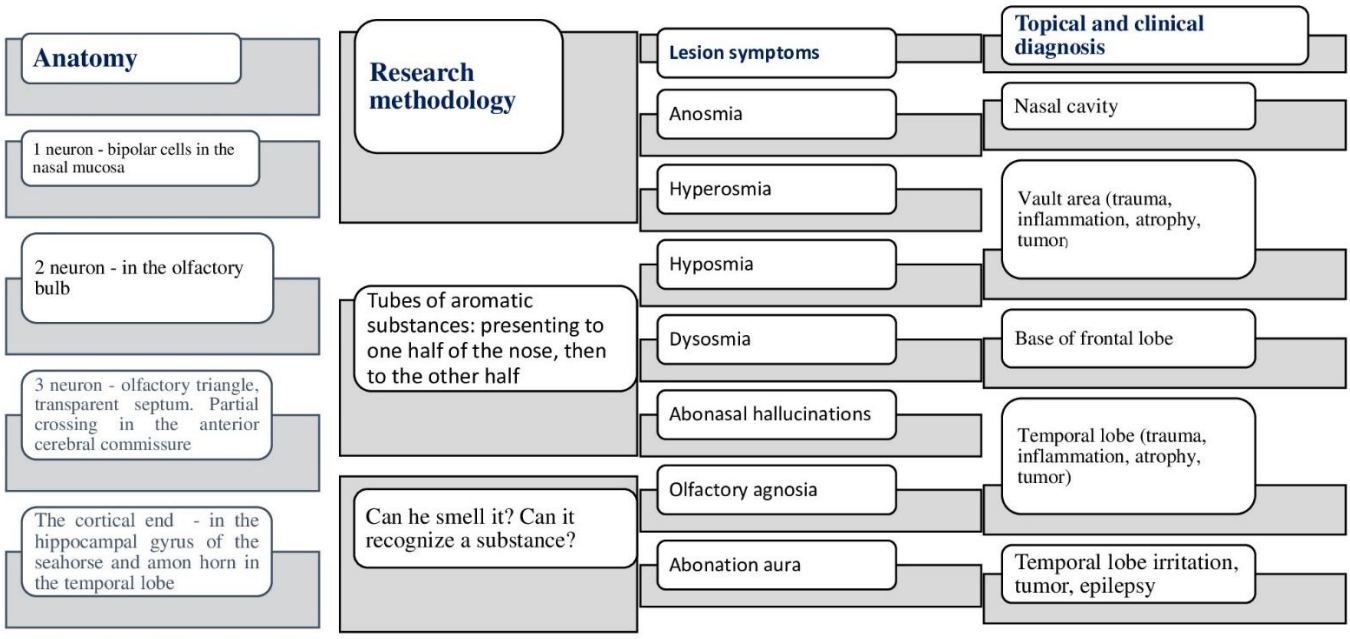

Diagram 5. Olfactory analyzer
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

4.2. Optic nerve

The visual analyzer consists of four neurons, the first three embedded in the retina.

The **first neuron** is the rods, which provide twilight vision, and the cones, which provide day and color vision.

The **second neuron** is bipolar cells.

The **third neuron** is the ganglion cells.

The axons form the optic nerve, which exits the retina through the optic disc, or "blind spot," enters the cranial cavity through the optic canal and reaches the base of the brain. It then reaches the sella turcica, in front of which it forms a chiasm - a partial crossing-over (fibers from the inner part of the retina cross, while fibers from the outer part of the retina continue on their side). Once crossed, the fibers are called the optic tract, with the left optic tract containing fibers from the left halves of the retinas and the right optic tract containing fibers from the right halves. Projection of the visible image onto the retina occurs by light rays passing through the refractive media of the eye. Because of this, the image is projected upside down along the horizontal axis, so the outer half of the visual field is projected in the inner part of the retina. The inner half is projected on the outer part of the retina. Therefore, the left visual tract carries information from the right half of the visual field, and the right visual tract carries information from the left half.

The **fourth neuron** is located in the outer geniculate bodies; its axons pass through the internal capsule, forming the Gratiolet's bundle, traverse the occipital

lobe, and reach the cortex of the occipital lobe in the area of the calcarine, lingual, and cuneus gyri, where analysis and perception of visual images occur.

Methodology for the examination of optic nerve function:

Visual acuity is examined using the Sivtsev table.
The patient stands 5 meters from the table, closes one eye in turn, and names the letters or signs visible to him or her on the table. The third row from the bottom is considered the norm and equals one.

Visual fields - the examination of visual fields is performed by perimetry. For this purpose, a perimeter is used: the patient sits with his back to the light, closes one eye, and with the open eye fixes his gaze motionless on the white dot of the device, the doctor sitting opposite marks with a pointer with a white dot the boundaries of the appearance of visibility of it from different sides of the patient. Computerized perimetry can be used.
The visual fields can be checked approximately at the patient's bedside using a towel or belt and the doctor's hammer, pen, or finger. The doctor sits opposite the patient and asks the patient to cover one eye and to look steadily at the bridge of the doctor's nose with the other eye. The doctor moves a hammer or pen or his index finger gradually approaching from the periphery of the imaginary circle around the patient to the center - to the patient's eye, from different directions and asks the patient to say when the patient sees the moving object.
When using a towel, the patient is asked to point to the center of the towel while fixing his gaze strictly in front of him, alternately closing one eye.

Color perception is examined using polychromatic charts like the Rabkin tables, where spots or dots of different colors depict numbers, letters, or figures.

Ophthalmoscopy is an examination of the fundus of the eye using an ophthalmoscope after prior dilation of the pupil with short-acting mydriatics. The optic disc is examined, as well as its borders, clarity or blurriness, inflammatory or atrophic phenomena, the state of the vessels and the retina itself, and others
Diagnostic special examination techniques include electroretinography, visual evoked potential studies, CT, MRI, ocular ultrasound, and fluorescence retinography.

Symptoms of optic nerve affections:

Amblyopia is a decreased visual acuity that occurs with partial affection of the first three neurons, optic nerve, eyeball, and retina on the affected side.

Amaurosis is a total absence of vision with total damage to any level, such as the first three neurons, or to the optic nerve, eyeball, or retina on the affected side.

Achromatopsia and **dyschromatopsia** are color perception disorders manifested in complete or partial loss of the ability to distinguish colors, occurring with cone dysfunction.

Hemianopsia is characterized by the loss of half of the visual fields.

Homonymous hemianopsia is a loss of homonymous halves of the visual field with lesions in the area of the optic tract, optic radiation, and occipital lobe of the opposite side.

Heteronymous hemianopsia is a loss of different halves of the visual field (right side on one side, left side on the other, or vice versa). Heteronymous hemianopsias include bitemporal and binasal hemianopsia.

Bitemporal hemianopsia is a loss of the outer visual fields when the inner part of the chiasma is affected, such as compression by a pituitary adenoma.

Binasal hemianopsia is a loss of the inner halves of the visual field with affections in the area of the outer parts of the chiasm on both sides.

Upper quadrant hemianopsia is a loss of ¼ of the visual field when the lingual gyrus or the inner part of the Gratiolet's bundle on the opposite side is affected.

Lower quadrant hemianopsia is a loss of ¼ of the visual field when the cuneiform gyrus or the outer part of the Gratiolet's bundle on the opposite side is affected.

Scotomas are losses of different shapes and sizes of separate parts of the visual fields.

Positive scotomas are losses of areas in the form of dark spots, with the patient noticing this defect - the lesion is in the inner layers of the retina or vitreous body.

Negative scotomas are losses of areas detected only by perimetry; the patient himself does not notice them. They indicate a partial affection of the optic nerves or in the overlying parts of the visual analyzer, particularly in the occipital lobe. The central visual field is preserved.

Visual agnosia occurs when the patient does not recognize previously familiar images when the occipital lobe is affected.

Metamorphopsias occur when images seen are distorted and irregular in shape and size (micropsia or macropsia); they also develop when the occipital lobe is affected.

Visual hallucinations or visual aura before an epileptic seizure occur when visible images do not exist in reality when the occipital lobe is irritated.

Ophthalmoscopy can reveal:

- **Congested optic disc** occurs in conditions associated with increased intracranial pressure of various origins.

- **Optic disc atrophy** - primary or secondary - occurs as a consequence of neuritis or disc congestion

- **Optic neuritis** - can occur with any infection [24-26] (Diagram 6).

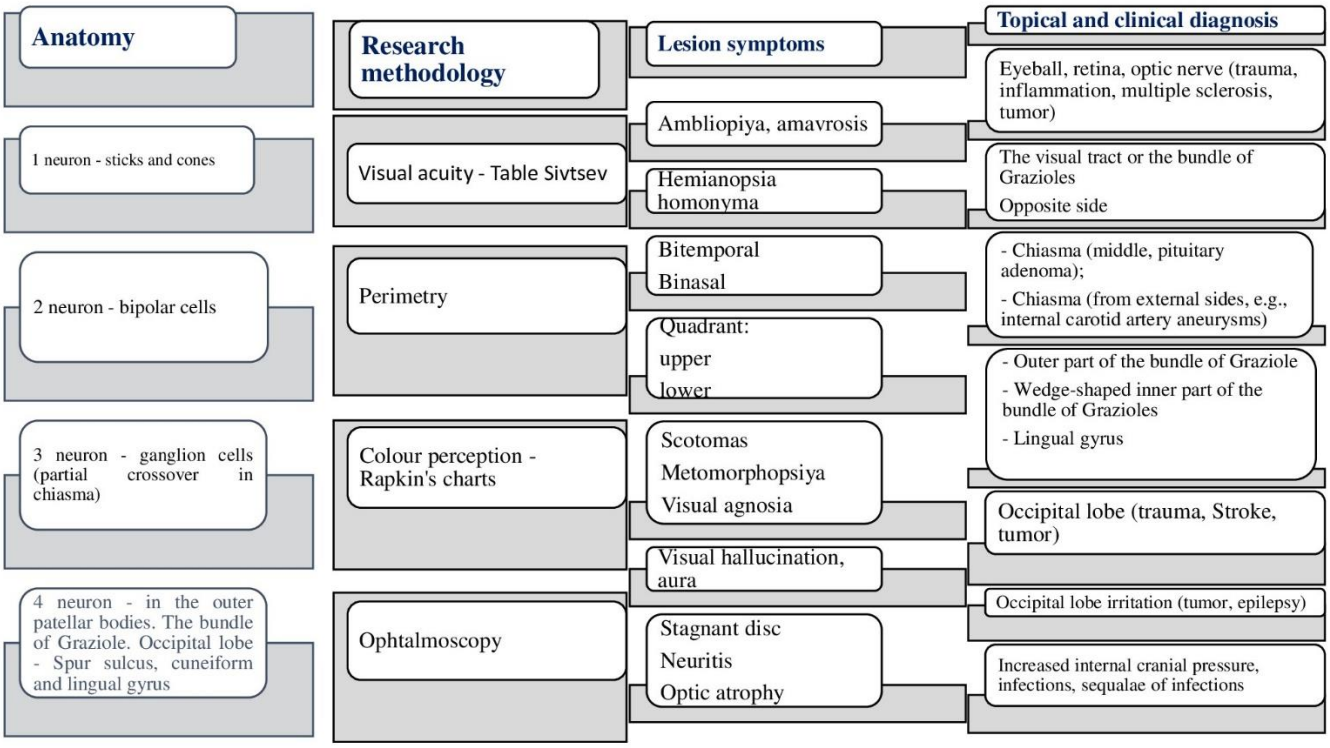

Diagram 6. Optic nerve
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

4.3. Oculomotor nerves (III pair - oculomotor nerve IV pair - trochlear nerve, VI pair - abducens nerve)

Eye movements are ensured by three pairs of cranial nerves: the oculomotor, trochlear, and abducens. In healthy people, eye movements are always synchronized vertically and horizontally. The dorsal longitudinal fasciculus system provides synchronicity.

All three nerves are components of the corticonuclear tract, which is known to consist of **two neurons: central and peripheral.**

The **first neurons** are located in the lower part of the anterior central gyrus; their axons pass through the internal capsule and reach the brainstem, where they make a partial crossing over the nuclei: 50% by 50%, that is, one-half of the fibers cross, and the other half goes on its side.

The **second neurons** are located in the nuclei of the brainstem.

The peripheral neurons of the **oculomotor nerve** are located in two large-cell somatic motor nuclei in the midbrain's tegmentum at the level of the anterior tubercles of the quadrigeminal plate. There are also three parasympathetic nuclei: the paired Yakubovich nucleus and the unpaired Perlia nucleus, which participate in the reflex arc of pupillary reactions. The nerve then exits the brain and goes to the cavernous sinus. The oculomotor nerve exits the cranial cavity through the superior orbital fissure. It innervates the following oculomotor transverse striated muscles: the muscle that raises the upper eyelid, the rectus muscles (upper, lower, and inner), the

inferior oblique muscle of the eye; as well as two smooth muscles - the ciliary muscle and the pupil constrictor muscle.

The **trochlear nerve** has a second neuron located in the midbrain tegmentum at the level of the lower tubercles of the quadrigeminal plate. It exits the brain, goes towards the cavernous sinus, exits the cranial cavity through the superior orbital fissure, and directs towards the single muscle - the superior oblique muscle of the eye.

The **abducens nerve** has a peripheral neuron located in the pontine tegmentum on the floor of the fourth ventricle. It exits the brain between the pons and the medulla oblongata, travels towards the wall of the cavernous sinus, exits the cranial cavity through the superior orbital fissure, and innervates the lateral rectus muscle of the eye. The function of the abducens nerve is to direct the eye to the external side.

Methods of examining the function of oculomotor nerves:

In the beginning, the patient is examined at rest; attention is paid to the size, width of the palpebral fissures, symmetry, position, condition, and mobility of the eyeballs, upper eyelids, size, shape, and size of the pupils.

Then, the eyeball movement is checked.

The scope of the eyeball movement. The patient is asked to keep the head fixedly straight, not to turn the head, but to follow with the eyes the movement of the hammer horizontally, vertically, and circumferentially to the tip of the nose. Holding the hammer at about 20 centimeters from the patient's face is recommended, then bringing it closer.

Pupillary response to light.

The patient should sit facing the light and should not cover their eyes. The doctor covers the patient's eyes from both sides with the palms of his hands and opens the eyes alternately, first one and then the other, noting the pupil's response to light and whether the size of the pupil decreases. This is a direct response of the pupil to light.

Conjoint response is tested by covering and opening only one eye with the palm with several repetitions while paying attention to the pupil size of the second eye. Normally, when one eye is opened, the pupil of the second eye narrows; when one eye is closed, the pupil of the second eye dilates.

Convergence and accommodation are tested by approaching the hammer from 50-60 centimeters to the nose, with pupils converging to the center, to the nose (this is called convergence), and narrowing in size (accommodation).

Symptoms of oculomotor nerve affection:

One-sided affections of central neurons of all three pairs of oculomotor nerves will not manifest focal symptoms because innervation of muscles will be ensured from the healthy side.

Only when the second neuron is affected will the symptomatology of peripheral paresis of muscles innervated by these nerves on the side of the affection be detected. In one-sided affections of peripheral neurons, a picture of peripheral paresis of the muscles they innervate on the side of the affection develops. Therefore, the symptoms described relate to peripheral neuronal affections of these nerves.

Symptoms and syndromes of oculomotor nerve damage (III pair): upper eyelid drooping **- *ptosis,*** pupil dilation **- *mydriasis,*** and ***divergent strabismus*.**

Alternating Weber syndrome - on the affected side - peripheral paresis of the oculomotor nerve: ptosis, mydriasis, divergent strabismus, on the opposite side - central spastic hemiparesis. In this case, the pathologic focus is located in the midbrain at the level of the anterior tubercles of the quadrigeminal plate on one side with involvement of the nucleus of the oculomotor nerve and the corticomuscular pyramidal tract.

Benedikt alternating syndrome is characterized by ptosis, mydriasis, and divergent strabismus on the affected side, while on the opposite side, there are athetoid hyperkinesia and intention tremor. The lesion is in the midbrain, which involves the oculomotor nucleus, red nucleus, and cerebellar tract.

Symptoms of trochlear nerve (IV pair) affections:

Diplopia when looking downward and mild limitation of downward mobility of the eyeball.

Symptoms of abducens nerve (VI cranial nerve) affections include ***convergent strabismus and diplopia****,* most pronounced when looking towards the affected nerve.

Foville alternating syndrome presents with peripheral paresis of the abducens nerve on the affected side (convergent strabismus with diplopia) and peripheral paresis of facial muscles. On the opposite side, there is central spastic hemiparesis. The lesion is the pons with involvement of the nuclei of the abducens and facial nerve and the pyramidal corticomuscular tract on one side.

The **Parinaud syndrome** is an affection in the area of the quadrigeminal plate resulting in paralysis of upward and downward gaze.

The **Magendie-Hertwig syndrome** is a vertical strabismus resulting from a focal affection with partial damage to the posterior longitudinal fasciculus.

Total ophthalmoplegia is complete immobility of the eyeball with ptosis and mydriasis, lack of pupil reaction, and occurs when all oculomotor nerves are affected. The pathological focus is in the cavernous sinus (for example, cavernous sinus thrombosis) or the superior ocular cleft, where all three nerves run together.

Anisocoria is different pupil sizes.

Anisocoria in severely comatose patients serves as a negative sign and may indicate brainstem compression during dislocation in cerebral edema.

The **Argyll Robertson syndrome** is the absence of pupillary response to light while maintaining response to convergence and accommodation, often associated with neurosyphilis.

The **reverse Argyll Robertson syndrome** is a preserved response to light, while lacking response to convergence and accommodation, often encountered in encephalitis lethargica [27] (Diagrams 7 and 8).

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
As part of the cortico-nuclear tract **1st neuron:** ➢ Betz cells in the lower part of the anterior central gyrus. ➢ Partial crossing 50-50% **2d neuron:** ➢ Upper tubercles of the quadratochalmia in two somatic nuclei. ➢ There are also vegetative nuclei: paired Jakubovich's and unpaired Perlea's nuclei ➢ It exits the skull through the superior ocular branch **Innervates the muscles:** -the muscle that lifts the upper eyelid, - three rectus muscles: upper, lower, inner -the lower oblique - Pupil constrictor muscle. - Ciliary	➢ - Examination of eyeballs, location, width, size of eye slits and pupils, their symmetry ➢ - Volume of eyeball movements ➢ - Reaction of pupils to light (direct and concomitant) ➢ - Pupillary response to convergence and accommodation	Ptosis Mydriasis Divergent strabismus Exophthalmos Weber syndrome (all of the above syndromes on the affected side and hemiparesis on the opposite side) Parinaud's syndrome Argylo Robertson syndrome Reverse Argyle Robertson syndrome	The level of the second neuron Midbrain, somatic nuclei + pyramidal tract Midbrain, autonomic nuclei

Diagram 7. The oculomotor nerve
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
As part of the cortico-nuclear tract **1st neuron:** ➢ Betz cells in the lower part of the anterior central gyrus. ➢ Partial crossing 50-50% **2d neuron:** - 2 neuron - inferior tuberosities of the quadriplegium for IV nerve - 2 neuron for VI nerve - Varolian bridge **Innervate the muscles:** -Anterior oblique (IV) - External rectus (VI)	- Examination of the location of the eyeballs - Volume of eyeball movements - Find out if there is double vision	- Double vision when looking down - Convergent strabismus and double vision when looking sideways - Fauville syndrome (VI,VII on the affected side + hemiparesis on the opposite side) - Ophthalmoplegia syndrome (all 3 nerves). - Eye immobile, absence of pupillary reflexes, ptosis, exophthalmos.	2 neuron of the IV nerve 2 neuron of nerve VI Varolian bridge Cavernous sinus or superior orbital fissure

Diagram 8. Trochlear and abducens nerves
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

4.4. Trigeminal nerve

The trigeminal nerve is mixed, consisting of sensory, motor, and autonomic fibers.

The **sensory component** consists of two tracts: the deep sensitivity tract and the superficial sensitivity tract.

The difference between the two tracts is only in the location of the receptors (respectively, the receptors of superficial sensitivity are located superficially, those of deep sensitivity - deeper) and in the localization of the second neuron. The rest is all the same. The first neuron is located in the Gasserian node. The second neuron for superficial sensitivity is located in the long spinal nucleus in the upper cervical spinal cord, and in the medulla oblongata, the second neuron for deep sensitivity is located in the terminal nucleus of the pons. Then, the axon of the second neuron makes a decussation and goes to the thalamus; in the thalamus - the third neuron, the axon of the third neuron passes through the internal capsule and ends in the lower parts of the posterior central gyrus of the opposite side.

When facial receptors are irritated, transduction occurs, i.e., nerve impulse formation and transmission of these impulses through the three divisions of the trigeminal nerve: ophthalmic, maxillary, and mandibular.

The **ophthalmic division** ensures skin sensitivity of the forehead, anterior scalp, upper eyelid, inner corner of the eye, and nasal bridge. It also ensures sensitivity to the mucous membrane of the upper part of the nasal cavity, the eye, ethmoidal sinuses, lacrimal gland, conjunctiva, cornea, dura mater, cerebellar tentorium, frontal bone, and periosteum.

The **maxillary division** ensures skin sensitivity of the temporal and zygomatic areas, lower eyelid and outer corner of the eye, mucous membrane of the posterior ethmoidal cells and sphenoidal sinus, nasal cavity, pharyngeal vault, soft and hard palate, tonsils, teeth, and upper jaw.

The **mandibular division** is a mixed nerve and has sensory fibers and motor fibers. It ensures skin sensitivity of the lower part of the cheeks, chin, lower lip, anterior part of the auricle, external acoustic meatus, part of the tympanic membrane, mucous membrane of the cheeks, the floor of the mouth, anterior two-thirds of the tongue, lower jaw, dura mater, teeth, and gums of the lower jaw.

Thus, it is most important to remember the three main divisions of the trigeminal nerve: ophthalmic, maxillary, and mandibular.

These main three divisions each pass through their respective foramen, so through the supraorbital foramen, the ophthalmic division goes, through the suborbital foramen, the maxillary division goes, and through the submaxillary foramen, the mandibular division goes. They enter the cranial cavity through the superior orbital fissure, the round foramen, and the oval foramen (in the order listed).

The **motor part** is a constituent element of the corticonuclear tract.

The **first neuron** is located in the lower parts of the anterior central gyrus.

Axons pass through the internal capsule, reach the pons, and make an incomplete 50% by 50% decussation over the nucleus.

The **second neuron** is located in the masseteric nucleus in the pons.

The axon of the second neuron within the third division of the trigeminal nerve, the mandibular division, reaches the masseter muscles and ensures their innervation. Namely mm. masseter, temporalis, pterigoideus medialis and lateralis, mylohyoideus, anterior belly of m.digastricus and m.tensor veli palatini.

The **autonomic part of the trigeminal nerve** is linked to parasympathetic and sympathetic nerve ganglia. The ophthalmic nerve's first division is linked to the ciliary ganglion. The second division - the maxillary nerve - is linked to the pterygopalatine ganglion. The third division - the mandibular nerve - is linked to the otic and submandibular ganglia. [27,28].

Methods of examining the function of the trigeminal nerve:

Examination of the masseter muscle:

In the beginning, the doctor examines the face at rest and whether there is a displacement of the lower jaw. After, the doctor examines when the mouth is open,

palpates the temporal and masseter muscles, puts the palms of the hands on these muscles, asks the patient to clench and unclench the teeth firmly, and assesses the degree of tension of these muscles on both sides, also asks to move the jaw to the sides.

Examination of all types of sensitivity on the face:

Superficial sensitivity:

Pain sensitivity is tested by using a sharp object to lightly prick the skin on symmetrical areas within the zones innervated by the three branches of the trigeminal nerve. Additionally, the Sölder areas are tested (with the patient's eyes closed, asking the patient to indicate the sensation and whether it is felt the same on both symmetrical areas of the face).

Temperature sensitivity is tested using cold and warm objects or tubes of water of different temperatures by touching facial skin areas. Light tactile sensitivity is examined by lightly touching, for example, a tissue to the facial skin.

Examination of trigger points on the face:

The exit points of the divisions of the trigeminal nerve on the face are checked by pressing on them with the pads of the fingers. When they are painful, these sites have marked soreness and reactivity in various processes with these divisions, more often inflammatory or neuralgic manifestations. These points on the face are called Valle points.

Deep sensitivity: vibratory, kinesthetic, deep tactile sense of pressure is tested on the face, similar to the methods of examination on the body, only on the zones of innervation of the three divisions.

Examination of reflexes:

The ***corneal reflex***: A soft tissue or absorbent cotton is used to touch the iris of the patient's eye lightly, and the eye reflexively closes.

The ***conjunctival reflex***: A tissue is used to touch the eye's conjunctiva lightly, causing the eye to close.

The ***supraorbital reflex*** is checked with a light blow of a hammer on the bridge of the nose and the supraorbital arch, which causes the eyelids to close.

The ***mandibular reflex*** is checked by tapping lightly with the hammer on the chin with a slightly open mouth; the jaws clench.

Taste on the anterior two-thirds of the tongue is also tested.

Symptoms of trigeminal nerve affections:

Motor disorders occur in the lesion of the second peripheral neurons in the motor nuclei or the lower division of the trigeminal nerve - the mandibular nerve, manifested by peripheral paralysis or paresis of the masticatory muscles on the side of the lesion: atrophy or hypotrophy and fibrillary twitching of fibers of the temporal muscle (depression in the temporal region), masticatory muscle (in the area of the angle of the mandible). During palpation, there is a decrease in the tension of these muscles when chewing, and when opening the mouth, the lower jaw is displaced to the side of the affected nerve. The mandibular reflex is decreased or absent.

In two-sided motor root affection, there is paralysis of the masticatory muscles on both sides, manifested by sagging of the lower jaw and a decrease or disappearance of the mandibular reflex.

When the mandibular nerve is damaged, in addition to the motor disorders mentioned above, there will be sensory disorders in this nerve's innervation zone. Gustatory sensitivity disorders are observed on the anterior two-thirds of the tongue since the mandibular nerve and chorda tympani are also involved in gustatory sensitivity.

When the upper two branches of the trigeminal nerve are damaged, all sensations in the zone of their innervation on the affected side are disturbed.

Anesthesia is a lack of sensitivity.

Hypesthesia is a decrease in sensitivity.

Hyperesthesia is an increase in sensitivity.

Paresthesia is numbness and sensation of crawling goosebumps.

Dysesthesia **is a perversion of sensation.**

Pain in one half of the face is prosopalgia.

Pain in the area of innervation of the divisions of the trigeminal nerve - with neuralgia of the divisions of the trigeminal nerve or any other, such as an inflammatory process.

When the nucleus of the long spinal cord is damaged, there is a disorder of pain and temperature sensitivity in the zones, in the form of **Sölder** concentric brackets (segmental, or nuclear, type): when the caudal part of the nucleus is destroyed, superficial sensitivity is lost in the most external parts of the face, when the oral part is damaged - in the central parts of the face.

Damage to the Gasserian ganglion is characterized by the onset of pain and sensory disturbances in the innervated area of the trigeminal nerve branches on the affected side and often presents with herpetic eruptions on the same side of the face.

Irritation of the trigeminal nerve or its divisions is manifested by pain in the zone of its innervation, autonomic manifestations, and lacrimation; there may be sweating disorders and hyperemia of some areas or the entire half of the face. Any touch or movement of the facial muscles can lead to pain attacks, so the patient "freezes" and tries not to move or touch the face. This is how trigeminal neuralgia manifests.

When the motor nuclei or masseter muscle centers in the cortex are irritated, there may be tonic tension of the masseter muscles in the type of trismus. During trismus, it is impossible to unclench the jaws, food intake and speech are impossible, and even breathing becomes difficult [27, p.28] (Diagram 9).

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
3 branches: -Oculomotor nerve - Maxillary nerve - Mandibular nerve	- Feel the exit points on the face of the branches **Sensitivity study:** - pain -temperature -tactile - kinesthetic sensitivity using branch zones and Zelder's brackets **Examine the reflexes:** -corneal -conjunctival -supraorbital **To examine the masseter muscles**	- Hypesthesia - Hyperesthesia - Anesthesia -Paraesthesia -Pains	Nerve root or branch lesions
Sensitive fibers			
1st neuron: ➢ Trigeminal ganglion (Gasser's ganglion)		Herpetic rashes Pains	Gasser's ganglion
2d neuron: - The nucleus of the spinal pathway (superficial sensitivity);		Dissociative disorder with loss of surface sensation	The nucleus of the spinal pathway according to Zeldorov brackets
- Terminal nucleus in the Varolian bridge, makes a complete crossing over the nucleus (deep sensitivity)		Dissociative disorder with loss of deep sensation	Terminal nucleus
3d neuron: The visual tubercle is the lower section of the posterior central gyrus of the cerebral cortex		Peripheral paresis of the vestibular muscles	3-d branch
Motor fibers			
1st neuron: Lower section of the anterior central gyrus of the cerebral cortex In the Varolian bridge makes a partial crossing over the nuclei		Wallenberg-Zakharchenko syndrome	In the medulla oblongata
2nd neuron: Chewing nuclei of the trigeminal nerve		Paresthesias in half the face	Irritation of the inferior posterior central gyrus

Diagram 9. Trigeminal nerve
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

4.5. Facial nerve

The facial nerve is a mixed nerve; it has motor, sensory, and autonomic components.

The **motor portion** of it is part of the corticonuclear tract.

The **first neuron** is located in the inferior part of the anterior central gyrus. The axon passes through the internal capsule, through the midbrain, and in the pons above the nucleus, makes a decussation, with a partial decussation over the upper part of the nucleus: 50% by 50%, and a complete decussation of 100% over the lower part of the nucleus.

The **second neuron** is located in the pontine nucleus. The axon of the second neuron exits the brain through the pontocerebellar angle, passes through the fallopian canal, and exits behind the ear onto the face through the stylomastoid foramen. It

divides into several branches resembling a "goosefoot," which ensures innervation of the facial muscles.

The **fallopian canal** has the following branches:

- The **greater petrosal nerve (n.petrosus major)** innervates the lacrimal gland,
- The **stapedius nerve (n.stapedius)** innervates the stapedius muscle within the inner ear.
- **Chorda tympani** consists of gustatory fibers that provide the anterior two-thirds of the tongue with gustatory sensitivity.

and the salivary secretory fibers, which innervate the submandibular and sublingual salivary glands.

Methods of examination of the functions of the facial nerve:

In the beginning, the face is examined **at rest**, with an assessment of the symmetry of the face, the expression of natural folds, whether there is no muscle twitching in the face, and whether there is no lacrimation or vice versa dry eye.

Mimic movements:

The patient is asked to raise their eyebrows, lower them, frown, close their eyes, open their eyes, smile, grind their teeth, open their mouth, close their mouth, puff up their cheeks, and whistle. The synchronization of movements, symmetry, and strength of cheek muscles when inflating them by lightly tapping the cheeks with finger pads is assessed.

Gustatory sensitivity can be tested by applying drops of different solutions by gustatory component (sweet, salty, and others) with a pipette to the anterior two-thirds of the tongue. Dry mouth or excessive salivation is also assessed.

It is also necessary to ask the patient if there is any murmur in the ear, which may be present when the stapedius nerve is affected.

Symptoms of facial nerve affections:

Central paralysis or paresis of the mimic muscles

develops when the central neuron of the facial nerve is affected.

In this case, on the opposite side, the patient develops paresis of the mimic muscles of the lower part of the face, manifested by asymmetry in the lower part.

The following symptoms are identified:

- Charcot's exclamation mark symptom is an asymmetry of the grin or smile, smoothing of the nasolabial fold,
- Sail symptom is when air comes out of the mouth when the patient is trying to puff up their cheeks;
- when eating, food gets stuck between cheek and teeth,
- When eating, food gets stuck between the cheek and teeth,
- If the patient is asked to whistle, it is difficult for the patient.

The upper part of the face is not significantly affected in central paresis because it receives innervation from the healthy side.

Peripheral paresis of facial muscles

Peripheral paresis of facial muscles develops due to the affection of the second peripheral neuron of the facial nerve either in the nucleus at the level of the medulla oblongata or its axon at any level: for example, at the cerebellopontine angle, within the fallopian canal, or in the stylomastoid foramen.

In this case, paresis of the mimic muscles of the entire half of the face is detected: both the upper and lower parts of the face on the affected side.

This symptom complex has been named Bell's Palsy or facial palsy.

Lagophthalmos is a condition where the eye becomes enlarged, and the patient notes an inability to close or fully close it. When attempting to close the eye, the white sclera may be visible beneath the iris. This symptom is also known as "Bell's sign" and "hare eye."

When attempting to raise the eyebrows, frown, or squeeze the eyes, the patient experiences difficulty on the affected side.

When attempting to smile, "exclamation mark" symptoms are noted; when attempting to puff up the cheeks, the "sail" symptom is noted.

All natural folds and wrinkles on the affected side are smoothed out, and there may even be puffiness of the face and drooping of half of the face downward.

The **alternating syndrome of Millard-Gubler** is an affection of the facial nerve nucleus in the pons, along with the corticomuscular pyramidal tract. It causes *peripheral paralysis* of the facial musculature on the affected side, while on the opposite side, it results in central spastic hemiparesis.

Foville alternating syndrome entails a peripheral paralysis presentation of the abducens nerve on the affected side, resulting in convergent strabismus with diplopia and peripheral paresis of the facial muscles. On the opposite side, it leads to central spastic hemiparesis. The lesion is the pons with involvement of the nuclei of the abducens and facial nerve and the pyramidal corticomuscular tract on one side.

The **cerebellopontine angle syndrome**, when affecting the facial nerve at the level of the cerebellopontine angle, presents with peripheral facial nerve palsy on the affected side and may involve the fifth and eighth (vestibulocochlear) cranial nerves.

When the facial nerve is damaged in the upper part **of the fallopian canal**, all its associated branches are involved in the pathological process: the greater petrosal nerve, stapedius nerve, and chorda tympani.

In this case, peripheral facial palsy is accompanied by the following symptoms: **xerophthalmia** (dryness of the eye), **hyperacusis** (murmur in the ear), **xerostomia** (dry mouth), **hypogeusia** (reduced sense of taste), or **ageusia** (absence of taste) on the anterior two-thirds of the tongue on the affected side.

If the pathological lesion is located after the branch of the greater petrosal nerve, it leads to lacrimation, and all the symptoms above are present.

If the lesion is located after the branch of the stapedius nerve, there is no hyperacusis, but gustatory and salivation disorders, together with peripheral facial palsy and lacrimation, are present. [27, p.32]

If the lesion is located below the fallopian canal after the branch of the chorda tympani nerve, the affection will manifest solely as motor disorders, presenting as peripheral facial palsy and lacrimation on the affected side (Diagram 10).

<table>
<tr><th>ANATOMY</th><th>RESEARCH TEQUNIQUES</th><th>LESION SYMPTOMS</th><th>TOPICAL DIAGNOSIS</th></tr>
<tr><td rowspan="3">1st neuron:
➢ Cortex 5th layer of Betz cells - in the lower parts of the anterior central gyrus
2d neuron:
- the nucleus in the pons Varolii
➢ makes a partial cross over the top of the core (50% by 50%)</td><td rowspan="3">Examine the facial muscles:
ask the patient to raise and furrow their eyebrows, close and squeeze their eyes, puff up their cheeks (feel them and lightly tap them with a finger to check their strength), smile, grin, bite their teeth, whistle.

To examine taste sensitivity: on the anterior two thirds of the tongue by dropping solutions of different tastes from a pipette

Examine lacrimal gland function (dryness or lacrimation)
To investigate (ask) tinnitus

Examine the lacrimal glands (dry mouth or increased salivation)</td><td>Central paresis
Asymmetry of the lower half of the face, on the opposite half of the face

Peripheral paresis on the affected side
Prosoparesis:
Bell's symptom
Hare's eye symptom
Sail symptom
Charcot's sign exclamation eye symptom

Alternating Miyar-Gubler-Fauville syndrome</td><td>A central neuron lesion

Peripheral neuron lesion

Varolii pons</td></tr>
<tr><td>Autonomic sensory branches in the Fallopian tube region</td><td>prosoparesis+xerophthalmia
hyperacusis
hypergeusia
xerostomia</td><td>lesion in the fallopian tube of all branches</td></tr>
<tr><td>1st: The great petrosal nerve innervates the lacrimal gland
2nd: nerve stapedius to the inner ear
3d: tympanic string. Innervates submandibular and hyoid salivary glands, the anterior two thirds of the tongue provides taste sensitivity</td><td>prosoparesis + lacrimation</td><td>The facial nerve after the fallopian canal</td></tr>
</table>

Diagram 10. Facial nerve
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

4.6. Cochleovestibular nerve

The **cochleovestibular nerve** has two parts: the auditory and vestibular. It is a sensitive sensory nerve, and its tracts are afferent.
The **auditory part** consists of three neurons.
The first neuron is located in the spiral node.
The second neuron is located in the dorsal and ventral nuclei in the pons.
The axons of the second neuron make a partial decussation.
The third neuron resides in the medial geniculate bodies and the lower tubercles of the quadrigeminal plate.
It terminates in the Heschl's gyrus in Brodmann's area 22.

Vestibular fibers also have a three-neuron structure.
The first neuron is the Scarpa's ganglion.

The second neuron is in the nuclei of Deiters, Schwalbe, Roller, and Bechterev in the medulla oblongata.
The axons of the second neuron make a partial decussation.
The third neuron is in the thalamus.
It terminates in the temporal lobe near the auditory projection zone.
The vestibular nerve connects with the spinal cord, the cerebellum, the posterior longitudinal fasciculus - the gaze innervation system, and the vagus nerve's nucleus.

Methods of examination of the cochleovestibular nerve:

When asking the patient questions, the doctor should clarify if there is any hearing loss, murmur, ringing, or auditory hallucinations. The external ear canal and tympanic membrane are examined.

Hearing acuity is tested by whispered speech at a distance of 5-6 meters from the patient, with the patient covering each ear in turn.

Tuning fork tests are used to determine the nature of hypoacusis:

The ***Weber test.*** A vibrating tuning fork (128 Hz) is placed on the midline of the patient's vertex, and the patient is asked if they hear the sound equally on both sides. If the patient hears better on the healthy side (lateralization of sound perception to the healthy side), it indicates sensorineural hearing loss due to damage to the auditory nerve (for example, neurinoma or Meniere's disease, and others).

If bone conduction is better on the affected side (lateralization of sound perception to the affected side), it indicates conductive hearing loss, which is associated with damage to the peripheral part (for example, impacted earwax in the external auditory canal, middle ear pathology).

The ***Rinne test.*** This test compares air conduction and bone conduction in a patient with hearing loss. A vibrating tuning fork (512 Hz) is placed on the mastoid process area of the patient. When the patient no longer hears it, the same tuning fork is placed close to the ear without touching it. The test is called positive if the patient hears sound through the air. This means that air conduction is better than bone conduction, indicating sensorineural hearing loss, a condition affecting the auditory nerve;

In the case where bone conduction is better than air conduction, the patient will not hear the sound of the tuning fork when it is placed close to the ear, indicating a negative Rinne test. This indicates conductive hearing loss.

- A hearing data sheet is filled out.
- Audiometry, a special instrumental test, is performed.
- Detecting the phenomenon of accelerating volume growth.
- Assessment of speech intelligibility.
- Index of fine increment of sound.
- Registration of auditory evoked potentials.

The following techniques are used to examine the vestibular apparatus:

During the examination, the doctor asks whether there is any dizziness, whether it depends on the head's position and turning, or whether it occurs upon standing up.

The following tests are performed:

- The Romberg test
- Walking with eyes open and closed in a straight line
- Flank walking - with side steps.
- Examination of nystagmus during rotational and caloric testing.

Symptoms of cochleovestibular nerve affections:

Hearing loss, or **hypoacusis**, can occur in three types: conductive (involving the conductive pathway of sound to the cochlear receptors, such as with impacted earwax or middle ear pathology), sensorineural (involving the cochlea and auditory nerve), and central (involving the brainstem nuclei and the temporal lobe cortex). The first type is primarily addressed by otolaryngologists, while the second and third types are also managed by neurologists.

Hypoacusis for low sounds more often indicates conductive hearing loss.
Hypoacusis to high sounds - auditory nerve damage - sensorineural hearing loss.

Anacusis, a total hearing loss, can occur with complete damage to the auditory nerve and significant processes in the peripheral parts of the auditory apparatus on the affected side. Anacusis may also be present when ventral and dorsal nuclei are affected in the brainstem on the affected side.

Hyperacusis is an aggravation of hearing.

Noise in the ear, or **tinnitus**, is auditory sensations such as ringing, buzzing, clicking, hissing, whistling, crackling, or pulsing in the ears.
Affection of the auditory apparatus may be accompanied by a noise, crackling, or whistling sensation.

Disorder in recognizing the time of appearance and disappearance of sound occurs when midbrain neurons are affected**.**

Auditory agnosia is an impairment of recognizing auditory signals due to the cortical endpoint of the auditory analyzer being affected. This condition is often accompanied by sensory aphasia, which is related explicitly to auditory agnosia.

Auditory hallucinations, or auditory aura, develop when the cortical centers of hearing are irritated.

Vestibular apparatus impairment:

True vertigo is perceived as an illusion of movement of the person or the surrounding objects. At any level of vestibular apparatus impairment, the patient experiences dizziness.
Systemic dizziness can occur both at rest and during movement.
Episodes of dizziness (labyrinthine attacks) lasting from several seconds to several hours are possible.

Meniere-like symptom complex with associated nausea, vomiting, and transient loss of consciousness.

Nystagmus - rhythmic movements of the eyeballs at extreme deviations, not disappearing immediately when the eyes are moved back to the center. Nystagmus can be jerky - clonic, or pendular. Nystagmus can be horizontal, vertical, or rotatory.

Vestibular ataxia **can be detected using the Romberg test and gait examination with eyes closed and open.** Deviation during the performance of these tests towards the side of the affected labyrinth occurs in cases of unilateral peripheral vestibular pathology. Vestibular ataxia intensifies with sudden changes in head position and gaze direction.

In the presence of vestibular ataxia, including **dizziness, nausea, vomiting, and horizontal and rotatory nystagmus**, the pathologic lesion is most likely to be in the semicircular channels and vestibular nerve.

If vestibular ataxia is accompanied by rotatory, vertical, and convergent nystagmus, the pathological focus may be in the brainstem.

If **psychosensory disturbances** accompany vestibular ataxia, the lesion may involve the temporal lobe [27, p. 38] (Diagram 11).

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
1st neuron: ganglion spirale cochleae **2d neuron:** - Nuclei dorsal and ventral to the pons Varolii (superficial sensitivity) - makes a partial crossing **Vestibular fibers** **1st:** ganglion vestibularae Scarpae **2nd:** - Vestibular nuclei of Bekhterev and Deiters in the medulla oblongata - Partial crossing **3d:** - Midbrain - Cortical end - temporal lobe	➢ **Hearing acuity, whispered speech at a distance of 6 meters** ➢ **Chambertonal tests:** - Weber: + on the healthy side, when the auditory nerve is affected; + on the affected side, when the inner ear is affected; -Rinne: + when air conduction is higher than bone conduction - when the auditory nerve is affected; - when bone conduction is higher than air conduction - in inner ear lesions ➢ Hearing Passport ➢ Audiogram ➢ Romberg test ➢ Walking with eyes open and closed ➢ Flank walking ➢ Examination of nystagmus during rotational and caloric gaze	Hypoacusis for low sounds Hypoacusis for high sounds or Anacusia Disorder in recognizing the time of appearance and disappearance of sound Auditory agnosia Sensory aphasia Auditory hallucinations Auditory aura Vestibular ataxia (VA): dizziness, vomiting, nausea, horizontal rotatory nystagmus VA+rotatory, vertical, convergent nystagmus VA+psychosensory disorders	Inner ear lesions Nerve damage Midbrain Temporal lobe Temporal lobe irritation Semicircular tubules, nerve brain stem Temporal lobe

Diagram 11. Cochleovestibular nerve
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

4.7. Glossopharyngeal nerve and vagus nerve

The glossopharyngeal and the vagus nerves are mixed nerves.

The **motor component** of both nerves is similar, as they are both part of the corticonuclear tract.

The first neuron is located in the lower regions of the central gyrus.

The axons of the first neuron pass through the internal capsule, then through the entire brainstem, and in the medulla oblongata, they make a partial decussation of 50% by 50%.

The second neuron is located in the reciprocal nucleus in the medulla oblongata.

The axons of the second neuron exit the brain and the cranial cavity through the jugular foramen and reach the stylopharyngeal muscle, the pharynx muscles, soft palate and larynx, epiglottis, and upper esophagus.

They ensure the participation of these muscles in swallowing, phonation, pharyngeal, gagging, cough reflexes, and the palatal reflex.

The sensory portion of the glossopharyngeal nerve.

The first neuron is located in the superior and inferior jugular nuclei.

The second neuron is in the medulla oblongata nuclei.

The axons of the second neuron make a partial decussation of 50% by 50%

The third neuron is in the thalamus.

It passes through the internal capsule.

It terminates in the inferior portions of the posterior central gyrus.

Thus, the glossopharyngeal nerve provides sensation to the posterior one-third of the tongue, soft palate, oropharynx, pharynx, anterior surface of the epiglottis, auditory tube, and tympanic cavity.

For the **gustatory sensitivity** of the posterior one-third of the tongue, the dendrites extend to the first neuron, located in the inferior ganglion of the glossopharyngeal nerve.

The second neuron is located in the medulla oblongata, specifically in the nucleus of the solitary tract (n.solitarius), which is shared with the cochlear nerve. The axons form a decussation over and reach the thalamus.

In the thalamus, there is a third neuron whose axon terminates in the temporal lobe within the parahippocampal lobule.

Parasympathetic fibers start from the hypothalamus, go to the inferior salivary nucleus in the medulla oblongata, and provide innervation to the parotid salivary gland.

The sensory portion of the vagus nerve has a three-neuron structure.

Receptors are interoceptors in internal organs.

The fibers are dendrites of the first neuron, reaching the superior and inferior nucleus near the jugular foramen.

The first neuron is located in these nuclei.

The second neuron is located in the medulla oblongata.

The axon of the second neuron makes a complete decussation.

The third neuron is located in the thalamus.

It goes through the internal capsule.

It terminates in the inferior portions of the posterior central gyrus.

It provides sensation to the dura mater of the posterior cranial fossa, the posterior wall of the external auditory canal, part of the skin of the auricle, the mucous membrane of the pharynx, larynx, upper part of the trachea, and internal organs.

The **autonomic parasympathetic portion of the vagus nerve** originates in the hypothalamus, then projects to the dorsal nucleus of the vagus nerve, and from there to the internal organs.

It provides parasympathetic innervation to nearly all internal organs. The impulses slow the heart rate, dilate blood vessels, constrict bronchi, and increase intestinal peristalsis [27, p.40].

Methods of examination of the glossopharyngeal and the vagus nerves

To assess the condition of the glossopharyngeal and vagus nerves, the doctor should pay attention to the patient's voice, the condition of the soft palate, and swallowing. The gag reflex from the soft palate should also be checked. Pulse, blood pressure, respiratory rate and digestive tract activity should be checked.

- *Ask the patient to open their mouth and say "A-A-A."*
- *Assess the scope of motion of the soft palate, the uvula,*
- *and the position of the arches and uvula.*
- *Assess the resonance of the voice.*

Examine the sensitivity:

- *pain sensitivity;*
- *temperature sensitivity;*
- *tactile sensitivity;*
- *gustatory sensitivity at the root of the tongue;*

Examine the reflexes:

- *swallowing reflex;*
- *palatal reflex;*
- *gag and cough reflexes;*

Examine the parotid glands.

Check *the pulse, BP, respiratory rate.*

Symptoms of damage to the glossopharyngeal and vagus nerves:

In the case of unilateral damage to the sensory fibers and peripheral motor neurons of the 9th and 10th pairs, the following symptoms may be observed on the affected side:

Hypesthesia, hyperesthesia, anesthesia, and pain in the area of innervation of these nerves

Nasolalia – a nasal tone of voice;

Hypophonia, aphonia, dysphonia – a decrease, lack of resonance of voice, sound disorder

Dysphagia – a swallowing disorder, choking when swallowing;

Hypogeusia – a decrease in gustatory sensitivity at the root of the tongue;

Ageusia – absence of taste on the base of the tongueж

Parageusia – a perversion of taste;

Hypo-areflexia – a decrease or absence of reflexes: pharyngeal, palatal, gag, and cough;

Xerostomia **– dry mouth;**

Soft palate arch prolapse and uvula deviation;

Tachycardia - a rapid heartbeat;

Autonomic disorders, disorders of internal organ function

Laryngoscopy identifies **vocal cord paralysis**.

Respiratory and cardiac arrest with a complete lesion of nuclei 9 and 10 pairs

Alternating Wallenberg-Zakharchenko syndrome is a unilateral lesion in the medulla oblongata with involvement of the nuclei of the 9th and 10th cranial nerves, the nucleus of the descending tract of the trigeminal nerve, and the spinothalamic tract; clinical manifestations on the affected side are paresis of the soft palate, pharyngeal muscles, larynx, vocal cords, drooping of the soft palate with a deviation of the uvula, hypophonia, dysphagia, nasolalia, reduced pharyngeal reflex, hypoesthesia of superficial sensitivity on half of the face; hemihypesthesia on the opposite side.

Alternating Avellis syndrome: The symptom complex manifests with symptoms of peripheral paresis of the 9th and 10th pairs of cranial nerves on the affected side and central spastic hemiparesis on the opposite side. The focus of the lesion is located in the medulla oblongata with involvement of the 9th and 10th cranial nerve nuclei and the corticomuscular pathway on one side.

Schmidt alternating syndrome is characterized by the clinical picture of involvement of the 9th, 10th, and 11th pairs of cranial nerves: paresis of the soft palate, vocal cords, sternocleidomastoid, and trapezius muscles on the affected side, and central hemiparesis on the opposite side [27, p.42]. (Diagram 12).

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
1st neuron: ➢ Ganglia: superior petrosum, jugularis nodosum **2d neuron:** - The medulla oblongata makes a complete cross **3d neuron:** ➢ Visual tubercle ➢ cortical end - posterior central gyrus **Additional nuclei in the medulla oblongata: n.Solivatorius, n.salitarii, n.Vegetative parasympathetic**	➢ Ask me to open my mouth A-A-A-A ➢ Estimate the volume of soft palate and uvula movements ➢ Location arcus and uvula ➢ Evaluate the sound of the voice ➢ Explore sensitivities: - pain -temperature -tactile - Taste on the root of the tongue ➢ Examine the reflexes: -pharyngeal -palatal -vomiting and coughing ➢ Examine the parotid glands ➢ Monitor pulse, BP, respiratory rate	Hypesthesia hyperesthesia Anesthesia pains Nasolalia Hypo-aphonia Dysphagia Hypogeusia Hypo-areflexia Xerostomia Palatal arch prolapse Uvula asymmetry Tachycardia Tachypnea Autonomic disorders Respiratory and cardiac arrest Wallenberg-Zakharchenko syndrome	Lesion of sensory fibers and peripheral motoneuron or nerves of pairs 9 and 10 Complete lesion of the medulla oblongata medulla oblongata
Motor fibers			
1st: lower section of the anterior central gyrus of the cerebral cortex oblongata, there's a partial crossing over the nuclei 50% by 50%. **2nd:** The medulla oblongata			

Diagram 12. Glossopharyngeal and vagus nerves
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

4.8. Accessory nerve

The accessory nerve is a purely motor nerve. It is a part of the corticonuclear tract.

The **first neuron** is in the lower parts of the anterior central gyrus. The axons pass through the internal capsule, then through the entire brainstem to its lower part, and in the medulla oblongata, they make a partial decussation of 50% by 50%.

The **second neuron** is located in two nuclei: the cerebral nucleus, located in the medulla oblongata, and the spinal nucleus, found in the anterior horns of the spinal cord and the upper cervical region.

The axons of the second neuron from the spinal nucleus ascend through the foramen magnum into the brain, where they join with the axons from the cerebral nucleus. Together, they exit the cranial cavity through the jugular foramen and proceed to innervate the trapezius and sternocleidomastoid muscles.

Methods of examination of the accessory nerve:

First of all, attention is paid to the position of the head and upper shoulder girdle of the patient at rest; whether there is any asymmetry, how the head is held, how the shoulders are positioned, attention is also paid to whether there is torticollis, muscle atrophy, and fibrillary twitching in the muscles of the neck, upper back, and arms. Some tests are then performed to determine if there is any pathology of the accessory nerve.

The patient is asked to move the head forward, backward, sideways, and towards the shoulders.

The patient is also asked to raise the shoulders and arms, join the hands with palms facing each other, and try to hold them in this position. The doctor should note if one arm falls or the shoulder lags.

During this, the muscle strength is checked and assessed using a five-point scale.

Symptoms of damage to the accessory nerve

When the lower anterior central gyrus is affected, there may be Salaam convulsions, frequent and rapid head tilts forward, or spastic torticollis.

In unilateral affections of the central neuron, no clinical symptoms are detected because the healthy side provides innervation to this muscle group.

In unilateral affections of the peripheral neuron, peripheral paresis of the accessory nerve develops, which manifests itself on the affected side; the patient cannot turn the head, raise the shoulder, lift the arm, and take it aside, torticollis may develop, atrophy of the trapezius and sternocleidomastoid muscles, and fibrillary twitching. The symptom of a drooping head manifests bilateral affections of the peripheral neurons in the upper cervical region[27, p.44]. (Diagram 13).

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
Motor fibers **1 st neuron:** ➢ **Lower section of the anterior cortical gyrus of the cerebral cortex** in the medulla oblongata makes a partial crossing over the nuclei 50% by 50% **2nd:** ➢ n cerebralis – в продолговатом мозге ➢ n spinalis C1-C5 – в передних рогах спинного мозга ➢ Иннервирует мышцы: m.sternocleidomastoideus, m.trapesius	➢ Palpate relevant muscles to check trophicity and tone ➢ Explore the volume of active movements of the arms and head ➢ Research tests: - for raising arms, shoulders -up -raising to the sides with hands ➢ Examine the strength of the muscles of the arms, upper shoulder girdle	Atrophy Hypotrophy Fibrillary twitching paresis of the corresponding muscles inability to perform tests The "hanging head" symptom Saalam's seizures	Second neuron lesion Bilateral lesion of 2 neurons Irritation of the inferior part of the anterior central gyrus on both sides

Diagram 13. Accessory nerve
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

4.9. Hypoglossal nerve

The hypoglossal nerve is part of the corticonuclear pathway and has a two-neuron structure.

The centrally located **first neuron** is located in the lower parts of the anterior central gyrus.

The axons descend through the internal capsule, then through the entire brainstem, and reach its lower portion, specifically the medulla oblongata.

Above the nucleus of the hypoglossal nerve, it undergoes a complete 100% decussation, unlike all other cranial nerves.

The **second neuron**, which is peripheral, is located in the nucleus of this nerve in the medulla oblongata.

The axons of the second neurons exit the brain through their own respective canal and reach the muscles of the tongue. The hypoglossal nerve controls movements of the tongue during chewing, speaking, and articulating speech.

Methods of examination of the hypoglossal nerve:

- When speaking with the patient, attention is paid to their ability to speak correctly and clearly and pronounce speech without dysarthria.

- During the examination, the patient is asked to open their mouth widely, and the position of the tongue in the oral cavity at rest is assessed, paying attention to its condition, trophicity, relief, whether the tongue is positioned in the middle, and if there are any fibrillary twitchings on the tongue, and others
- The patient is asked to protrude their tongue from the mouth and hold it along the midline; attention is paid to any deviation, atrophy, fibrillary twitchings, and others

Symptoms of hypoglossal nerve affections:

Central paresis of the hypoglossal nerve indicates damage to the central neuron and its axon at any level before the medulla oblongata. It is characterized by **dysarthria**, **impaired tongue participation in chewing, and deviation of the tongue** to the opposite side of the lesion.
Furthermore, since this is a central paresis, there will be no fibrillary twitching or atrophy of the tongue.

Peripheral paresis of the hypoglossal nerve is damage to the second neuron at any level from the medulla oblongata to the target tongue muscles.
It manifests with dysarthria, impairment of the chewing process, deviation of the tongue towards the pathological focus, **atrophy, and fibrillary twitching on the affected side of the tongue.**

The **alternating Jackson syndrome** involves a pathological focus located in the medulla oblongata on one side with the involvement of the nucleus of the hypoglossal nerve and the corticomuscular pathway. Clinically, it manifests as peripheral paresis of half of the tongue on the affected side, while on the opposite side, there is central spastic hemiparesis [27, p.46]. (Diagram 14)

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
Motor fibers **1st:** Inferior section of the anterior central gyrus of the cerebral cortex in the medulla oblongata makes a complete crossing over the nuclei **2nd:** ➢ nuclei in the medulla oblongata ➢ Innervates the muscles of the tongue	➢ Ask the patient to open the mouth; assess the position of the tongue in the mouth ➢ Ask the patient to: stick the tongue out of the mouth; assess the position of the tongue in the oral cavity ➢ Examine the amount of tongue movement ➢ Examine the trophics of the tongue ➢ Presence of fibrillary twitches on the tongue ➢ Explore the involvement of the tongue in speech and chewing	➢ Central paresis: deviation of the tongue in the opposite direction of the focus dysarthria Peripheral paresis: deviation of the tongue in the direction of the lesion fibrillary retractions atrophy of half of the tongue dysarthria Jackson's Alternating Syndrome	Lesion of neuron 1 Lesions of the second neuron second neuron nucleus and corticomuscular tract

Diagram 14. Hypoglossal nerve
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

4.10. Syndromes of multiple cranial nerve affections

Sphenoidal fissure syndrome: symptoms of dysfunction of the nerves passing through this fissure develop: cranial nerves II, III, IV, V, and VI: ophthalmoplegia, hypoesthesia in the innervation zone of the first branch of the trigeminal nerve, complete or partial blindness on the affected side.

Anterior cranial fossa lesion syndrome - Foster-Kennedy syndrome: consists of symptoms of functional disorders on the side of the I and II pairs of cranial nerves on the affected side: hyposmia or anosmia, amaurosis or amblyopia, optic nipple atrophy; optic disc congestion on the opposite side.

Middle cranial fossa lesion syndrome is a complex of symptoms formed by the dysfunctional symptoms of cranial nerves III, V, and VI on the affected side, namely ptosis, mydriasis, and divergent or convergent strabismus, as well as hypoesthesia on one side of the face.

Posterior cranial fossa lesion syndrome consists of symptoms affecting the nerves on the affected side - VII, VIII, IX, X, XII, and the cerebellum: peripheral paralysis of the facial nerve, hypoacusis or anacusis, paralysis of the soft palate, decreased or absent swallowing reflex, hypophonia, dysphagia, deviation of the tongue with atrophy of half of the tongue and fibrillary twitching, and cerebellar ataxia.

Cerebellopontine angle syndrome consists of symptoms indicating the loss of functions of the following nerves: V, VII, VIII cranial nerves: peripheral paralysis of the mimic muscles, hypoacusis or anacusis, hypoesthesia in half of the face, and cerebellar ataxia, all of which occur on the affected side.

Bulbar palsy: *The pathological focus is located bilaterally in the motor nuclei of the medulla oblongata involving the IX, X, and XII cranial nerves***.** It is characterized by peripheral paresis of the muscles innervated by these nerves. It manifests as a speech disorder, which becomes slurred (*dysarthria*), may be completely lost (*anarthria*), voice resonance changes (*dysphonia*), swallowing disorder (dysphagia), the patient chokes when eating, and food may regurgitate through the nose. The tongue muscles are atrophied, and fasciculations or fibrillations are observed. The soft palate is drooping, and the palatal and swallowing reflexes are absent.

Pseudobulbar palsy *develops with bilateral supranuclear lesions of the corticonuclear pathways.* It is characterized by the absence of muscle atrophy, increased mandibular reflex, oral automatism reflexes, and involuntary laughter and crying. [4, p.70].

Questions on the topic "Cranial nerves":

1. What alternating brainstem syndromes do you know?
2. What is the difference between bulbar syndrome and pseudobulbar syndrome?
3. Ophthalmoplegia syndrome.
4. Argyll-Robertson syndrome and reverse Argyll-Robertson syndrome.

5. CEREBRAL CORTEX

5.1. Anatomical data of the cerebral cortex

The cerebral cortex consists of 14 billion neurons and covers the hemispheres of the brain. The hemispheres are divided by a deep fissure that extends to the corpus callosum, which connects both hemispheres. In each hemisphere, there are distinct frontal, parietal, temporal, and occipital lobes. The surface of each hemisphere has numerous sulci that divide the lobes and gyri. The largest sulci are the central sulcus (Rolandic fissure) separating the frontal lobe from the parietal lobe, the lateral sulcus (Sylvian fissure) separating the frontal and parietal lobes from the temporal lobe, and the parieto-occipital sulcus separating the parietal lobe from the occipital lobe. The cerebral cortex is divided into the new, ancient, old, and intermediate cortex.

The new cortex (neocortex) occupies about 96% of the entire surface of the cerebral hemispheres and includes the occipital, inferior parietal, superior parietal, postcentral, precentral, frontal, temporal, insular, and limbic areas.

The cortex has six layers:

1 - Molecular layer
2 - External granular layer
3 - External pyramidal layer
4 - Internal granular layer
5 - Internal pyramidal layer
6 - Multiform layer

The cortex is divided into areas, subareas, fields, and subfields.

Functionally, the cortex is divided into projection and association fields. Projection fields are connected to the periphery via specific conducting pathways. Association fields are not directly connected to the periphery but have extensive connections with projection fields and subcortical centers. They are involved in processing incoming information.

The most important projection fields:

Precentral gyrus: It serves as the origin of the motor analyzer and is responsible for regulating voluntary movements. Due to the presence of decussation in the pyramidal tract, the precentral gyrus is connected to the opposite side of the body. The projection of the body onto the precentral sulcus is inverted.

Postcentral gyrus: It represents the cortical end of the sensory analyzer. Similar to the precentral sulcus, it has contralateral connections to the periphery, and the projection is inverted.

Heschl's Gyrus: It represents the cortical end of the auditory analyzer. Given the presence of incomplete decussation (50% by 50%), the connection with the inner ear is bilateral.

The gyrus hippocampi and the cornu ammonis represent the cortical end of the olfactory and gustatory analyzers, with connections existing bilaterally.

The occipital lobe, along with the calcarine sulcus, cuneus, and lingual gyrus, constitutes the cortical end of the visual analyzer.

The right visual center is connected to the left halves of the retinas of both eyes and provides the left visual fields, and vice versa.

The most important association fields:

Field 44 - Broca's area - responsible for expressive motor speech, located in the lower parts of the dominant hemisphere's frontal lobe.

Field 22 - Wernicke's area - provides impressive speech, located in the posterior parts of the dominant hemisphere's superior temporal gyrus.

Fields 39-40 - the center of semantic speech located at the junction of the parietal, temporal, and occipital lobes in the dominant hemisphere.

The mnestic speech center, located in the posterior parts of the dominant hemisphere's temporal lobe - field 37.

The writing center - in the posterior parts of the second frontal gyrus in the dominant hemisphere - field 6.

The reading and counting center, in the lower parietal lobe in the area of the angular gyrus in the dominant hemisphere.

The tactile gnosis center in the lower parietal lobe has contralateral connections to the periphery.

Praxis centers are located in the parietal lobe, the corpus callosum, and in the frontal lobes.

The nondominant hemisphere's parietal lobe is responsible for spatial gnosis.

The human abilities to speak and think, perceive objects (gnosis), and act with objects (praxis) are primarily ensured by the highly developed cortex of the cerebrum [6, p.185].

If we adopt a system of topically significant relationships, we can carry out a neuro-psycho-linguistic differentiation of focal lesions of the cerebral cortex at different functional levels.

Interhemispheric relationships. The development of lateralization of hemispheric centers is a distinctive feature of the human brain. In right-handed people, the left hemisphere is responsible for logical and analytical functions mediated by words. The right hemisphere is more specialized in general perception and emotional reactions necessary for survival and interaction with the external environment. The left hemisphere specializes in forming correct verbal responses. The brain simultaneously operates different areas of both hemispheres. The left (dominant in right-handed individuals) hemisphere, which controls executive functions, may ignore conflicting information from the right hemisphere; however, this does not prevent the accumulation of information by the left hemisphere for later use. [29].

5.2. Methods of examination of the functions of the cerebral cortex

Examination of expressive speech:

• Spontaneous speech. The patient is asked to describe their illness, work, or family. If the patient cannot perform this task, the examination is conducted through a dialogue: the patient should provide a brief answer to a specific question regarding their illness, work, or family.
• Repeated speech. Repetition of vowels and consonant sounds one by one, in pairs. It is necessary to select sounds that are similar in place or manner of articulation, for example: "b", "p", "g", "k" - plosive, "sh", "z", "shch", "kh" - fricative, "t", "d", "n", "l" - predental, "m", "p", "b" - bilabial. Pairs of consonant sounds for repetition: "b-p", "t-d", "g-k", "l-l", "l-k", "m-n", "l-n". Pairs of consonant syllables: "ba-pa", "da-ta", "ta-ka", "to-do", "ra-la", "ra-la-na", "da-ta-la". Repeating simple (for example, "izba", "les", "kholod", and others) and more difficult words for articulation (for example, "polkovnik", "polovnik", "portnoy", "korablekhrusheniye"). Repeating phrases (for example, "A plane is flying in the sky") and tongue twisters ("Three grey rabbits in the grass. Grow roses for us").
• Automated speech. Counting, enumerating days of the week, months (in forward and backward order).
• Naming objects shown in pictures with their images, naming actions in the pictures.

Examination of impressive speech:

• Phonemic hearing. The patient is offered to distinguish similar phonemes: "ba-pa", "da-ta", "sa-za" with a preliminary instruction to repeat them if the patient's expressive speech is not impaired, or to raise the right hand on the syllable "ba", "da", "za" (voiced) if speech motor function is impaired.
• Understanding the meaning of words. The patient is asked to show the pictures or body parts (nose, eye, ear) named by the doctor one by one or in pairs (pie-phone, ear-nose). The patient is asked to explain the meaning of words such as "caterpillar," "pillar," "barrel," "bar," and "bud."
• Understanding the meaning of complex logical-grammatical constructs: comparative ("Olya is darker than Sonya, but lighter than Katya. Who is the lightest?"), reflexive ("Does the Earth light up the Sun or does the Sun light up the Earth?"), attributive ("Is father's brother and brother's father the same?").
• Understanding and executing simple and complex instructions. ("Tap the table 3 times, put the pencil under the book, touch the left ear with the index finger of the right hand," and others).

Examination of written speech - the patient is asked to write a text**.**
Examination of reading - the patient is asked to read the text aloud.
Examination of calculation - the patient is asked to perform arithmetic operations.

Examination of praxis and gnostic functions is performed according to the following scheme:

1. Reproduction of finger positions.
2. Oral praxis (stick out the tongue, touch the corners of the mouth, upper and lower lips with the tongue).
3. Examination of the dynamic organization of the motor act: fist-palm-rib test, finger I-II-I-V. Drawing from the example.
4. Spatial and constructive praxis. Head's test (the patient confuses the frontal and sagittal planes, right and left sides). Making geometric figures from a set of sticks (matches).
5. Reproducing gestures: showing how to threaten with a finger, waving goodbye, beckoning.
6. Reproducing actions with imaginary and real objects.
7. Recognizing object images, plot pictures, faces of people of different nationalities, colors, letters, and images composed of two halves of different animals [30-33].

5.3. Symptoms and syndromes of cortical function lesions

Aphasia is an acquired speech function disorder characterized by the loss of the ability to use speech for communication, for symbolically expressing thoughts and feelings, or by the complete or partial loss of the ability to understand speech. Aphasia is caused by damage to the brain's dominant hemisphere (typically the left hemisphere in right-handed individuals) cortex in the absence of disorders affecting the articulatory apparatus and hearing.

Different areas of the brain's dominant hemisphere are distinguished, damage to which can lead to aphasia: the anterior part of the brain, specifically the **premotor** area in the inferior frontal gyrus (cortical **areas 44, 45**), damage to which leads to "anterior aphasia," and the posterior regions of the brain - the posterior part of the superior temporal gyrus (**area 22**), and at the junction of the parietal, temporal, and occipital lobes - **areas 39, 40**, which are the causes of "posterior aphasia."

The structure of speech primarily comprises two processes: the pronunciation of words and the perception of speech. The impairment of the pronunciation process of words is called motor, expressive aphasia, and the impairment of speech perception is called impressive aphasia.

Motor aphasia occurs with damage to the lower parts of the premotor area, Broca's area, areas 44 and 45. There are three forms of **expressive** speech disorders: afferent, efferent, and dynamic **motor aphasia**.

Afferent motor aphasia occurs when the patient has difficulty in articulation, especially the so-called homorganic sounds, similar in place (for example, predental: "t," "d," "l," "n") or manner (fricative: "sh," "z," "shch," "kh") of articulation.

Efferent motor aphasia. In contrast to afferent motor aphasia, the articulation of individual sounds is not impaired. The defect concerns switching from one speech unit (sound, word) to another. The kinetics of the speech process are disturbed due to difficulties in switching and disorders of speech mechanisms - perseveration (repetition of words and phrases).

Dynamic motor aphasia occurs in prefrontal lesions anterior to Broca's area. The central defect of this form of aphasia is the impairment of active, productive speech. The patient cannot actively express a thought or ask a question.

Impressive speech disorders manifest themselves in two primary forms: sensory aphasia and semantic aphasia.

Sensory aphasia occurs when Wernicke's area (area 22) is damaged. Phonemic hearing impairment is at the root of speech comprehension disorders. A patient with sensory aphasia cannot repeat syllables such as "ba-pa," "ta-da," or "sa-za." Unable to understand the speech of others, the patient strives to speak all the time.

Semantic aphasia occurs due to damage to the left hemisphere's tertiary areas (39, 40). At the root of this aphasia is a disorder of spatial synthesis, resulting in the patient's inability to understand speech formulations reflecting spatial relations. For example, the patient cannot correctly follow the instructions, "Draw a circle under the square" and "Draw a triangle above the circle."

Amnestic aphasia and **anomic aphasia** are characterized by difficulty in naming objects while retaining the ability to characterize them. Patients struggle with word retrieval, cannot recall the names of objects, and often express themselves using verbs. For example, instead of "pen," they may say: "the thing used for writing." And they often say "you know, like... you know?"

The lesion focus is in the temporoparietooccipital area.

Aprosodia is an impairment in the ability to speak melodically, expressively, emotionally, or to understand the melody of speech (centers correspond to Broca's and Wernicke's areas, right side only)[28]

Agraphia is an impairment in the ability to write, resulting from a focal lesion of the cortex of the brain's dominant hemisphere frontal lobe.

Alexia is a reading disorder caused by impairment in understanding written text due to damage to the left angular gyrus in the parietal lobe (area 39).

Acalculia is an impairment in the ability to perform arithmetic calculations, caused by damage to the dominant hemisphere's parietal lobe (area 39).

Apraxia is the loss of the ability to perform previously well-known, automated, purposeful actions while retaining the elementary movements that make up those actions. It occurs when the parietal-temporal divisions' cortex or the corpus callosum's conductive pathways are affected.

Ideational apraxia is the inability to perform a sequence of learned multi-step actions (for example, making coffee, cooking, or sending a letter). It arises from diffuse brain damage and damage to the frontal lobe.

Ideomotor apraxia is the inability to perform familiar learned actions upon command (for example, raising the hand, clenching fists, adding and stirring sugar with a spoon in a glass) with the lesion located in the lower part of the parietal lobe of the dominant hemisphere.

Kinetic apraxia is a loss of dexterity and coordination in performing fine movements with the distal parts of the extremities, associated with damage to the contralateral motor cortex.

Movements and actions are performed within a system of spatial coordinates, which can be disrupted by lesions in the inferior temporal lobe (areas 39, 40). In this case, spatial apraxia can occur, which can be identified using two-handed Head's tests: the patient confuses the frontal and sagittal planes, the right and left sides, and has difficulty constructing geometric figure from individual parts, such as a triangle or square (constructive apraxia). Constructive apraxia is a difficulty in copying figures and drawings and is associated with damage to the right temporal lobe. In severe cases, a harsher form of spatial apraxia may develop, such as dressing apraxia, which involves difficulties in dressing due to an inability to orient the body axis and extremities relative to clothing openings. In this case, the focal lesion is typically in the parietal lobe of the non-dominant hemisphere.

Agnosia is characterized by the inability to recognize sensory stimuli from external sources and within one's body while retaining sensitivity and consciousness.

Agnosia is classified depending on the stimulus: visual, auditory, olfactory, taste, tactile, anosognosia, and autopagnosia.

Visual agnosia. In this case, the patient can see but cannot recognize what they see. The lesions are localized in the occipital-parietal lobes.

Color agnosia, or cerebral achromatopsia, is an acquired disorder of color vision that can also develop with lesions in the parietal and inferior temporal lobes. The combination of color agnosia and letter agnosia is encountered in cases of left-hemispheric temporal-occipital lesions. In this case, characteristic difficulties include recognizing and sorting threads of wool or pieces of colored cardboard into color categories (color agnosia), as well as identifying similar letters in terms of their shapes, such as "n", "g", "p", "y", or "v," "r", "b", "d" (letter agnosia).

Visual-spatial agnosia (predominantly due to left-hemispheric lesions in the inferior parietal and parietal-occipital regions). It is characterized by difficulties in determining the position of clock hands, the right and left sides of objects, comparing two figures with specific spatial arrangements of elements, and others. In cases of similar right-hemispheric lesions, fragmented perception of spatially oriented objects is characteristic, along with impaired topographical memory leading to failure in recognizing familiar streets and rooms and "ignoring" the left side of visual space.

Auditory agnosia (focal lesions of the superior temporal gyrus). The patient can hear but cannot recognize previously familiar sounds and is experiencing difficulty recognizing object-related sounds (rustling of crumpled paper, tapping of chalk on a board or spoon, stirring tea in a cup, train whistle, and others). In the presence of verbal auditory agnosia (lesion in the Wernicke's area), difficulties arise in recognizing sounds of native language, leading to impaired understanding of spoken language with a sense of detachment from the meaning of words and difficulties in repetition.

Tactile-kinesthetic agnosia, or **astereognosis**, develops with focal lesions in the parietal lobe. The patient cannot recognize familiar objects when touching them with closed eyes.

Simultaneous agnosia is the inability to recognize complex visual images or scenes despite recognizing individual elements. The lesion is located in the superior occipital-parietal region.

Prosopagnosia, also known as agnosia for faces, can be caused by bilateral lesions in the occipital-parietal region affecting both fusiform gyri. The patient cannot recognize familiar faces.

Olfactory agnosia is a condition in which the patient cannot recognize familiar odors; they can smell but cannot identify them. The focal lesion is located in the temporal lobe in the region of the hippocampus.

Anosognosia is a condition in which the patient denies their illness; it occurs predominantly with lesions in the right parietal region.

Autotopognosia is a disorder of recognizing the pattern of one's own body; it occurs with lesions in the right parietal region.

Topographical agnosia is the inability to orient oneself in complex multi-element systems; it predominantly occurs with lesions in the right parietal-occipital region or the temporal-parietal regions on both sides.

Ignoring half of the space or losing attention to half of the space predominantly occurs with lesions in the right parietal region, less commonly in the left frontal and left parietal regions [4, p.77].

Symptoms of cortex irritation (positive symptoms +symptoms):

Hallucinations or aura before an epileptic seizure

- *Contralateral Jacksonian motor variant of epilepsy seizures and opercular seizures - irritation of Frontal lobe*
- Contralateral sensitive variant of Jacksonian epilepsy - *irritation of parietal lobe*
- Gustatory, auditory, olfactory hallucinations - *irritation of temporal lobe*
- Visual hallucinations - *irritation of occipital lobe* [34-36] (Diagram 15).

Questions on the topic "Cerebral cortex":

1. The structure of the cerebral cortex.
2. What deficit symptoms occur when the cerebral cortex is damaged?
3. What positive syndromes occur when the cerebral cortex is irritated?

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
Frontal lobe ➢ **Anterior central gyrus** - motor area ➢ **The parietal lobe** Posterior central gyrus - sensory area ➢ **Temporal lobe** - Heschl's gyrus is the auditory zone. - Uncus hippocampi and Ammon's horn - olfactory and gustatory zone ➢ **Occipital lobe** - Sulcus calcarinus, cuneiform and lingual gyrus - Visual area **Associative fields** Field 44 is Broca's Center, Field 22 Wernicke's Center, Field 39-40 is the center of gnosis, praxis, reading, counting, and stereognosia, and Field 6 is writing	➢ Explore motor skills To explore sensitivity: -supernasal -deep -complex ➢ Speech research: -impressive -expressive - mnestic ➢ Examine praxis ➢ Explore reading, writing ➢ Explore counting ➢ Explore gnosis: auditory, gustatory, visual, olfactory, tactile gnosis, spatial gnosis, gnosis of one's own defect. ➢ Rotation of the head and eyes to the sides	Hemihypesthesia-paresthesia- posterior central gyrus Hemiparesis-convulsions-anterior central gyrus Motor aphasia (Broca Center (44 field)) Sensory aphasia (Wernicke Center) (22 field) Semantic aphasia-field 39, 40 Amnestic aphasia - field 37, 39, 40. Apraxia - inferior parietal lobe, frontal lobe Acalculia - parietal lobe Agraphia-- frontal lobe. Astereognosia - inferior parietal lobe Anosognosia - parietal lobe. Autotopognosia - parietal lobe Auditory agnosia is Heschl's gyrus. Auditory hallucinations, aura - Heschl's gyrus irritation Olfactory agnosia - hippocampus and amon's horn Taste hallucinations, aura - hippocampal irrigation Visual agnosia, hemianopsia - occipital lobe Photopsias, visual hallucinations, aura - occipital lobe irritation Head and eye rotation to the side of the lesion - frontal lobe	

Diagram 15. The cerebral cortex
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

6. AUTONOMIC NERVOUS SYSTEM (ANS)

6.1 Anatomical data of the autonomic nervous system

The autonomic nervous system regulates the activity of all internal organs and systems, maintaining the body's homeostasis and tissue trophism.

However, the autonomy of the autonomic nervous system is not entirely absolute; it is under the control of the cerebral cortex. A close anatomical and functional relationship exists between the autonomic and somatic parts of the nervous system. Autonomic nerve fibers are present in the cranial and spinal nerves. Both central and peripheral divisions are distinguished within the autonomic nervous system, including suprasegmental and segmental structures and sympathetic and parasympathetic components.

The ANS operates through reflex arches: the spinal-rostral-mesencephalic (segmental), diencephalic-hypothalamic-limbic (first suprasegmental), and subcortical-cortical (second suprasegmental) ones.

The main difference between the sympathetic and parasympathetic components lies in the functional innervation characteristics and their response to agents affecting the autonomic nervous system. The sympathetic component is stimulated by adrenaline, while the parasympathetic component is stimulated by acetylcholine. Ergotamine exerts an inhibitory effect on the sympathetic component, while atropine affects the parasympathetic component.

The **sympathetic component** of the autonomic nervous system has structures located in the cerebral cortex, hypothalamic nuclei, brainstem, reticular formation, and in the spinal cord (in the lateral horns). From the cells of the lateral horns of the spinal cord at levels from CVIII to LII, peripheral structures of the sympathetic component originate.

These include preganglionic fibers, the sympathetic trunk with ganglia, postganglionic fibers, para- and pre-vertebral ganglia, and intramural ganglia. The most important ones in practical terms include the ciliospinal center of Budge (C8-T2), the posterior cervical sympathetic nerve (Frank), the sinuvertebral recurrent nerve (Luschka), and the autonomic peripheral ganglia of the face (ciliary, pterygopalatine, and otic). The sympathetic trunk runs along the lateral surface of the spine and consists of 22-24 pairs of sympathetic ganglia: 3 cervical, 10-12 thoracic, 5 lumbar or abdominal, and 4 sacral ones.

Transmittance: Acetylcholine is released at the endings of preganglionic fibers of the SNS, while sympathin (adrenaline and noradrenaline) is released at the endings of postganglionic fibers, stimulated by hormones from the adrenal glands and thyroid gland.

The SNS promotes pupil dilation, palpebral fissure widening, mucus secretion reduction, tachycardia, increased BP, bronchial dilation, atonia, decreased sweating, and constriction of skin blood vessels.

The **parasympathetic component** of the autonomic nervous system. There are mesencephalic (midbrain) and bulbar divisions in the brainstem and sacral divisions in the spinal cord. The mesencephalic division includes the cells of the cranial nerves: III pair - the accessory nucleus of Yakubovich (paired, small-celled), which innervates the muscle that constricts the pupil; the Perlea nucleus (unpaired, small-celled) innervates the ciliary muscle involved in accommodation. The bulbar division comprises the superior and inferior salivatory nuclei (VII and IX pairs); the X pair - a vegetative nucleus that innervates the heart, bronchi, gastrointestinal tract, its digestive glands, and other internal organs. The sacral division is represented by cells in the SIII–SV segments, the axons of which form the pelvic nerve, innervating the urogenital organs, sigmoid colon, and rectum.

Transmittance: Acetylcholine is synthesized in pre- and postganglionic fibers, stimulated by hormones from the pancreas and parathyroid glands, promoting constriction of the pupil and palpebral fissure, increased salivation and sweating, bradycardia, decreased BP, enhanced peristalsis, intestinal spasms, increased urination, and dilation of skin blood vessels.

Suprasegmental section

The hypothalamic-limbic-reticular complex (HLRC) provides autonomic support for the appropriate adaptive activity.

The dual innervation of organs of the sympathetic and parasympathetic divisions exerts a synergistic effect, which is controlled by the suprasegmental level.

Suprasegmental structures are divided into ergotropic and trophotropic systems. The ergotropic system is active and accompanied by increased mental, motor, and autonomic functions, while the trophotropic system is passive, coinciding with rest periods. Both systems work synergistically. During ergotropic system activity, catabolic processes occur, mobilizing the sympathoadrenal apparatus, whereas, during trophotropic system activity, anabolic processes occur, mobilizing the vagoinsular complex. The coordinated functioning of these two systems is aimed at maintaining homeostasis. Homeostatic parameters can fluctuate within different ranges, within acceptable limits. These include arterial blood pressure, heart rate, blood sugar levels, pH, and others. The suprasegmental division aims to maintain homeostatic balance under any conditions, demands, or loads. The hypothalamus contains osmo-, glyco-, and thermoreceptors that perceive shifts in the body's internal environment. Hypothalamic nerve cells have high specific sensitivity to vital homeostatic parameters. The hypothalamus comprises 32 highly differentiated nuclei, the most studied of which are the supraoptic nucleus, responsible for mineral metabolism; the paraventricular nucleus, responsible for carbohydrate metabolism; the tuberal nucleus, responsible for temperature regulation; and the ventromedial and dorsomedial nuclei, responsible for endocrine regulation. The Lewis nucleus is responsible for sweat secretion. The hypothalamus regulates autonomic functions, temperature, water-salt, carbohydrate, fat, protein metabolisms, endocrine glands, internal organs, and cardiovascular and respiratory systems.

The hypothalamus is considered the center of autonomic-somatic integration since it cooperates with both autonomic and somatic synergies.

The limbic brain comprises the medial basal surface of the temporal and frontal lobes, hippocampus, olfactory bulb, olfactory tract, piriform, and cingulate gyrus. The primary function of the limbic brain is emotional, as it contains centers for pleasure and punishment. Damage to the medial basal areas of the temporal lobe by any pathological process can lead to depression, euphoria, anger, sexual and psychomotor excitement. The motivational-mnestic function also refers to the limbic brain.

The reticular formation is located in the tegmentum of the brainstem, occupying 3/5 of the stem. It connects the limbic brain and hypothalamus and contains activating and inhibitory fibers going in different directions. Ascending activating fibers help maintain cortical tone, which may affect perception, attention, and memory levels. Sleep and wakefulness, with corresponding autonomic support, depend on the state of the reticular formation. Descending fibers regulate muscle tone at the spinal level.

The reticular formation is considered an "energy generator" [6, p.240].

6.2. Methods of examination of the autonomic nervous system

For examination of the baseline autonomic tone, special tables are used, which contain data specifying the subjective state as well as objective indicators of autonomic functions (nutrition, skin color, condition of sweat glands, body temperature, pulse, blood pressure, ECG, vestibular manifestations, respiratory function, gastrointestinal tract function, pelvic organ function, performance capacity, sleep, allergic reactions, characterological, personality, emotional characteristics, and others). Kerdo vegetative index (VI) is calculated using the formula:

VI = 100 * (1 - DAD/pulse), where

DAD is the diastolic arterial pressure, and the pulse is the heart rate. The VI value greater than zero indicates a predominance of sympathetic tone, while a negative value indicates parasympathicotonia.

After determining the state of autonomic tone, autonomic reactivity is studied in response to the influence of pharmacological agents or physical factors. Pharmacological agents such as adrenaline, insulin, mesatone, pilocarpine, atropine, histamine, and others are used for this purpose.

To assess the state of the autonomic nervous system, the following functional tests are used.

Cold test. At the beginning of the examination, the heart rate is counted while the patient is lying down, and arterial blood pressure is measured. After that, the palm of the other hand is immersed for 1 minute in cold water at a temperature of 4°C. Then, the hand is removed from the water, and blood pressure and pulse rate are measured every minute until the values return to the initial level. In normal conditions, this process typically takes about 2-3 minutes. If the blood pressure increases by more than 20 mmHg, the response is interpreted as pronounced

sympathetic; if it increases by less than 10 mmHg, it is considered moderate sympathetic, and if the pressure decreases, it is interpreted as parasympathetic.

Oculocardiac reflex (the Aschner–Dagnini reflex). Applying pressure to the eyeballs causes typically a decrease in heart rate by 6–12 beats per minute. If the number of heartbeats slows down by 12–16, this is interpreted as a sharp increase in the tone of the parasympathetic division. The absence of slowing down or even accelerating heartbeats by 2–4 per minute indicates an increase in excitability of the sympathetic system.

Reflexus Solaris. The patient should lie on their back while the doctor applies pressure with their hand on the upper part of the abdomen until they feel the abdominal aorta's pulsation. After 20-30 seconds, heartbeats should normally decrease by 4-12 beats per minute. Changes in cardiac activity are assessed in the same way as in the oculocardiac reflex.

Carotid sinus reflex (Czermak-Hering reflex).

Pressure is applied to the carotid sinus area in the upper one-third of the sternocleidomastoid muscle with two fingers (thumb and index) until the pulsation of the carotid artery is felt for 15-30 seconds, and the result is assessed in the same manner as in the oculocardiac reflex.

The orthoclinostatic test is used to determine the autonomic support of activity.

Orthoclinostatic reflex. The test is conducted in two stages. The patient, lying on their back, has their heart rate counted, and then they are asked to stand up quickly (orthostatic test). When a horizontal position changes to a vertical one, the heart rate increases by 12 beats per minute with a rise in arterial pressure of 20 mmHg. When the patient returns to the horizontal position, pulse and pressure readings return to the initial ones within 3 minutes (clinostatic test). The degree of acceleration of the pulse during the orthostatic test is an indicator of the excitability of the sympathetic part of the autonomic nervous system. Significant slowing of the pulse during the clinostatic test indicates increased excitability of the parasympathetic part.

Segmental autonomic formations are also examined.

Pilomotor reflex. The "goosebump" reflex is elicited by pinching or by applying a cold object (a test tube with cold water) or a cooling substance (cotton wool soaked in ether) to the skin of the upper arm or the nape of the neck. On the corresponding half of the chest, "goosebumps" appear as a result of the contraction of smooth arrector pili muscles.

Acetylsalicylic acid test. The patient is given 1 gram of acetylsalicylic acid with a glass of hot tea. Diffuse sweating appears. If the hypothalamic area is affected, asymmetry may be observed. When the lateral horns or anterior roots of the spinal cord are affected, sweating is disrupted in the innervation zone of the affected segments. When the transverse section of the spinal cord is affected, taking acetylsalicylic acid induces sweating only above the level of the lesion.

Skin thermometry. Electric thermometers are used. Skin temperature reflects the blood supply to the skin, an essential indicator of autonomic innervation. Areas of hyper-, normal, and hypothermia in symmetrical areas are identified. The difference

in skin temperature at 0.5 °C on symmetric areas is a sign of autonomic innervation disorders.

The **Shcherbak reflex** involves cooling the hand for 20 minutes in water at 32 degrees Celsius and warming it for 10 minutes to 42 degrees Celsius, reflected in changes in rectal temperature. Normally, the temperature immediately after the bath increases by 0.3-0.5 degrees Celsius, and after 30 minutes, it returns to the initial level.

Dermographism. The vascular reaction of the skin in response to mechanical irritation on the skin surface (hammer handle, blunt end of a pin) is examined. Typically, a red line appears at the site of irritation, the width of which depends on the state of the autonomic nervous system. Some individuals may have a line raised above the skin (elevated dermographism). When the sympathetic tone is increased, the line is white in color (white dermographism). Very wide lines of red dermographism indicate an increased parasympathetic nervous system tone. The reaction occurs in the form of an axon-reflex and is local in nature.

Reflex dermographism is elicited by irritation using a sharp object (running the tip of a needle along the skin). A line with irregular festooned edges appears. Reflex dermographism represents a spinal reflex. It disappears when the posterior roots, spinal cord, anterior roots, and spinal nerves are affected at the lesion level; it can be used for topical diagnosis. Above and below the affected area, the reflex is usually preserved [4, p. 83].

6.3. Symptoms and syndromes of autonomic nervous system dysfunction

Damage to the suprasegmental level - the HLRC - manifests as the development of autonomic crises: sympathetic-adrenal, vagoinsular, or mixed.

A **sympathetic-adrenal crisis** manifests as increased blood pressure, tachycardia, tremors, hyperthermia, anxiety and fear, chest pain, and cold extremities.

A **vagoinsular crisis** is characterized by decreased blood pressure, bradycardia, extrasystole, a sensation of heart fluttering, increased peristalsis, and weakness.

With predominant hypothalamic involvement:

Hypothalamic syndrome - disturbances in thermoregulation, metabolic, and endocrine disorders [6, p.242].

With predominant involvement of the **limbic system**, emotional, intellectual-mnestic, and motivational disorders prevail. Temporal lobe epilepsy, paroxysmal psychomotor agitation, and facial hyperkinesia may develop.

When the **reticular formation** is affected - myasthenic-like syndrome, pseudo-myopathic syndrome, akinetic mutism, spastic torticollis, and disturbances in sleep and wakefulness.

Damage to the peripheral branches of the autonomic nervous system:

Sympathalgic syndrome. It is characterized by severe pain, often described as burning, pressing, or throbbing, with a tendency to spread around the area of primary localization gradually. Pain is provoked and intensified by changes in barometric pressure and environmental temperature.

Vascular syndrome: marked by local changes in skin and mucous membrane coloration, local alterations in temperature, spasm or dilation of peripheral blood vessels, asymmetry, and instability of these symptoms.

Trophic syndrome: manifests as local tissue trophic disturbances (skin, mucous membranes, muscles, bones, joints) in various segments and with varying degrees of severity (skin dryness, brittle nails, trophic ulcers, bone fractures, arthropathies, tendovaginitis, ant others).

Visceral syndromes: depending on the segments affected, corresponding to specific organs, present with diverse symptoms. For example: C5-C8 - heart; Th5-Th6 - esophagus; Th3-Th10 - lungs; Th5-Th8 - stomach; Th7-Th9 - pancreas; Th6-Th10 - duodenum, liver, gallbladder; Th7-Th10 - spleen; Th9-Th11 - cecum; Th9-L1 - kidneys; Th12-S1 - appendages, uterus.

The right sympathetic trunk innervates the liver, gallbladder, cecum, and ascending colon. The left sympathetic trunk innervates the heart, spleen, pancreas, stomach, descending colon, and sigmoid colon. The small and large intestines receive bilateral innervation [6, p.260].

The Claude-Bernard-Horner syndrome

In lesions affecting the ciliospinal center of the lateral horns at the level of segments CVIII-ThI and cervical sympathetic ganglia, a triad of symptoms develops: ptosis, miosis, enophthalmos. It is often accompanied by decreased intraocular pressure and facial vessel dilation.

There may be impaired sweating and dysfunction of the pilomotor function, vessel dilation, and increased temperature in the face and neck. Due to decreased laryngeal muscle tone, hoarseness of voice and hypophonia may occur.

Affection of the upper cervical sympathetic ganglion leads to dilation of the palpebral fissure and pupil (mydriasis), exophthalmos, and reverse Claude-Bernard-Horner syndrome. It may also manifest as sharp pains in the face and teeth. [4, p.85]. (Diagram 16).

Questions on the topic "Autonomic nervous system":

1. The structure of the autonomic nervous system.
2. The sympathetic-adrenal crisis.
3. The vagoinsular crisis.
4. Damage to the suprasegmental level.

ANATOMY	RESEARCH TEQUNIQUES	LESION SYMPTOMS	TOPICAL DIAGNOSIS
Suprasegmental structures Limbic brain hypothalamus thalamus Mendal bodies Hippocampus Reticular formation	**Investigation of vegetative tone**: pulse, BP, respiratory rate, ECG Kerdo autonomic index: VI=1- (BP diast.: HR)*100 If greater than 0, parasympathetic tone. **Examine autonomic reactivity:**	ADS, SH Homeostatic disorders Metabolic disorders Dyshormonal systems Disorders of thermoregulation Sweating disorders	Hypothalamic and thalamic lesions
Segmental structures **Sympathetic trunk:** ➢ upper cervical ganglia ➢ middle cervical ganglia ➢ ganglii stellatum in the spinal cord in the ocular columns C8-L1 **Parasympathetic Division:** ➢ Cranial part: nuclei of 3,7,9,10 pairs of cranial nerves ➢ Sacral part: S1-S5 n.splanchici pelvic plexus	Ashner Danini ocular-cardiac reflex. Cermak-Gering sinocarotid reflex. Normal heart rate is 9 to 12. If more than 12, parasympathetic responsiveness increases. If less than 9, or even more frequent, sympathetic. **Investigate autonomic support of activity:** orthostatic test Examine dermagraphism. Examine the pilomotor reflex Thermometry Scherbik's reflex Cold test	Motivational disorders Emotional disorders Mnestic disorders Sleep disorders Myasthenia gravis-like symptoms Akinetic mutism Horner syndrome - arrhythmias, visceral pain, sympatalgia, vascular syndrome, trophic disorders Pupillary reflexes are decreased, salivation, lacrimation, visceral disturbances, bowel dysfunction, pelvic distress	Limbic Reticular formation Sympathetic system (gangliaC8-Th1, Th2-Th4,Th8-Th11, local peripheral vessels) Parasympathetic system: Midbrain, rhomboid fossa, vagus nerve, sacral area

Diagram 16. Autonomic nervous system
(Atantayeva E.B. «Основы топической диагностики в неврологии», 2022) [4]

7. TOPICAL DIAGNOSIS OF NERVOUS SYSTEM DAMAGE:

The brain

Damage to the hemispheres is characterized by irritation of the cortex or destruction of gray or white matter, manifested by symptoms of irritation or loss, respectively.

***Frontal lobe*:**

Destruction:

- Mental disorders - "frontal psyche"
- Frontal ataxia, Janiszewski's symptom, opposition symptom
- Paresis of head and eye turning in the opposite direction
- Central paresis of facial and hypoglossal nerves contralaterally
- Contralateral monoparesis or hemiparesis
- Motor aphasia, agraphia (dominant hemisphere)
- Aprosody (non-dominant hemisphere)
- Symptoms of oral automatism
- Hypo- or anosmia (base of frontal lobe)
- Irritation: Jacksonian epilepsy seizures, opercular seizures

Premotor cortex

- Irritation: Seizures starting with head and eye turning in the opposite direction
- Destruction: Clumsiness, slow movements, and speech, perseveration, motor freezing, apathetic-abulic syndrome, contralateral hemiparesis

Precentral gyrus

- Irritation: Contralateral Jacksonian motor variant of seizures
- Destruction: Depending on somatotopy – contralateral central monoparesis of the arm or leg, or hemiparesis, central paresis of facial and hypoglossal nerves

Parietal lobe

Postcentral gyrus

- Irritation: Contralateral sensitive variant of Jacksonian epilepsy
- Destruction: Paresthesia in the hand or foot, on half of the face, monohypesthesia, monohyperesthesia, monoanesthesia, depending on somatotopy, or contralateral hemihypesthesia

Upper parietal lobule

- Disorders of localization, discrimination, kinesthesia, stereognosis (astereognosis), body pattern (autotopognosis), denial of defect (anosognosia)
- Afferent paresis of the hand (clumsy movement)

Lower parietal lobule

- Apraxia (mainly non-dominant hemisphere)
- Autotopognosis (often non-dominant hemisphere)
- Alexia (dominant hemisphere)
- Semantic and amnestic aphasia (at the junction with the temporal lobe, dominant hemisphere)
- Acalculia (dominant hemisphere)

- Asteregnosis

Temporal lobe

- Irritation: Gustatory, auditory, olfactory hallucinations, development of a generalized seizure, petit mal epilepsy, psychic equivalents, hypnois state, state of "deja vu."
- Damage: Ataxia, amusia, sensory aphasia, aprosody, auditory and olfactory agnosia, memory disorders, drowsiness, apathy, depression, homonymous hemianopsia.

Occipital lobe

- Irritation: Visual hallucinations, visual auras.
- Homonymous hemianopsia, negative scotoma of opposite visual fields, metamorphopsia, photopsia, visual agnosia, amblyopia, amaurosis with preserved pupillary response to light.

Corpus callosum

- Memory impairment, inappropriate behavior, disorientation in place and time, Korsakoff syndrome, twilight consciousness disorders, lethargy, apathy, lack of initiative, decreased criticism, pseudobulbar palsy, apraxia, urinary incontinence, decreased intellect.

Internal capsule (contralateral)

- Homonymous hemianopsia
- Hemiplegia
- Hemianesthesia
- Central paralysis of facial and hypoglossal nerves

Thalamus (contralateral)

- Hemianesthesia
- Hemihyperpathy
- Hemihyperesthesia
- Hemiataxia
- Thalamic pain
- Homonymous hemianopsia
- Autonomic, trophic, and endocrine disorders

Hypothalamus

- Autonomic crises
- Endocrine and metabolic disorders

Limbic brain

- Emotional, motivational, intellectual and mnestic disorders
- Temporal lobe epilepsy, paroxysmal psychomotor agitation, facial hyperkinesias.

Reticular formation

- myasthenic-like syndrome
- pseudomyopathic syndrome
- akinetic mutism
- spastic torticollis
- sleep and wakefulness disorders

Basal ganglia

- Akinetic-rigid syndrome
- Hyperkinetic syndrome

Cerebellum

- Static and dynamic ataxia

Brainstem

- Alternating syndromes
- Bulbar palsy

Spinal cord

- Symptoms of damage to the posterior horns and commissure:
  Decreased or lost superficial sensitivity with preserved deep sensitivity (dissociated type)
  - Level C4-T10 - "half-jacket" type on the affected side.
  - Level C4-T10 in the commissure or with bilateral affection of the posterior horns - complete "jacket" syndrome.
- Symptoms of affection of the anterior horns - peripheral paralysis and fibrillary twitching in the muscles corresponding to the segments.
- Symptoms of affection of the lateral horns: autonomic, vasomotor, and trophic disorders; at the level of C8-T1, Claude-Bernard-Horner syndrome (ptosis, miosis, enophthalmos).
- Symptoms of affection of the lateral columns: central paresis on the same side below the level, pain, and temperature sensitivity disorders on the opposite side.
- Symptoms of affection of the posterior columns: decreased or lost deep sensitivity on the affected side below the level of the lesion, sensory ataxia, positive Romberg sign with closed eyes.
- Level C1-C4 - intramedullary process - deep sensitivity impairment, first in the lower extremities, then in the upper ones; the extramedullary pathological process starts from the upper extremities.
- Syndrome of half-cross-section spinal cord lesion in the thoracic region: central lower monoparesis, disturbance of deep sensitivity on the affected side, and disturbance of superficial sensitivity below the lesion level on the opposite side. Brown-Séquard syndrome.
- Symptoms of complete cross-section spinal cord lesion at the level of the upper cervical region C1-C4: paralysis of respiratory muscles, diaphragm, central tetraparesis, disturbance of all types of sensitivity below the chin in a spinal conductor type, pelvic disorders of a central type [38].
- Symptoms of complete cross-section spinal cord lesion at the level of the cervical enlargement: mixed tetraparesis, which includes peripheral upper paraparesis and central lower paraparesis; disturbances of all types of sensitivity below the level of the clavicle in a spinal conductor type; pelvic disorders of a central type; Horner's syndrome (ptosis, miosis, enophthalmos).
- Symptoms of complete cross-section spinal cord lesion at the T4 level of the thoracic region: lower paraplegia or paraparesis of a central type, disturbances

of all types of sensitivity below the level of the mammary glands in a spinal conductor type, pelvic disorders of a central type.

- Symptoms of complete cross-section spinal cord lesion at the T10 level of the lower thoracic region: lower central paraparesis, disturbance of all types of sensitivity below the umbilical level in a spinal conductor type, pelvic disorders of a central type (urinary and fecal urgencies), absence of the middle abdominal reflexes.
- Symptoms of complete cross-section spinal cord lesion at the level of the lumbar enlargement: lower peripheral paraparesis predominantly in the proximal parts of the legs, disturbance of all types of sensitivity below the level of the inguinal folds in a spinal conductor type, pelvic disorders.
- Epiconus (S1-S2): peripheral paraparesis of distal legs, a disorder of all kinds of sensitivity on the perineum, on the posterior-external surface of the shins and thighs.
- Conus: sensitivity impairment in the perineal and gluteal areas, impaired pelvic functions in a peripheral pattern (incontinence of urine and feces), absence of the anal reflex, bedsores in the sacral region.
- Cauda equina: L2-S5 roots are shooting pains in the anus, perineum, buttocks, legs, sensory disorders in the zone of innervation of the roots, peripheral paralysis of legs and perineal muscles, dangling feet, paretic gait steppage, incontinence type urinary disorders [39,40].

Peripheral nerve structures

- Anterior root: peripheral paresis of muscles to which fibers from this root go, loss of reflexes.
- Posterior root: disorders of all kinds of sensitivity in the zone of innervation of this root, on the extremities like "stripes," on the trunk like "belts," symptoms of tension of the roots, pain syndrome.
- Spinal intervertebral ganglion - disorders of all kinds of sensitivity in the zone of innervation of this node, on the extremities like "stripes," on the trunk like "belts," pain syndrome, herpetic rashes.
- Plexus - peripheral paresis in the muscles for which the plexus is responsible, for example, brachial plexus - mono paresis in the arm, a disorder of all kinds of sensitivity in the zone of innervation of this plexus by plexus type, autonomic disorders.
- Peripheral nerve - peripheral paresis of the muscles innervated by this nerve, violation of all types of sensitivity by mononeuritic type.
- Multiple nerves in the distal extremities - peripheral tetraparesis in the distal arms and legs, impairment of all types of sensitivity by polyneuritic glove-and-sock type [41,42].

CONCLUSION

The study of clinically significant features of the nervous system and the principles of topical diagnosis, based on the analysis of identified symptoms and syndromes during the neurological examination of patients, is essential for general practitioners, neurologists, neurosurgeons, oncologists, anesthesiologists, traumatologists, dentists, otorhinolaryngologists, ophthalmologists, and other medical specialists.

This content provides a concise overview of the anatomy of the central and peripheral nervous systems, examination techniques, and the symptoms and syndromes of functional and organic disorders. It also demonstrates and explains the relationship between the identified symptomatology and damage to specific neurological structures, offering suggested clinical diagnoses frequently encountered in topical disorders.

The ability of students to understand symptomatology and syndromology, to combine symptoms into syndromes, and to associate syndromes with specific pathologies in certain structures of the nervous system enables them to navigate the topical diagnosis of neurological damage and propose possible clinical diagnoses. These skills are critical for physicians of any specialty to provide timely emergency care and reduce the risk of mortality and disability, which are often encountered in neurology.

Knowledge of the methods for clinically examining the nervous system is key to achieving accurate topical and clinical diagnoses. Simplified, ready-made diagrams provided in each chapter outline specific steps in diagnostic algorithms and highlight precise tactics for further investigation. This approach fosters clinical reasoning skills among students, interns, residents, and young doctors.

The material is presented in a logical manner, detailed, and supplemented with updated information from modern sources. At the end of each chapter, diagrams are included to simplify and reinforce the presented content, and questions on the topic are provided for students to practice. Additionally, the guide concludes with test tasks and answer templates for self-assessment.

The academic manual is developed in accordance with the educational program, curriculum, and syllabus for undergraduate students in general medicine and intern doctors at the International Medical Faculty. Advanced study of clinical neurology during internships and residencies is not feasible without a foundational understanding of topical diagnostics in neurology.

The academic manual, *"Neurology: Topical Diagnostics,"* presented with methodological rigor and accessible content, serves as an effective primary and supplementary resource for general practitioners. It is particularly beneficial for those managing patients with comorbid or standalone neurological diseases in their daily practice.

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Tests:

Topic: Sensory area

1

The examination revealed reduction of all types of sensitivity in distal parts of hands and feet by "gloves" and "socks" type.
What type of sensation disorder is it?
A) Spinal posterior horn-segmental
B) Peripheral polyneuritic
C) Spinal commissural
D) Spinal conduction
D) Cerebral stem

2

A 23-year-old man complains of sensory distortion, noting that he perceives cold as warm and warm as cold.
What type of sensory impairment is it?
A) Hyperesthesia
B) Polyesthesia
C) Dysesthesia
D) Paresthesia
E) Hypesthesia

3

A 45-year-old man with neurosyphilis complains of poor coordination and shaky gait in the dark. Examination revealed instability in Romberg's test with closed eyes and decreased deep sensitivity in the legs.
What type of ataxia is most likely?
A) Dynamic cerebellar
B) Static cerebellar
C) Vestibular
D) Sensitive
E) Frontal

4

A 38-year-old man presents with complaints of balance and coordination disorders in the dark time of day, revealing a disorder of deep sensitivity in the legs; pain and temperature sensitivity is preserved.
Which structures are affected?
A) Spinothalamic tract
B) Peripheral nerve
C) posterior root
D) Gaulle bundles
E) Posterior horn

5
A 55-year-old woman, after a stroke, appeared to have severe pain in the left extremities and numbness in them. Objectively: hemihypesthesia, hyperpathy, hemianopsia on the left.
Where is the pathological focus located?
A) Frontal lobe of the brain
B) Internal capsule
C) Medulla oblongata
D) Optic tubercle
E) Pons

6
A 20-year-old girl complains of frequent attacks of numbness and "crawling of goosebumps" in the left side of the body, starting from the foot and spreading upwards.
Where is the pathological focus located?
A) Lower part of the right anterior central gyrus
B) Upper part of the left anterior central gyrus
B) Middle part of the right posterior central gyrus
D) Upper part of the right posterior central gyrus
E) Lower part of the left posterior central gyrus

7
A 30-year-old man with weakness, paresthesias, and shooting pains in the legs. Examination revealed loss of all types of sensitivity below the level of the umbilicus and in the legs.
What is the sensory disorder syndrome?
A) Mononeuritic
B) Polyneuritic
C) Conductor
D) Root
E) Cortical

8
The patient complains of pain and weakness in his right arm. Examination revealed the symptom of a "dangling" hand on the right, decreased sensitivity on the dorsal surface of the hand, and three fingers with half of the fourth finger on the right.
What is the sensory disorder syndrome?
A) Mononeuritic
B) Polyneuritic
C) Capsular
D) Root
E) Cortical

9
A 20-year-old girl with a history of multiple burns complains of a lack of painful sensations; examination revealed the absence of pain and temperature sensitivity in the "half-jacket" type zone on the left.
What is the sensory disorder syndrome?
A) Posterior horn-segmental
B) Mononeuritic
C) Polyneuritic
D) Capsular
D) Root

10
The examination revealed loss of pain and temperature sensitivity on half of the face on the right side; and loss of pain and temperature sensitivity on the trunk and extremities on the left side.
Where is the patient's pathologic focus most likely to be located?
A) Inner capsule on the left
B) Brain stem on the right
C) Brain stem on the left
D) Thalamus on the right
E) Cortex on the left

11
The patient complains of pain and temperature sensitivity disorder; examination revealed hypesthesia of superficial sensitivity within TIV-TVII dermatomes on both sides by the "belt" type (deep sensitivity is preserved).
Where is the affection focus most likely to localize?
A) Nerve
B) Root
C) Posterior horn
D) Anterior horn
E) Anterior gray commissure

12
A 30-year-old man presents with a "shooting" pain in the lumbar region after weight lifting, radiating down the posterior surface of the thigh and the posterior external surface of the shin and foot on the right side. Objectively: decrease in Achilles reflex and all types of sensitivity in the area of the pain localization.
Which tension sign is most likely to be positive?
A) Brudzinsky
B) Wasserman
C) Matskevich
D) Lasegue

E) Neri

13
A 45-year-old woman experiences Brown-Séquard syndrome after a spinal injury, exhibiting peripheral paresis in the left arm and central paresis in the left leg, with reduced deep sensitivity in these areas.
What type of sensitivity is lost on the patient's right side?
A) Discriminatory
B) Superficial
C) Vibratory
D) Kinesthetic
E) Complex

14
A 50-year-old woman complains of "burning" chest pain on the right side and herpes eruptions in the same area.
Where is the pathological focus located?
A) Spinal ganglion
B) Anterior root
C) Anterior column
D) Posterior column
E) Posterior horn

Topic: Motor area

15
The examination revealed dorsal flexion of the first toe and fan-shaped spreading of the toes of the foot when the hand was pressed on the crest of the patient's tibia.
Which of the pathologic signs is described?
A) Gordon
B) Schaeffer
C) Grossman
D) Oppenheim
E) Babinsky

16
The examination revealed hyperreflexia, hypertonia, and pathological plantar reflexes in the left leg. To detect paresis, the doctor asked the patient to raise his legs and hold them in this position for a few seconds.
Which test is most likely used by the doctor in this patient?
A) Stewart-Holmes

B) Upper Barré
C) Lower Barré
D) Romberg
E) Westphal

17
A 60-year-old man suffering from hypertension complains of weakness in his left extremities. Objectively: On the right side, decreased muscle strength up to 3 points, increased tone, hyperreflexia, and pathologic foot reflexes on the right side.
What syndrome is described?
A) Flaccid hemiparesis
B) Flaccid hemiplegia
C) Central hemiplegia
D) Central hemiparesis
E) Central lower paraparesis

18
The examination revealed limited active movements in the upper and lower extremities, increased muscle tone and tendon reflexes, and pathological reflexes in the feet.
What is the name of the syndrome of motor disorders?
A) Flaccid paraparesis
B) Flaccid tetraparesis
C) Combined tetraparesis
D) Spastic hemiparesis
E) Spastic tetraparesis

19
A 67-year-old woman who has atherosclerosis found weakness and numbness in her right extremities in the morning after sleeping. Objectively: the left nasolabial fold is smoothed, the tongue deviates to the left when sticking out, and central hemiparesis, hemianopsia, and hemihypesthesia are on the left.
What syndrome does the patient have?
A) Stem
B) Capsular
C) Thalamic
D) Meningeal

E) Segmental

20
The examination revealed no active movements in the legs, increased muscle tone in the legs, animated knee and Achilles reflexes, no muscle atrophy, clonus of the feet, and positive foot reflexes.
What part of the nervous system is affected?
A) Spinal cord above the cervical thickening
B) Peripheral nerves on both sides
C) Cervical thickening of the spinal cord
D) Spinal cord in the thoracic region
E) Lumbar thickening of the spinal cord

21
The examination revealed muscle atrophy of the upper extremities, decreased tendon reflexes, muscle strength, and fibrillary jerks in the arm muscles. In the legs, increased tendon reflexes and muscle tone of the spastic type, pathologic reflexes, and clonus in the feet.
What is the topical diagnosis?
A) Thoracic region
B) Lumbar spine
C) Sacral region
D) Cervical thickening
E) Upper cervical spine

22
The muscle tone is reduced in the distal arms and legs, hypotrophy in them, carporadial and Achilles reflexes are absent.
Which syndrome is most likely to be identified?
A) Peripheral tetraparesis
B) Peripheral hemiparesis
C) Peripheral paraparesis
D) Central tetraparesis
E) Central paraparesis

23
A 38-year-old man complains of weakness and thinning of muscles in the arms; he has been ill for about a year, and the weakness developed

gradually. Objectively: muscle strength in the arms reduced to 3 points, hypotrophy, hyporeflexia, and fibrillary jerks in the muscles of the arms.
Which examination method is most appropriate?
A) REG
B) EEG
C) USDG
D) ENMG
E) EchoEG

24
The examination revealed limited active movements in the lower extremities, decreased muscle tone and tendon reflexes, and muscle hypotrophy.
What is the name of the syndrome of motor disorders?
A) Flaccid paraparesis
B) Flaccid tetraparesis
C) Spastic paraparesis
D) Spastic hemiparesis
E) Spastic tetraparesis

25
A 40-year-old man developed paraparesis in the lower extremities, decreased tone in the legs, hyporeflexia, and hypotonia in them.
Which structures are most likely to be affected?
A) Broca's area
B) Visual tubercle
C) Reticular formation
D) Anterior central gyrus
E) Anterior horns of the spinal cord

26
A 43-year-old man complains of pain in the lumbosacral region of the spine and anterior surface of the thigh and weakness in the right leg. Objectively: lumbar lordosis is smoothed, positive Wasserman and Matskevich signs on the right side, hypoesthesia on the anterior surface of the right thigh, decreased right knee reflex.
Which structures are most likely to be damaged?
A) Lumbar plexus
B) Brain

C) Spinal cord
D) L2-L4 nerve roots
E) L5-S1 nerve roots

27
A 30-year-old man presents with complaints of weakness in the left leg for six months. Objectively: peripheral paresis in the left leg and decreased deep sensitivity in it, and in the right leg, decreased superficial sensitivity from the level of the inguinal fold.
Which structures are affected?
A) Ganglion
B) Posterior horn
C) Posterior root on the right
D) Half of the transverse section of the spinal cord on the right
E) Half of the transverse section of the spinal cord on the left

28
A 55-year-old man complains of numbness and pain and weakness in the right foot, and difficulty pressing the pedal while driving a car. Examination revealed a decrease in sensitivity in the area of the sole of the right foot, and weakness in the flexors of the foot is noted.
Which structure is affected?
A) Anterior central gyrus
B) Tibial nerve
C) Posterior root on the right
D) Posterior root on the left
E) Ganglion

Topic: Extrapyramidal system and cerebellum

29
The examination revealed a doll-like gait, fine tremors of hands like "counting coins," increased muscle tone in the extremities like "cogwheel," quiet and monotonous speech, hypomimia, bradypsychia.
Which syndrome is most likely in the patient?
A) Meningeal
B) Akinetic-rigid
C) Hyperkinetic
D) Cerebellar ataxia

E) Alternating syndrome

30
A 59-year-old woman complains of rest tremors in the hands, more on the right side, slow movement, and stiffness in the extremities, which have gradually increased for two years. Objectively: "shuffling" gait, bradykinesia, bradylalia, tonus increased by plastic type, propulsion, lateropulsion.
What is the most likely diagnosis?
A) Wilson-Konovalov disease
B) Essential tremor
C) Parkinson's disease
D) Huntington's chorea
E) Tourette's disease

31
A 58-year-old man noted a gradual increase in stiffness and slowness of movement over a year. Objectively: muscle rigidity of the "cogwheel" type, bradykinesia, Westphal, and Foix-Thevenard phenomena are positive.
Which structures of the nervous system are most likely to be affected?
A) Pallidonigral complex
B) Striar complex
C) Spinal cord
D) Cerebellum
E) Cortex

32
A 70-year-old woman complains of the slowness of movement. Objectively: bradykinesia, rest tremor, instability during posture change, turning, propulsion, retropulsion, amimia, micrographia.
Which neurotransmitter deficiency is most likely in this patient?
A) Acetylcholine
B) Serotonin
C) Dopamine
D) Glutamate
E) GABA

33
A 12-year-old girl was admitted with complaints of violent movements of mimic muscles: she raises and frowns her eyebrows, shrugs her shoulders, cannot hold her tongue out of her mouth, and overextension of the fingers of her hands is noted. All movements are fast and sprawling, non-stereotypical, intensified with excitement.
Which syndrome is most likely to occur in a girl?
A) Meningeal
B) Akinetic-rigid
C) Hyperkinetic
D) Cerebellar ataxia
E) Alternating syndrome

34
A 28-year-old man complains of involuntary movements that disappear in a dream and increase with emotional and mental stress, noting a weakening of memory and attention, relatives also had this disease.
What is the most likely diagnosis for the patient?
A) Rheumatic chorea
B) Huntington's chorea
C) Friedreich's disease
D) Wilson-Konovalov disease
E) Tourette's disease

35
The examination revealed hypotonia in the extremities, instability of the patient in the Romberg position, inability to stand, falls forward or backward, adiadochokinesis, dysmetria.
What type of ataxia is most likely in this patient?
A) Dynamic cerebellar
B) Static cerebellar
C) Vestibular
D) Sensitive
E) Cortical

36
A 30-year-old man presented with sprawling, unsteady, asynergic movements with a "drunken wobbly gait," during which the patient

spread his legs wide for stability. There is significant swaying to the sides when walking a single line. Nystagmus, adiadochokinesis, macrographia.
What type of ataxia is most likely in this patient?
A) Frontal
B) Vestibular
C) Sensitive
D) Cerebellar static ataxia
E) Cerebellar dynamic ataxia

37
The examination revealed nystagmus and instability in Romberg's test, the patient leaned to the right, and performed coordination tests with a miss on the right.
Where is the affection focus most likely to be located?
A) Right hemisphere of the cerebellum
B) Left hemisphere of the cerebellum
C) Internal capsule
D) Cerebellar vermis
E) Frontal lobe

Topic: Cranial nerves

38
A 50-year-old woman developed ptosis, divergent strabismus, and mydriasis on the right side and spastic hemiparesis on the left side due to increased BP.
What is the name of the alternating syndrome?
A) Claude
B) Weber
C) Jackson
D) Millard-Gubler
E) Wallenberg-Zakharchenko

39
The neurological picture: symptoms of "lagophthalmos, sail, exclamation mark" on the right, convergent strabismus on the right, restriction of the right eyeball movement to the outside, reflexes increased in the left extremities, Babinski's sign on the left.

What is the name of the syndrome?
A) Jackson
B) Foville
C) Thalamic syndrome
D) Capsular syndrome
E) Parkinson's syndrome

40
A 60-year-old man gradually developed weakness in his left arm and leg, and his right eye stopped closing. Objectively: symptoms of "lagophthalmos," "sail," and "exclamation mark" on the right, spastic hemiparesis on the left.
What is the patient's alternating syndrome?
A) Weber syndrome
B) Capsular syndrome
C) Meningeal syndrome
D) Thalamic syndrome
E) Millard–Gubler syndrome

41
The examination revealed convergent strabismus on the left, the left eye not closing, the nasolabial fold being smoothed on the left, the "sail" symptom, and the exclamation mark symptom on the left. There is a spastic hemiparesis on the right.
Where is the focus of affection localized?
A) Inner capsule on the left
B) Cerebral cortex on the right
C) Midbrain area on the left
D) Area of the pons on the left
E) Area of the medulla oblongata on the right

42
A 30-year-old man complained of headaches, fever, general weakness, and increased fatigue. Neurologic status: convergent strabismus on the left side, diplopia, rigidity of the occipital muscles.
Which nerve is affected?
A) Abducens
B) Oculomotor
C) Facial

D) Trochlear
E) Accessory

43
The examination revealed lacrimation on the right, the right eye not closing, the eyebrow not being raised, when inflating the cheeks, the right cheek "sailing", when grinning, the right corner of the mouth lagging.
Which nerve is affected?
A) Trigeminal
B) Facial
C) Abducens
D) Trochlear
E) Hypoglossal

44
A 60-year-old man, after a stroke, examination revealed dysphagia, dysarthria, nasolalia, hypophonia, paresis of the soft palate, atrophy of the tongue, and fibrillary jerks of the tongue muscles.
What syndrome was identified?
A) Akinetic-rigid
B) Hyperkinetic
C) Alternating
D) Pseudobulbar
E) Bulbar

45
A 40-year-old man complains of severe shooting pains in the entire left half of his face. Attacks are not controlled by non-steroidal anti-inflammatory drugs, provoked by chewing hard food, yawning, washing, and brushing teeth.
What is the most likely diagnosis for the patient?
A) Facial nerve neuropathy
B) Trigeminal neuralgia
C) Glossopharyngeal neuralgia
D) Trigeminal neuropathy
E) Ophthalmoplegia

46
A 30-year-old man complains of headache, fever, drowsiness, double vision. Objectively: divergent strabismus, ptosis, pupil response to light is preserved, but there is no response to convergence and accommodation.
What syndrome is the patient found to have?
A) Foville
B) Weber
C) Millard-Gubler
D) Argyll Robertson
E) Reverse Argyll Robertson

47
A young man in the background with an acute respiratory viral infection developed pronounced "burning" pains in the left half of the face and left eyeball. Vesicular rashes and skin hyperemia appeared on the forehead, upper eyelid, and cheek on the left side.
Where is the focus of affection localized?
A) Thalamus
B) Cerebral peduncles
C) Pons
D) Medulla oblongata
E) Gasserian ganglion

48
A 40-year-old woman has pain in the root of the tongue, severe, sometimes burning, attack-like, and poorly controlled by analgesics. Examination revealed that pupils are equal, the face is symmetrical, the tongue is along the midline, the uvula is along the midline.
What is the most likely diagnosis?
A) Prosoparesis
B) Glossopharyngeal neuralgia
C) Facial nerve neuropathy
D) Pterygopalatine ganglion neuralgia
E) Neuralgia of the II division of the trigeminal nerve

49
A 35-year-old man presents with attack-like pain in the left side of the face that begins in the upper lip area. The duration of attacks is from a

few seconds to one minute; attacks are provoked by chewing, washing, and shaving.
What is the most likely diagnosis?
A) Prosoparesis
B) Tumor at the pontine level
C) Facial nerve neuropathy
D) Pterygopalatine ganglion neuralgia
E) Neuralgia of the II division of the trigeminal nerve

50
A 30-year-old woman complained of restricted movements of the eyeballs on the left side and double vision when looking to the side. Examination revealed a convergent strabismus on the left side.
Which one of the following muscles is weak?
A) Lower oblique
B) Lower straight
C) Outer straight
D) Upper oblique
E) Inner straight

Topic: Cerebral cortex

51
A 25-year-old girl complains of visual impairment and weakness in the legs. Objectively: visual acuity in the right eye is reduced, spastic paraparesis in the legs. On the ocular fundus, there is a picture of retrobulbar neuritis.
Which symptom of optic nerve damage is described?
A) Amaurosis
B) Amblyopia
C) Hemianopsia
D) Color blindness
D) Visual agnosia

52
A 30-year-old woman complains of headaches, menstrual irregularities, and visual impairment for a year. Objectively: "cushingoid" type obesity, bitemporal hemianopsia.

Where is the pathologic focus most likely localized?
A) Occipital lobe
B) Gratiolet's bundle
C) Visual tract
D) Thalamus
E) Chiasma

53
The examination revealed dysarthria, tongue deviation to the left, hypotrophy and fibrillary twitching on the left half of the tongue, and central hemiparesis symptoms detected on the right side.
Where is the pathologic focus most likely localized?
A) Medulla oblongata on the right
B) Medulla oblongata on the left
C) Pons on the right
D) Midbrain on the right
E) Midbrain on the left

54
A 55-year-old woman complains of drooping eyelids on the right and right strabismus. Objectively: ptosis, mydriasis, divergent strabismus on the right.
Which nerve is affected?
A) Oculomotor
B) Trochlear
C) Trigeminal
D) Abducens
E) Facial

55
A 60-year-old man after a stroke shows swallowing impairment, choking while swallowing, hypophonia, nasal speech, dysarthria, and involuntary laughing and crying.
Which syndrome is most likely?
A) Bulbar
B) Pseudobulbar
C) Argyll Robertson
D) Ophthalmoplegic
E) Alternating

56
A 35-year-old man, during the examination of eye movements, has a fixed left eyeball in the midline, ptosis on the left, mydriasis, and lack of pupillary reactions on the left side as well.
Which syndrome is most likely?
A) Bulbar
B) Pseudobulbar
C) Ophthalmoplegic
D) Alternating
E) Ataxic

57
A 65-year-old man during neuropsychological examination is found to forget the names of objects, names objects using verbs, for example: pen - "it's what they write with," understanding of speech is preserved.
What speech disorder is observed in the patient?
A) Dysarthria
B) Motor aphasia
C) Sensory aphasia
D) Semantic aphasia
E) Amnestic aphasia

58
A 48-year-old woman notes that she sees everything distorted; objects appear in peculiar shapes, enlarged, or diminished in size.
What syndrome does the patient have?
A) Metamorphopsia
B) Hemianopsia
C) Amaurosis
D) Amblyopia
E) Scotoma

59
A 55-year-old man shows speech disturbances, can only pronounce isolated sounds (emboli), but understands the speech of others, and has weakness in the right extremities with increased muscle tone and tendon reflexes.

What form of speech disorder is most likely in the patient?
A) Amnestic aphasia
B) Semantic aphasia
C) Sensory aphasia
D) Motor aphasia
E) Dysarthria

60
A 45-year-old man complains of seizures in the left half of his body, starting from the foot and spreading upwards. On examination: mild central paralysis of the left leg with pathological plantar reflexes.
What is the name of the syndrome?
A) Weber
B) Horner
C) Benedikt
D) Wallenberg
E) Gagarin-Jackson

61
A 65-year-old man with diabetes gradually developed untidiness, inappropriate behavior, euphoria, and a tendency towards flat jokes.
What is the name of the described syndrome?
A) Frontal psychosis
B) Autotopognosis
C) Asteriognosis
D) Anosognosia
E) Ataxia

62
A 69-year-old man who has had a stroke does not realize his illness; despite severe hemiplegia, he plans unrealistic plans for his condition, intends to go to work, and claims to be fully healthy.
What is the name of this syndrome?
A) Aphasia
B) Alexia
C) Acalculia
D) Anosognosia
E) Autotopognosis

63
A 38-year-old woman complains of periodically experiencing the smell of "rotten meat," which does not exist in reality, headaches, and nausea. Objectively: cerebral syndrome, mild left hemiparesis. At the fundus of the eye: congested optic nerve discs.
Which part of the cortex irritation causes these complaints?
A) Frontal
B) Parietal
C) Temporal
D) Occipital
E) Parietal and occipital

64
A 55-year-old man who had a hemorrhagic stroke does not complain. Despite his disability and manifestations of severe hemiparesis, he does not realize his illness, intends to work, and makes unrealistic plans for his condition.
Where is the pathological focus in the described syndrome?
A) Frontal lobe
B) Parietal lobe
C) Occipital lobe
D) Base of brain
E) Temporal lobe

65
A 65-year-old man who has had a stroke has a speech disorder and can only say one word when trying to speak but understands speech addressed to him.
Where is the pathological focus located?
A) Broca's area
B) Wernicke's area
C) Temporal lobe
D) Parietal lobe
E) Occipital lobe

66
A 70-year-old woman had a stroke; she can speak but does not understand speech, neither her own nor others. Her speech contains numerous perseverations, making it difficult to understand.

Where is the pathological focus located?
A) Broca's area
B) Wernicke's area
C) Temporal lobe
D) Parietal lobe
E) Occipital lobe

Topic: Autonomic nervous system

67
A 30-year-old woman complains of attacks of whitening of the fingertips with sensations of numbness and crawling tingling, accompanied by pulsating pain in response to cold exposure.
Which syndrome is MOST likely in the patient?
A) Raynaud
B) Horner
C) Guillain-Barré
D) Lambert-Eaton
E) Miller-Fisher

68
A 35-year-old woman presented with complaints of heart palpitations, headaches, increased blood pressure up to 140/100 mmHg, increased sweating, chills-like tremors, and a pronounced sense of fear and anxiety. The duration of the attack ranges from 40 minutes to an hour. She associates it with psychoemotional stress.
What condition is most likely described?
A) Asthenic syndrome
B) Hypertensive crisis
C) Meningeal syndrome
D) Sympathoadrenal crisis
E) Intracranial hypertension syndrome

69
A 30-year-old woman complains of paroxysms of pain in the epigastric region, accompanied by increased peristalsis, a feeling of fear, decreased blood pressure and heart rate, increased sweating, and shortness of breath with a feeling of air shortage.
Which crisis is most likely?

A) Akinetic
B) Hypertensive
C) Myasthenic
D) Vagoinsular
E) Sympathoadrenal

70
A 35-year-old woman complains of pressing chest pain, shortness of breath, palpitations, fatigue, and headache after emotional stress. Objectively: facial hyperemia, pronounced hyperhidrosis of the palms and soles, agitation, emotional lability, respiratory rate - 20 per minute, heart rate - 105 per minute, blood pressure - 140/90 mmHg.
Which of the listed diagnoses is MOST likely?
A) Arterial hypertension
B) Newly developed angina
C) Small-focal myocardial infarction
D) Autonomic dystonia
E) Endocrine cardiomyopathy

Appendix 1

Test keys

1- B	15 – D	29 – B	43 – B	57 - E
2- C	16 – C	30 – C	44 – E	58 - A
3- D	17 – D	31 – A	45 – B	59 – D
4- D	18 – E	32 – C	46 – E	60 – E
5- D	19 – B	33 – C	47 – E	61 – A
6- D	20 – D	34– B	48 – B	62 – D
7- C	21 – D	35 – B	49 - E	63 – C
8- A	22 – A	36 – E	50 – C	64 - B
9- A	23 – D	37 – A	51 – B	65 – A
10 – B	24 – A	38 – B	52 – E	66– B
11 – E	25 – E	39 – B	53 – B	67 – A
12 – D	26– D	40 – E	54 – A	68 – D
13 – B	27– E	41 – D	55 – B	69 – D
14 – A	28– B	42 – A	56 - C	70 – D