

MINISTRY OF HEALTH OF UKRAINE
ODESA NATIONAL MEDICAL UNIVERSITY

Faculty of Dentistry

Department of Neurology and Neurosurgery

APPROVED BY

Vice-Rector for Scientific and Educational Work

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TEACHING MATERIAL
FOR ISW CLASSES ON THE ACADEMIC SUBJECT

Faculty, Course: Stomatological, 3th year

Academic Discipline: **Neurology**

Approved by:

Meeting of the Department of Neurology and Neurosurgery

Odesa National Medical University

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Topic: The main stages of the development of neurological science.

Actuality of theme : The first studies of diseases of the nervous system were carried out during the time of Hippocrates, Galen, and Avicenna. The study of neurology later took place in the universities of the Middle Ages and in the Renaissance. Later, domestic neurological schools played a significant role in the development of neurological science. Modern directions of the development of neurology: differentiation of neurological science (creation of separate centers and scientific units for the study of cerebrovascular, demyelinating diseases, epilepsy, neuromuscular pathology, etc.) and integration with other sciences (somatoneurology, vertebroneurology, neurosurgery).

As a result of independent study of this topic, applicants should:
know:

1. The main stages of the development of neurological science .
2. Contribution of domestic scientists to neurology.
3. Current state of neurological science
4. Promising directions in neurology

be able:

1. It is argued to defend the priorities of the national school of neurology.
2. Identify promising areas in neurology

Tasks for independent work during preparation for the lesson

A list of the main terms, parameters, and characteristics that the student should learn when preparing for the lesson:

Term: descriptive period in the history of neurological science, differentiation of neurological science.

Theoretical questions for the lesson:

1. Classification of the main stages in the development of neurological science.
2. Current state of neurological science

Topic content :

The first information about diseases of the nervous system can be found in ancient written sources. In Egyptian papyri about 3000 years BC. e. Paralysis, sensitivity disorders are mentioned. In the ancient Indian book of Ayur-Veda, seizures, fainting, and headache are reported. Clinical manifestations of various neurological diseases, methods of their diagnosis and treatment are described in the writings of Hippocrates, Raza, and Ibn Sina. Already at that time, certain conditions were clearly identified as brain diseases (epilepsy, migraine, etc.).

The development of neurology as a science is associated with the emergence and improvement of methods for researching the nervous system. In the Middle Ages, D. M. Morganhi and T. Villizius were able to connect certain neurological disorders with the corresponding structures of the brain. An important contribution to the development of the doctrine of the morphology of the nervous system was made by Andrii Vesalius, Jacob Sylvius, Constanzo Varolii. Descartes formulated the concept of reflex. This is how the foundations of neurophysiology were laid.

18th century was a descriptive period in the development of neurology. More and more new information appeared about individual symptoms, syndromes and diseases of the nervous system. Attempts were made to treat them.

In the 19th century intensively developed methods of studying the structure and functions of the nervous system, methods of chemical research of the brain. Pathological studies were systematized. It became possible to fix and stain nervous tissue, obtain serial sections, and carry out microscopic studies of the nervous system. Comparative anatomical, embryological and experimental studies played a major role in the development of neurology. They served as the methodological basis for progress in the study of the physiology of the nervous system. The development of this direction is associated with the names of I. M. Sechenov, I. P. Pavlov, N. E. Vvedenskyi, A. A. Ukhtomskyi, Magnus, Sherrington, and others.

I. M. Sechenov (1829-1905) was the founder of the reflex theory of human mental activity, he showed that the reflex is a universal way of the brain's response to a wide variety of external influences. I. M. Sechenov spoke against the centuries-old belief that the work of the brain does not obey the laws of the material world and is inaccessible to objective study.

However, I. M. Sechenov's ingenious assumption that any manifestations of a person's mental life are reflexes could become a scientific theory only as a result of the discovery of specific forms of reflex activity of the brain.

This task was solved by I. P. Pavlov (1849-1936) and his school, which developed the doctrine of higher nervous activity. The term "conditioned reflex" proposed by I. P. Pavlov, according to his definition, means a temporary, changeable, flexible connection of any variation of signals with the corresponding activity of the body. Conditioned reflexes are formed in the process of individual experience of animals or humans according to the principle of the greatest correspondence to the currently formed conditions. They are one of the mechanisms for reflecting the material world. I. M. Sechenov, I. P. Pavlov and their students N. E. Vvedenskyi and A. A. Ukhtomskyi developed the foundations of the theory of nervousness, thanks to which the understanding of the mechanisms of human brain functioning was significantly expanded.

The successes achieved in the field of neurology created the prerequisites for the separation of the doctrine of diseases of the nervous system into an independent field of scientific medicine. This field was called neuropathology.

Neuropathology has been enriched with new data in the field of pathological anatomy, electrophysiology, as well as data obtained during the study of clinical symptoms of diseases. A large number of independent forms of pathology were described, methods of their diagnosis and treatment were developed. In the 19th century Charcot created the French school of neuropathologists. Its representatives were Duchenne, Dezherin, Babinsky, Raimon, Bourneville, Brissot, and others.

The authors of classical works on neuropathology in Germany were Strümpel, Westphal, Wernicke, Romberg, Friedrich, Erb, Oppenheim, and others. In England in the XIX century. such scientists as Jackson, Hovers, Parkinson, and Thomsen were representatives of neuropathology.

The history of the establishment and development of the Odessa School of Neurologists begins on October 11, 1903, when the Department of Nervous Diseases was opened in Odessa.

Professor Mykola Mykhailovych Popov (1854-1939) became the first head of the Department of Nervous and Mental Diseases. Dissertation for the degree of doctor of medicine "Materials for study on acute myelitis of toxic origin" M.M. Popov defended in 1882. He is the author of 32 scientific papers, including 2 on normal brain histology; 10 - on the pathoanatomy of nervous diseases with a clinical analysis of toxic myelitis, Asian cholera, rabies, spinal tuberculosis, acute delirium; 20 works are devoted to the clinic of neuroses, progressive paralysis and the treatment of malaria. He wrote "Lectures on Contemporary Psychopathology", 1925.

Oleksiy Erastovich Yanyshkevskyi (1873-1936) - professor of the Department of Nervous and Mental Diseases (1916-1920). In 1903, he defended his thesis for the degree of doctor of medicine "On the commissural systems of the fibers of the cerebral cortex (corpus callosum, anterior commissure and David's lyre)". Author of 38 scientific publications, including two textbooks ("Symptomatology of diseases of the nervous system", 1918; "Textbook of nervous diseases", 1929).

Professor Volodymyr Mykolayovych Obraztsov (1873-1926) headed the Department of Nervous and Mental Diseases from 1920 to 1926. In 1920, he was approved as the director of higher women's medical courses, took part in their merger with the Odessa Medical Academy. Then, in addition to being the head of the department of nervous diseases of the Odesa Medical Institute, he worked as a professor of nervous diseases at the Odesa Clinical Institute, a professor at the Institute of National Economy, head of the neuropathology section of the research department of experimental medicine.

He defended his thesis for the degree of doctor of medicine in 1904, and from 1912 he worked as a professor. The author of more than 30 scientific works devoted to the diagnosis, clinic and treatment of nervous and mental diseases. He was a member of many scientific societies, was the secretary of the "Neurological Herald" editorial office. From the day of its founding of the Odesa Society of Neuropathologists and Psychiatrists until his death, he was its constant chairman.

From 1926 to 1952, the department of nervous diseases was headed by Hryhoriy Ivanovich Markelov (1880-1952) - professor, academician, outstanding neuropathologist-vegetologist. All his scientific activity is connected with Odessa. From October 1, 1930, he part-time managed the Odesa Research Psychoneurological Institute.

G.I. Markelov is the author of more than 100 scientific works devoted to the physiology and pathology of the autonomic nervous system. He developed a classification of diseases of the autonomic nervous system and proposed methods of examining patients.

Many years of experience in the study of the autonomic nervous system enabled the scientist to conclude that the autonomic nervous system should be studied by a special science, which he called evolutionary vegetology ("Evolutionary vegetology and its tasks" (1948). G.I. Markelov was one of the first clinical scientists took part in the creation of a new science - biocybernetics. He is the author of the monographs "Extrapyramidal System" (1929), "Vegetative Neuralgia and Neuritis" (1930), "Semiotics and Diagnosis of Diseases of the Autonomic Nervous System" (1934), etc. Monograph "Diseases of the Autonomic System " (1948) is a fundamental guide to diseases of the autonomic nervous system.

G.I. Markelov is one of the organizers of the Odesa Psychoneurological Institute, he was its scientific director in 1930-1952. It was this institute and the Department of Nervous Diseases of ODMU that became the basis for the formation of his scientific school.

To the older generation of the school of Professor G.I. Markelov should include the following outstanding neuropathologists, psychiatrists and representatives of related specialties: E.A. Shevaleva, L.A. Mirelzona, L.L. Papadato, O.S. Kondratieva, V.P. Kuznetsova, R.O. Khersonskiy, M.O. Gornyya, O.S. Lyvshina, I.I. Dobrovolskiy, V.D. Usatova, B.Ya. Sosyuru, I.M. Shcherbakova, B.O. Benesovicha, T.P. Shesterikov, O.D. Gasquet, K.A. Yelizarov, D.I. Rahman, N.B. Vishnevskaya and many others.

Pupils and successors of Academician G.I. Markelov were professors K.V. Mosqueti (psychiatrist), Yu.O. Petrovych (biochemist), senior researcher, candidate of medical sciences V.V. Russev (physiologist), candidate of medical sciences E.O. Sokolova (neurophysiologist-neuropathologist), senior researcher, candidate of medical sciences E.L. Puchkovskaya (neuropathologist-vegetologist), professor S.B. Aksentiev (psychiatrist), E.P. Dmitrieva, E.I. Mitrofanova and N.N. Mygulya-Dmytrenko (neuropathologists), professor Yu.L. Kurako (neurologist) and many others who worked in different cities of the country.

Professor Lev Levovich Papadato (1887-1955) headed the department of nervous diseases of the pediatric faculty from 1938 to 1941, and in 1952-1953 he headed the department of nervous diseases. L.L. Papadato graduated from the medical faculty of the University of Paris, where he studied with Dezherin and Lyapik. For the work "Cervical hypertrophic pachymeningitis" he was awarded the medal of this faculty (1912). In 1940, he defended his thesis for the degree of doctor of medical sciences "Vegetoendocrine factors in the genesis of genuine epilepsy" and in 1945 was approved for this degree. In 1946, he was awarded the academic title of professor at the Department of Nervous Diseases.

L.L. Papadato is the author of more than 45 scientific works, including the monographs "Anatomy of the subarachnoid space. Spinal cord and blood-brain barrier" (1929) and "Vegetative hypothalamopeduncular syndromes" (1934). His scientific research is devoted to issues of pathogenesis, clinic and therapy of epilepsy, hypothalamic syndromes, child psychoneurology, physiology of cerebrospinal fluid, etc.

Professor Borys Ivanovich Sharapov (1897-1969) headed the Department of Nervous Diseases from 1953 to 1956. In 1935, B.I. Sharapov defended his thesis for the degree of doctor of medical sciences "To the pathological anatomy of tuberculosis of the spinal cord". In 1940, he was approved as a professor at the Department of Nervous Diseases. The author of more than 90 scientific works devoted to the issues of brain blood circulation disorders, injuries of the central nervous system, poliomyelitis, meningitis, he was one of the first to start studying clinical problems of the reticular formation of the brain. In 1965, he published the fundamental book "Studies of the clinic and pathological anatomy of the reticular formation of the brain", in fact, the first monograph that shed light on the clinical aspects of the problem.

Professor B.I. Sharapov and Yu.L. Curacao, in 1953, a well-equipped pathohistological laboratory was created at the department, which became a base for the execution of theses and scientific works.

Professor Georgy Havrylovich Sokolyanskyi (1899-1982) headed the Department of Nervous Diseases from 1956 to 1972. Just like B.I. Sharapov, H.G. Sokolyanskyi was a student of the famous neuropathologists L.V. Blumenau and S.N. Davydenkova. In 1937, he defended his thesis for the degree of doctor of medical sciences "On the morphogenesis of peripheral myelin nerve fibers and their development in humans." In 1944, he received the title of professor at the Department of Nervous Diseases. H.G. Sokolyanskyi is the author of more than 30 scientific works on clinical, diagnostic, pathological anatomy and treatment of various diseases of the nervous system (acute viral infections, hereditary diseases, epilepsy, disorders of cerebral circulation, etc.). He is the author of the method of staining myelin fibers ("Sokolyansky's method"), which is widely used in neurohistological practice.

Since 1964, the Department of Nervous Diseases has included a neurosurgery course headed by Professor Korol O.P. (1930-2003). O.P. The king is the author of more than 300 printed works and several inventions. Under his leadership, 6 candidates and 12 neurosurgeons from Asia, Africa, and Latin America were trained and protected. Oleksandr Pavlovich and his students made a significant contribution to the development of current problems of neuro-oncology, neurotraumatology, and cerebrovascular pathology. For many years, Professor Korol O.P. was the head of the Odesa Scientific Society of Neurosurgeons (since 1994, the Association of Neurosurgeons of the Odesa Region), a member of the editorial board of the journals "N.N. Burdenko Questions of Neurosurgery" and "Ukrainian Neurosurgery Journal", a member of the Problem Commission of the Ministry of Health on Neurotraumatology.

Professor Yuriy Lvovich Kurako headed the Department of Nervous Diseases with Medical Genetics and the Neurosurgery Course from 1972 to 2002. Yu.L. Kurako graduated with honors from the medical faculty of the Dnipropetrovsk Medical Institute (1951). He is a student of Academician G.I. Markelov, professors N.V. Mirtovskiy, B.I. Sharapova, L.L. Papadato, H.G. Sokolyanskyi. Yu.L. Kurako is the author of more than six hundred printed scientific works, twenty-three inventions, and twelve monographs. One of them - "Mild closed craniocerebral trauma" - in 1989 was awarded the republican award named after Academician B.M. Mankovsky. In 1999, by decree of the President of Ukraine, together with a group of leading scientists, he was awarded the state prize in the field of science and technology for the cycle of scientific works "Organic damage to the nervous system in children, development and implementation in practice of new methods of diagnosis, treatment, prevention, medical rehabilitation and social adaptation".

In 2002, the neurosurgery course returned to the Department of Nervous Diseases, since then the department has been called the "Department of Neurosurgery and Neurology". The department was headed by Professor Anatoly Sergeyevich Son, a student of Professor M.M. Mosichuk (Dnipropetrovsk). Professor A.S. Son graduated from the Dnipropetrovsk State Medical

Institute in 1978. In 2001, he defended his doctoral thesis "Surgical treatment in the acute period of complicated subarachnoid hemorrhages due to the rupture of arterial aneurysms of the anterior sections of the arterial circle of the cerebrum", in 2003 he was awarded the title of professor. Professor A.S. Son is a member of the editorial board of the journals "Ukrainian Neurosurgical Journal", "Problems of Biology and Medicine", "International Neurological Journal", a member of the board of the Ukrainian Association of Neurosurgeons, the head of the Association of Neurosurgeons of the Odesa Region and the Odesa Scientific and Practical Society of Neurologists.

The modern directions of development of neurology are as follows:

- differentiation of neurological science (creation of separate centers and scientific divisions for the study of cerebrovascular, demyelinating diseases, epilepsy, neuromuscular pathology, etc.)
- integration with other sciences (somatoneurology, vertebroneurology, neurosurgery).

Literature:

Basic.

1. Neurology: study guide / [I.A. Hryhorova, L.I. Sokolova, R.D. Gerasymchuk, A.S. Son, etc.] edited by I.A. Grigorova, L. I. Sokolova - 3rd edition - Kyiv, Medical University "Medicine", 2020 - 640 p.
2. Topical diagnosis of pathology of the nervous system. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.
3. Methods of examination of a neurological patient: teaching. Guide / edited by L. I. Sokolova, T. I. Ilyash. K., 2020. - 144 p.
4. Emergency medicine. Emergency medical care: textbook / I.S. Zozulya, V.I. Bobrova, H.G. Roschyn and others / edited by I.S. Cuckoos - 3rd edition, trans. and additional - Kyiv. - VSV "Medicine", 2017. - 960 p.
5. Negrych T.I., Bozhenko N.L., Matvienko Yu.Sh. Ischemic stroke: secondary inpatient care: education. manual Lviv: LNMU named after Danylo Halytskyi, 2019. – 160 p.

Additional literature

1. Bozhenko M.I., Negrych T.I., Bozhenko N.L., Negrych N.O. Headache. Study guide.-K.: Medknyga Publishing House, 2019. - 48 p.
2. Davidson's Medicine: Principles and Practice: 23rd Edition: In 3 Volumes. Volume 1 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2020. - 258 p.
3. Davidson's Medicine: Principles and Practice: 23rd Edition: In 3 Volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 778 p.
4. Davidson's Medicine: Principles and Practice: 23rd Edition: In 3 Volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021 . - 642 p.
5. Medical Books On-line Library (Neurology) – free download
<http://medbookshelf.info/category/neurology/>

Electronic information resources

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

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

ISW No. 2

Topic: Epilepsy. Non-epileptic paroxysmal states with and without convulsions.

Actuality of theme:

The prevalence of epilepsy and paroxysmal disorders of consciousness similar to it necessitates their study and detection in order to provide emergency aid for convulsive and paroxysmal conditions, to carry out rehabilitation and social rehabilitation measures with the aim of improving the body, ensuring the opportunity for such patients to live actively.

Late recognition and treatment of these conditions often does not lead to a clinical effect, causes disability, and leads to a chronic course of the disease. Timely diagnosis makes it possible to successfully treat these diseases, to carry out effective preventive measures. For the correct assessment of various clinical forms of these diseases, thorough knowledge is required.

Specific goals:

- the acquirer must know:
 - a) Modern classification of epilepsies and epileptic syndromes.
 - b) Symptomatic epilepsy.
 - c) Idiopathic epilepsy.
 - d) Etiology, pathogenesis, main clinical manifestations of epilepsy.
 - e) Criteria and assessment of clinical symptoms and syndromes of seizures and epilepsy, disorders of consciousness and psyche.
 - f) Types of aura.
- to be able to examine patients:
 - a) For epilepsy (with major convulsive attacks).
 - b) With small epileptic attacks.
 - c) With epileptic status.
 - d) Prescribe treatment for status epilepticus and epileptic seizures.
 - e) Carry out differential diagnosis between epilepsy and hysteria.

Questions for the lesson to be discussed:

1. Modern classification of epilepsy.
2. Idiopathic and symptomatic epilepsy. Etiology, pathogenesis.
3. Types of auras and their topic.
4. Clinical course of epilepsy.
5. Differential diagnosis of epilepsy.
6. Modern treatment of various epileptic attacks.

New terms:

Aura
Digestive aura
Resistant epilepsy
Jacksonian March
Postictal paralysis
Epistatus

Questions for determining the quality of students' mastery of the lesson topic:

1. Where is cerebrospinal fluid produced and how is it exchanged?

Answer: CSF is produced in the choroidal plexuses of the lateral ventricles of the brain. Through the III and I ventricles and the openings of Lyushka and Mozhandi, it enters the subarachnoid

space of the brain (between the soft and arachnoid membranes), circulates around the brain and is absorbed by the pachyon granulations of the brain and spinal cord.

2. What is the composition of cerebrospinal fluid?

Answer: Protein - 0.33 ‰, cells - 1.7 per cubic meter. mm; glucose - half of the patient's blood glucose level; chlorides – 112 mg/%

3. What modern anticonvulsant drugs do you know?

Answer: luminal, benzonal, difenin, ethosuximide, eunocin, valproic acid preparation (decapine, convulsafin, orphyryl, acediprol, etc.), seduxen, carbamazepine, lamictal.

4. Etiological factors of epileptic attacks?

Answer: increased convulsive readiness of the cerebral cortex, epileptic focus, external epileptogenic stimulus.

5. Provocative factors of epileptic attacks?

Answer: alcohol, various infections, overwork, operations, abortion, childbirth, exposure to the sun, somatic diseases, craniocerebral injuries.

Literature:

Basic

1. Neurology: study guide / [I.A. Hryhorova, L.I. Sokolova, R.D. Gerasymchuk, A.S. Son, etc.] edited by I.A. Grigorova, L. I. Sokolova - 3rd edition - Kyiv, Medical University "Medicine", 2020 - 640 p.
2. Topical diagnosis of pathology of the nervous system. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.
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5. Negrych T.I., Bozhenko N.L., Matvienko Yu.Sh. Ischemic stroke: secondary inpatient care: training. manual Lviv: LNMU named after Danylo Halytskyi, 2019. – 160 p.

Additional

1. Bozhenko M.I., Negrych T.I., Bozhenko N.L., Negrych N.O. Headache. Study guide.-K.: "Medknyga" Publishing House, 2019. - 48 p.
2. Davidson's Medicine: Principles and Practice: 23rd Edition: In 3 Volumes. Volume 1 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2020. - 258 p.
3. Davidson's Medicine: Principles and Practice: 23rd Edition: In 3 Volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 778 p.
4. Davidson's Medicine: Principles and Practice: 23rd Edition: In 3 Volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 642 p.

ISW No. 3

Topic: Pathology of the autonomic nervous system.

Relevance of the topic: The autonomic (autonomic) nervous system is often affected after

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

Lesson goals:

To create in the students an idea about the importance of the normal functioning of the autonomic nervous system for the balanced adaptation of the organism to the conditions of the external environment.

Specific goals:

- to know

- 1) The structure and functions of the hypothalamic region.
- 2) Anatomy of the peripheral part of the autonomic nervous system.
- 3) The main symptoms of damage to the central and peripheral parts of the autonomic nervous system.
- 4) Causes of damage to the autonomic nervous system.

Based on theoretical knowledge of the topic:

- be able to investigate and diagnose:

1. The main symptoms of damage to the central part of the autonomic nervous system.
2. The main symptoms of damage to the sympathetic division of the autonomic nervous system.
3. The main symptoms of damage to the parasympathetic department of the autonomic nervous system
4. Vegetative status of the patient and his disorders.

Topic content .

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

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

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

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

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

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

the parasympathetic division is phylogenetically older than the sympathetic.

The sympathetic nervous system contributes to the rapid mobilization of energy and adaptation of the body to the constantly changing conditions of the external environment. Through adrenergic structures, it provides somatovegetative correlation in various manifestations of the body's activity. In particular, in behavioral acts, in the processes of physical and mental work. This is basically an ergotropic system associated with catabolic (dissimilatory) processes.

The parasympathetic nervous system, on the contrary, contributes to ensuring the stability of the internal environment, manages the recovery processes of the body's losses of energy and nutrients, and increases the activity of assimilative processes. It plays a particularly important role in the regulation of digestion and certain phases of sleep. This is a trophotropic system associated with anabolic (assimilative) functions

Anatomy of the autonomic nervous system

In the central nervous system, there is a segmental apparatus of the autonomic nervous system (cells located in the brain stem and spinal cord), which give rise to preganglionic efferent fibers, and a suprasegmental section, which includes the nuclei of the hypothalamus, as well as the limbicorecticular complex and some sections of the associative zone of the cerebral cortex, which make mainly suppressive effect on the hypothalamus.

The cortex of the central lobe of the medial surface of the cerebral hemispheres (limbic lobe) is the cortical lobe of the visceral analyzer. The term limbic system belongs to the American scientist McClean (1952), who proposed his scheme of the structure and activity of the limbic system or visceral brain. According to McClean, the limbic system includes a number of cortical and subcortical structures, in particular:

- olfactory bulbs, tracts, triangle,
- anterior perforated substance (substantia perforata anterior).
- on the medial surface of the hemispheres, it includes:
 - septum pellucidum,
 - girdle gyrus (g.cinguli),
 - seahorse twist,
 - orbital lobes of the frontal lobe,
 - the front part of the islet,
 - the pole of the temporal lobe.

Important structures of the limbic system are the hippocampus, the dentate gyrus, the island of the old cortex near the corpus callosum (Idusium griseum), the base and prebase of the hippocampus.

The subcortical structures of the limbic system are the caudate nucleus, shell, amygdala nucleus, anterior (nonspecific) nuclei of the thalamus, hypothalamus, and frenum nuclei. Among the subcortical structures of the limbic system, the central place belongs to the hypothalamus. Thus, the limbic system is a complex intersection of ascending and descending pathways that form multiple closed circles anatomically connected to the reticular formation of the brain stem.

Physiology of the limbic system.

1. The limbic system is a visceral brain that receives afferent impulses from internal organs.
2. It takes part in the implementation of emotional reactions.
3. It is the nervous substrate of memory, which stores not genetically inherited, but acquired experience. Bilateral removal of the medial surface of the temporal lobes of the brain causes severe memory disorders, retrograde amnesia occurs, the ability to remember new things is impaired, and short-term memory suffers sharply.

4. The limbic system provides motivation for thirst, hunger, sexual desire, regulates the rhythm of sleep and alertness, participates in the regulation of the respiratory, cardiovascular, digestive, genitourinary systems, vegetative reactions, ensures the integration of the cardiovascular and respiratory systems, endocrine and vegetative systems functions. It influences the activity of the new cortex, makes visceral sensations conscious, especially in pathological conditions of

internal organs.

The second representative of the central department of the autonomic nervous system is the hypothalamic area, in which higher subcortical mechanisms of regulation of sympathetic and parasympathetic innervation are carried out.

There are 32 groups of cell nuclei in the hypothalamus: central gray matter, paraventricular, supraoptic (n. supraopticus), nucleus of the gray hump (n. tuberis cinerei), infundibulo-tuberal nuclei. Posterior group (n. mamilloinfundibularis), nuclei of mammillary bodies, nuclei of Lewis bodies. A pair of medial and connecting nuclei are located in the walls of the III ventricle. In the mammillary bodies, the nuclei of the mammillary bodies (mammillary, premammillary, medial, supramammillary, lateral). The hypothalamus is connected to other parts of the nervous system: the cerebral cortex, the visual humps, the extrapyramidal system, the lower nuclei of the brainstem and spinal cord, the reticular formation, and the pituitary gland. The front part of the hypothalamus is with the parasympathetic nervous system, the back part is with the sympathetic nervous system.

In the hypothalamus, 7 tropic neurohormones were found, activating the release of the corresponding pituitary hormone - releasing factors and 3 inhibiting (inhibiting) the release of tropic hormones from the pituitary gland.

Releasing factors include:

1. ACTH - releasing factor - corticoliberin
2. Thyrotropin - releasing factor - thyroliberin
3. Releasing factor of luteinizing hormone - luliberin
4. Releasing factor of the follicle-stimulating hormone - foliliberin
5. Somatotropin-releasing factor - somatoliberin
6. Prolactin-releasing factor - prolactoliberin
7. Releasing factor of melanocyte-stimulating hormone - melanocytoliberin

Inhibitory (inhibiting) factors include:

1. Prolactin - inhibitory factor - prolactostatin
2. Melanocytostatin - melanocytostatin
3. Somatotropin-inhibiting factor - somatostatin

A change in the concentration of hypothalamic neurohormones affects the biosynthesis and release of the corresponding tropic hormones by the anterior lobe of the pituitary gland, which leads to the development of endocrine diseases: acromegaly, Itsenko-Cushing's disease, goiter, hypothyroidism, pituitary cachexia, pluriglandular syndrome, and others.

The hypothalamus takes part in the regulation of the following body functions:

1. Activities of the cardiovascular system
2. Thermoregulation
3. Water, carbohydrate, fat, mineral, mucopolysaccharide metabolism
4. Permeability of blood vessels and tissue membranes
5. Functions of endocrine glands both through the pituitary gland and through the sympathetic and parasympathetic divisions of the autonomic nervous system
6. Morphology
7. Functions of organs of the gastrointestinal tract.
8. Ensures stability of the internal environment
9. In adaptive adaptation, thanks to the cortical-subcortical relationship
10. Plays an important role in emotional behavior.

The hypothalamus is connected with the reticular formation and the limbic system.

The parasympathetic department of the autonomic nervous system is represented by four lobes:

1. hypothalamic
2. mesencephalic
3. bulbar

4. sacral.

In the hypothalamus, the parasympathetic nervous system is represented in the anterior part by the supraoptic nuclei. Its cells, preganglionic neurons, control the cells of the posterior lobe of the pituitary gland, which are modified postganglionic neurons.

In the mesencephalic section, the parasympathetic nervous system is represented by autonomic nerve cells and fibers of the oculomotor nerve (Yakubovich's nuclei and Perle's nucleus at the bottom of the aqueduct of the midbrain), the fibers of which innervate the pupillary sphincter —m. sphincter pupillae and ciliary muscle (function of accommodation).

They belong to the bulbar department

- the upper salivary nucleus, which innervates the lacrimal and submandibular and sublingual salivary glands;
- the lower salivary nucleus, which innervates the parotid salivary glands
- the posterior core of the vagus nerve, from which fibers go to the larynx, trachea, bronchi, heart and other organs of the chest and abdominal cavity, that is, to all internal organs, walls, vessels, except for the pelvic organs.

The sacral division of the parasympathetic nervous system includes cell groups in the gray matter of the spinal cord at the level of II, III and IV sacral segments. Their axons form internal pelvic nerves (nn splanchnici pelvini), which innervate the muscles and mucous membrane of the pelvic organs (bladder, rectum, external and internal genitals).

The sympathetic division of the autonomic nervous system, in addition to the central representation in the hypothalamic division (posterior lobe), is represented by cell groups (first neurons) located in the gray matter of the lateral horns of the spinal cord from the VIII cervical to the II and III lumbar segments. The axons of these cells in the composition of the anterior roots, and then the white connecting branches (rr communicantes albi) enter the nodes of the sympathetic trunk (gangl. truncis sympathici), which are placed symmetrically in the form of chains on the sides of the spinal trunk, 20-25 nodes on each side. In the coccyx, both chains are connected by means of an odd knot (gangl. impar). Some fibers penetrate the nodes of the symmetrical sympathetic trunk and end in them or in intramural nodes.

In these nodes, there are second neurons, the processes of which go directly to one or another organ. Thus, prenodal (preganglionic) and postnodal (postganglionic) vegetative fibers are distinguished. Fibers that go to the nodes of the abdominal cavity, merging into large nerve trunks - the large internal nerve (n. splanchnicus major) from the V-IX thoracic nodes and the small internal nerve (n. splanchnicus minor) from the X-XI thoracic nodes. The largest prevertebral nodes are the paired abdominal node (gangl. celiacus), upper and lower mesenteric nodes (gangl. mesentericum superius et inferius).

Parasympathetic fibers from the vagus nerve join prevertebral and intramural nodes and plexuses. Sympathetic fibers in the muscular membrane of the stomach form the musculo-intestinal plexus (Auerbach - plexus niyenerius), and from it fibers go to the submucosal base of the gastric mucosa, forming the submucosal plexus (Meissner) - plexus submucosus). These plexuses extend to the intestine, esophagus and pharynx.

Part of the fibers of the cells of the lateral horns, which do not go to the paravertebral, prevertebral and intramural nodes, approach the somatic peripheral nerves and in their composition go to the muscles, blood vessels, skin and its appendages (sweat glands and muscles that raise the hair).

From the nodes of the sympathetic trunk, sympathetic fibers go to organs and parts of the body. Therefore, a certain clinical picture corresponds to the lesion of each node.

For example, a lesion of the upper cervical node (gangl. cervicale superius) is characterized by narrowing of the pupil, narrowing of the eye slit, enophthalmos (Bernard-Horner syndrome);

with damage to the cervicothoracic or stellate node (gangl. cervicothoracicum seu stellatum) heart disorders, pain and sensitivity disorders in the upper limb and upper chest are characteristic.

The thoracic part of the sympathetic trunk consists of 10-12 nodes. After the ganglionic

fibers from them go to the intercostal nerves, vessels and organs of the thoracic and abdominal cavity; from the I-V nodes - to the cardiac plexus, from the V-X nodes - the large and small internal nerves go to the abdominal (solar) plexus and mesenteric nodes.

The lumbar region consists of 4-5 nodes, from which the fibers go to the sacral root nerves, the abdominal plexus, and the abdominal part of the aorta.

Sympathetic innervation does not have such a clear distribution as somatic:

- sympathetic fibers coming from VIII cervical and I, II, III thoracic segments innervate the face and neck;
- from IV-VII – upper extremity,
- from VIII and IX - trunk,
- from X-XII thoracic, I and II lumbar segments - the lower limb.

Sympathetic postganglionic fibers (together with parasympathetic) form a plexus around vessels and internal organs of the chest and abdominal cavity. The largest plexuses are the thoracic aortic, abdominal aortic, superior mesenteric, and superior hypogastric plexuses.

Peculiarities of the activity of the autonomic nervous system.

The autonomic nervous system regulates the processes that take place in organs and tissues. However, these processes can be disturbed in case of dysfunction of the autonomic nervous system; numerous disorders arise. Most pathological processes in it are caused not by loss of functions, but by irritation, that is, increased excitability of the central and peripheral apparatus. Repercussions are a feature of the autonomic nervous system: disturbances in some parts of this system can lead to changes in others.

Clinical manifestations of lesions of the autonomic nervous system

Pathological processes localized in the cerebral cortex can lead to the development of trophic disorders in the innervation zone.

When the lobulus paracentralis is damaged, urination is disturbed according to the central type, and when the limbic-reticular complex is damaged, the emotional sphere (of a permanent or affective nature) is disturbed. Anorexia or bulimia appears (rice), sexual disorders, memory is disturbed in the type of amnestic Korsakovsky syndrome, in which the patient loses the ability to remember current events, sleep disorders, psychosensory disorders, changes in consciousness can be manifested by akinetic mutism, attacks of truncal and temporal epilepsy, disorders of the cardiovascular and respiratory systems.

Irritation of limbic structures is accompanied by an elevated background of mood, euphoria, extreme emotional reactions, excited restlessness, hypersexuality.

Functional suppression of the limbic system is manifested by a lowered mood background, abulia, a decrease in sexual desire, hypochondriac fixation of unpleasant sensations from internal organs, a state of panesthesia.

When the hypothalamus is damaged, various clinical manifestations (syndromes) may occur:

1. Vegetative-vascular-visceral
2. Exchange and endocrine
3. Nervous-trophic
4. Neuromuscular
5. Violation of thermoregulation
6. Sleep disturbance
7. Psychopathological
8. Diencephalic epilepsy.

Most often, damage to the hypothalamus manifests itself in vegetative-vascular, neuro-endocrine disorders, disorders of thermoregulation, water, mineral, fat and protein metabolism, sleep and alertness disorders. Women aged 20-40 are more often ill.

Hypothalamic syndrome with vegetative-vascular disorders (vegetative-vascular form) is most common. In the inter-crisis state, patients complain of general weakness, increased fatigue, physical and mental exhaustion, increased sensitivity to changes in meteorological factors, pain in the heart, palpitations, a feeling of lack of air, and irregular breathing. Symptoms from the gastrointestinal tract are often leading: pain in the epigastrium, unpleasant sensations in the intestines, nausea, belching of air and bile, urges to defecate, sometimes diarrhea. An objective examination reveals invigoration of tendon and periosteal reflexes, asymmetry of blood pressure, its fluctuations with a tendency to increase, tachycardia, lability of the pulse, increased sweating, persistent diffuse red dermographism, trembling of the eyelids and fingers of outstretched hands, a tendency to allergic reactions. Emotional disorders (anxiety, fear), sleep disturbances are observed. More often, vegetative landslides have a sympathetic direction.

Less often, in the vegetative-vascular form, parasympathetic manifestations dominate, but they can be combined (mixed form). Against the background of permanent vegetative disorders, vegetative-vascular paroxysms (up to 15-20 minutes) or crises (from 15-20 minutes to 2-3 hours or longer) occur. More often, they are provoked by emotional stress, changes in weather conditions, menstruation, pain factors, stressful situations, etc. Attacks occur more often in the afternoon or at night, without warning. Sometimes attacks are preceded by changes in mood, headache, unpleasant sensations in the heart area, a state of brokenness, weakness.

Depending on the dominance of autonomic disorders, crises can be:

- sympathetic-adrenal,
- vago-insular (parasympathetic)
- mixed

In sympathetic-adrenal crisis:

- a headache appears or worsens,
- there is a heartbeat,
- numbness and coldness of the extremities,
- blood pressure rises to 150/90-180/110 mm Hg. art.,
- the pulse increases to 110-140 per minute,
- unpleasant sensations in the area of the heart, numbness, "goosebumps" are noted.
- in some patients, the body temperature rises to 38-39 °C,
- there is excitement, motor restlessness,
- fear of death.

Vagoinsular crises are manifested:

- a feeling of heat in the head and face,
- headache, heaviness in the head,
- sometimes there are unpleasant sensations in the epigastric area, nausea,
- heart failure,
- general weakness, dizziness, sweating.

During the examination, the following is noted:

- slowing of the pulse (up to 45-50 per minute),
- decrease in blood pressure to 80/50-90/60 mm Hg.
- urges to defecate, increased intestinal peristalsis may occur,
- breathing becomes difficult,
- possible allergic manifestations in the form of urticaria or Quincke's edema.

Mixed crises. They are characterized by a combination of symptoms typical for sympathetic-adrenal and vago-insular crises or their alternating manifestations. Autonomic paroxysms can occur not only when the hypothalamic area is affected, but also the temporal lobes, brain stem, autonomic ganglia, plexuses, and other structures of the autonomic nervous system.

By frequency, they distinguish:

frequent crises - 5 or more per month (including daily)
average frequency - 3-4 per month
rare - 1-2 per month

Neuroendocrine-metabolic form. Damage to the hypothalamus in most cases is accompanied by a violation of the function of the glands of internal secretion and most often the functions of the pituitary gland. There are disorders associated with hyper- and hypofunction of the pituitary gland and other glands of internal secretion. Common endocrine disorders occur against the background of vegetative disorders. Violations of fat, carbohydrate, protein, water-electrolyte metabolism, appetite in the form of bulimia or anorexia, thirst, sexual disorders, isolated forms of endocrine gland damage may be observed (for example, diabetes insipidus, thyrotoxicosis, etc.). However, more often there are violations of the functions of a number of endocrine glands. Suppression of the gonadotropic function of the pituitary gland is often noted, manifested by amenorrhea or dysmenorrhea in women, a decrease in potency in men. Neuroendocrine syndromes may develop: Itsenko-Cushing, Froehlich-Babinsky adeno-genital dystrophy, Lawrence-Moon-Bardet-Biddle, Morganhi-Stuard-Morel, Prader-Willi, Kleine-Levin, Alström-Halgren, Edwards, Barraker-Simons leukodystrophy syndrome, Dercum's disease, Madelung's disease, and mixed forms of cerebral obesity. Early menopause in young women, changes on the part of the thyroid gland of hypothalamic genesis, and acromegaloïd phenomena are noted.

Neurodystrophic forms. Trophic disorders of the skin (itching, dryness, neurodermatitis, ulcers, bedsores), muscles, bones (osteomalacia, sclerosing), widespread ulcers in the mucous membrane of the stomach, the lower part of the esophagus, acute perforations of the esophagus, stomach, and duodenum.

Neuromuscular syndrome is manifested by periodic myasthenic or myotonic disorders, as well as paroxysmal myoplegia. Combinations of different types of muscle disorders are often observed.

Violation of thermoregulation. It is characterized by a prolonged subfebrile temperature with its periodic increase in the form of hyperthermic crises (up to 38-40 °C). Manifestations of vegetative dystonia of sympathetic-adrenal or mixed type are also noted. Temperature disturbances do not affect the well-being of patients. Their feature is that the temperature mostly rises in the morning and falls in the evening. Inflammatory changes in the blood and urine are not detected. The use of aspirin in such patients does not reduce the temperature.

Sleep and alertness disorders. They are manifested by difficulty falling asleep, superficial, disturbed sleep at night and drowsiness during the day. Pathological drowsiness is less common. Sometimes hypersomnia resembles separate symptom complexes: narcolepsy, Kleine-Levin syndrome, Pickwick syndrome and other hypersomnic manifestations. In the mechanism of the development of hypersomnia, a certain role is assigned to the reduction of the activating effect on the cortex of the cerebral hemispheres of the reticular formation, which is localized at the hypothalamic-stem level.

Hypothalamic syndrome with neuropsychiatric disorders is manifested by asthenia, sleep disturbance, and decreased mental activity. Patients have synestopathy, restlessness, hyperpathic syndrome or hypochondriac disorders, characteristic affective disorders, mood changes from depression to elevated background. Often there are crises with the picture of a vaso-vegetative storm, so-called "panic attacks".

When the mesencephalic part of the autonomic nervous system is affected, anisocoria, mydriasis, accommodation disorders, and Argyle-Robertson syndrome occur.

In case of damage to the bulbar department (nn. salivatorii), lacrimation and saliva secretion are disturbed. When the nuclei of the vagus and glossopharyngeal nerves are affected, bulbar paralysis, disorders of respiratory function and cardiac activity occur.

Spinal vegetative syndromes. When the lateral horns of the spinal cord are affected, there

are vegetative crises with vascular and sweaty signs, cyanosis, edema of the extremities, changes in pulse and breathing rates, and dysfunction of the abdominal cavity and pelvic organs.

When the ganglia of the adjacent trunk are affected, a picture of truncitis with causalgias appears. When the upper cervical sympathetic node is damaged, ptosis, miosis, enophthalmos (Horner's syndrome) occurs.

When it is irritated, there is an expansion of the eye slit and pupil (with indro m Pti), paleness of the face, auricle.

Thoracic sympathetic truncated cells and lesions of the stellate node are accompanied by sensitivity disorders of the type of hyperpathy, breathing disorders, palpitations, lability of the pulse, arrhythmia, pain in the neck and head, with radiation to the abdominal areas. Attacks occur according to the type of angina pectoris.

Lumbar sympathetic truncytes are difficult to distinguish from thoracic ones. Sensations of the vegetative type are localized in the abdominal cavity and the corresponding lower limb. Sweating disorders and vascular disorders are observed.

When the segments S3-S5 are affected, there are disorders of the functions of the pelvic organs according to the peripheral type: intohtinentio vera (true urinary incontinence) or ischuria paradoxa (paradoxical urinary incontinence - the release of urine in drops when the bladder is full).

Peripheral autonomic syndromes are observed when the peripheral nervous system is damaged due to damage to autonomic nerve cells, fibers or due to functional changes in their activity. Syndromes of peripheral nervous disorders consist of vegetative and somatic signs. They are most often observed in polyradiculoneuritis, plexitis, mononeuropathies.

- When the tone of the parasympathetic nervous system increases, the following are noted:
- narrowing of the pupils,
- cold, wet, bluish skin,
- bradycardia, decrease in blood pressure,
- asthmatic breathing,
- increased salivation,
- sweating,
- increased acidity of gastric juice, spastic constipation, which is replaced by diarrhea, relaxation of sphincters,
- frequent urges to urinate,
- bright red dermographism, tendency to obesity, edema.

There is apathy, asthenia, depression, tendency to unconsciousness, drowsiness

The state of vagotonia is characteristic of a sleeping person suffering from bronchial asthma.

The sympathicotonia is characterized by:

- shiny eyes, exophthalmos, wide pupils,
- pale dry skin,
- tachycardia, increased blood pressure,
- tendency to waste,
- weakened intestinal peristalsis, tendency to constipation,
- expansion of the bronchi,
- paresthesias, chills,
- unpleasant sensations in the area of the heart, retention of urine and stool.

Such persons do not tolerate sunny weather, bright light, noise, tremors, fear, wakefulness often appear,

In pathological conditions, both departments of the autonomic nervous system do not act antagonistically, but synergistically.

With frostbite (frostbite), the tone of both departments of the autonomic nervous system increases. In shock states - decreases.

Amphotony - balance. Hyperamphotonia is noted during puberty, and hypoamphotonia - during the involution period. Normotonia – balance of both departments of the autonomic nervous system (diphthonia – imbalance).

Vegetative dysfunction can be:

- generalized
- regional - manifested by changes in one visceral system (cardiovascular, digestive, genitourinary, thermoregulation, etc.)

Vegetative dysfunction can have:

- permanent
- paroxysmal character.

It should be noted that the majority of pathological processes in the autonomic nervous system are caused not by loss of its functions, but by irritation, that is, increased excitability of the central and peripheral apparatus. Repercussions are a feature of the autonomic nervous system: disturbances in some parts of this system can lead to changes in other parts.

Methods of research of the autonomic nervous system

Skin manifestations of vegetative disorders: discoloration, erythema, pigmentation, moisture, swelling, greasiness, hair growth (hypertrichosis, alopecia), nail trophic (thickening, delamination, fragility, Mesa strips), trophic ulcers, skin changes after herpes. Determination of iris color and pigmentation, size (mydriasis, miosis), uniformity (anisokoria). Claude-Bernard-Horner syndrome - ptosis, miosis, enophthalmos - occurs when cervical sympathetic nodes are affected.

Vascular reflexes in case of mechanical irritation of the skin. Distinguish between white (sympathicotonia); pink (Eytonia); red, elevator (vagotonia).

Reflex dermatographism – stroke pressure with a sharp object. It disappears when the responsible segments of the spinal cord are affected.

Sweat reflexes are studied when the body is warmed up, the introduction of diaphoretic substances (pilocarpine, aspirin). Minor's method (iodostarch test). Research of electrocutaneous resistance (SHGR - galvanic skin response or evoked skin sympathetic potentials - VSHSP). Study of the permeability of blood vessels (hydrophilicity) by McClure and Aldrich. Pharmacological tests (histamine, adrenaline, etc.). The most convenient is the study of vegetative-cardiac reflexes.

- Study of autonomic and vascular reactions of the skin with the help of ultraviolet radiation
- acceleration and deceleration of the reaction. Suppression of erythema in segmental lesions of the central nervous system.

- Thermal imaging study - objectifies some reflex-vascular reactions during scanning of infrared radiation against the background of viscerocutaneous connections, during pain reactions, etc.

Currently, the study of the "vegetative portrait", which includes:

The study of vegetative tone (special tables) allows to assess the state of the nervous system at rest.

The study of autonomic reactivity - with the help of a large number of cardiovascular tests (for example, Dan'ini-Aschner, Thomas-Roux) - evaluates the speed of response to irritation and recovery after bringing the brain out of a state of rest, which is important for assessing the severity of damage to the central nervous system, prognosis and others

Vegetative maintenance of activity (physical, mental, emotional) - assesses the ability of the central nervous system to long-term support of vegetative reactivity at a certain level.

It is also necessary to palpate the projection of nodes of the sympathetic trunk, plexuses (periarterial, cervical, solar, etc.).

Research of vegetative algic points of Markelov-Birbrair, which are symmetrically located on the sides of the body and occur in the pathology of different departments of the central nervous system. Sliding palpation is performed. Pain reactions are assessed on a five-point scale. Construction of "algic curves" can determine regional, generalized, symmetrical, asymmetrical syndromes.

The study of sensitivity with the help of Zakharyin-Hed zones (viscero-cutaneous ligaments) indicate irritation of the segmental-peripheral visceral apparatus of the ANS.

Identify and differentiate paroxysmal autonomic disorders (dizziness, fainting, acrocyanosis, Quincke's edema, urticaria, vasomotor rhinitis, hypothalamic crises, trigeminal pain, migraine, sleep attacks, autonomic-visceral auras, etc.).

Cardiovascular tests:

The Kerdo index is used to assess tone in the cardiovascular system, the Kerdo index is calculated according to the following formula: $K = (1 - D/P)$, where K is the Kerdo index, D is diastolic pressure, P is pulse rate, eutonia, or vegetative equilibrium is observed when the value of the index is from -3 to +3, sympathicotonia – at values greater than +3, parasympathikotonia – at values less than -3.

The Aschner-Danini pericardiac reflex allows you to assess the autonomic reactivity of the parasympathetic nervous system. The reflex is induced by pressing for 20 seconds with the pads of the doctor's fingers on the front-side surfaces of the patient's eyeballs. Normally, the pulse rate decreases by an average of 8 beats in 1 minute. In the case of vagotonia, the pulse slows down by more than 10 beats per minute, in the case of sympathicotonia it does not change or becomes more frequent.

The Chermak reflex is very similar in mechanism to the Aschner reflex and occurs when the mm is pressed. sternocleidomastoidei.

Thomas-Roux solar reflex – pressing on the cells of the solar plexus projection, the reaction is similar to Aschner's reflex.

Prevel's orthostatic reflex consists in an increase in pulse rate and blood pressure as a result of a change in the position of the body in space - from horizontal to vertical and characterizes the vegetative support of activity. Normally, the pulse increases by 8-12 beats per minute, and blood pressure increases by 5-10 mm Hg.

Danielopolo's clinostatic reflex is characterized by a slowing of the pulse by 6-8 beats in 1 minute and a decrease in blood pressure by 5-10 mm Hg. in response to a change in body position from vertical to horizontal.

Lesions of the autonomic nervous system can manifest as psycho-autonomic disorders. Therefore, they conduct a study of the patient's emotional and personal characteristics, study the mental anamnesis, the possibility of mental injuries, perform a psychological examination using various methods and tests (Staberg, Eysenck, Kettel, Rorschach projective test, etc.) .

Syndromes of dysfunction of the pelvic organs. Regulation of the function of urine excretion consists of two components: involuntary-reflex (at the level of the segmental apparatus of the spinal cord due to autonomic innervation of the non-striated muscles of the internal sphincter and detrusor of the bladder) and voluntary (involving the cerebral cortical zone, somatic fibers and striated muscles ligaments of the external sphincter, urinary canal, anterior abdominal wall and diaphragm of the pelvic floor).

Schematic innervation of the bladder can be depicted as follows:

1. The spinal center of parasympathetic innervation of the bladder is located in the lateral horns of the gray matter of the sacral spinal cord (segments S2 — S4).

Afferent impulses to this center come from the proprioceptors of the bladder wall through intervertebral ganglia S2 —S4, then the internal pelvic nerves (nn. splanchnici pelvici), pelvic nerve and posterior roots. The efferent part of the arc passes through the anterior roots, then the pelvic nerve (n. pelvici) and terminates in the intramural parasympathetic ganglia of the bladder wall. Postganglionic fibers innervate the bladder detrusor and partly the internal sphincter.

2. The center of sympathetic innervation is located in the lateral horns at the level of L1 —

L2 segments of the spinal cord.

Preganglionic fibers leave the spinal cord together with the anterior roots, pass through the sympathetic chain and terminate in the lower mesenteric node, where they pass to the second neuron. Postganglionic fibers in the composition of n. hypogastricus fit the non-striated muscles of the bladder. Efferent sympathetic fibers mainly regulate the lumen of the bladder vessels.

Automatic bladder emptying is provided by two segmental reflex arcs — parasympathetic and somatic. When the walls of the bladder are irritated due to its stretching, impulses are transmitted to the spinal parasympathetic center. From there, they go along the efferent fibers and cause the contraction of the bladder detrusor and the relaxation of the internal sphincter. The entry of urine into the urethra due to the opening of the internal sphincter leads to the start of a reflex arc for the striated sphincter. Relaxation of the external sphincter is accompanied by the release of urine. Such an involuntary call appears if the intravesical pressure increases by 5 mm Hg.

Voluntary regulation of the act of urination is carried out with the participation of the cortical sensory and motor zones of the bladder. Impulses reach the sensory area (gyrus fornicatus) from the sensory neurons of the intervertebral nodes S2-S4 through the posterior roots, posterior cords, and through the medulla oblongata. Via associative fibers, impulses from the sensory zone are transmitted to the motor neurons of the paracentral lobe, and then, as part of the pyramidal path, they reach the neurons of the anterior horns S2-S4. Further, the impulses go along the front roots through the genital plexus and in the composition of n. pudendus reach the external sphincter. Voluntary regulation of urine output includes, in addition to controlling the external sphincter of the urethra, also controlling the muscles of the abdomen, diaphragm, and pelvis.

In the case of a lateral violation of the cortical-spinal connections (transverse damage to the spinal cord at the cervical or thoracic level), a pathology of voluntary regulation of urine output occurs. If the disorder occurs acutely, retention of urine (retentio urinae) first develops. In the future, in connection with the increase in reflex excitability of the segmental apparatus of the spinal cord, urinary retention is replaced by periodic incontinence (incontinentio intermit tens).

In milder cases, imperative urges to urinate are observed.

In the case of acute processes, in which the parasympathetic innervation of the bladder is disturbed, there is a delay in the release of urine. Denervation of the internal and external sphincters is accompanied by true urinary incontinence (incontinentio vera). Such incontinence develops in case of damage to the sacral segments of the spinal cord, the roots of the horse's tail, n. hypogastricus and n. pudendus.

Paradoxical urinary incontinence (ishuria paradoxa) occurs in the presence of elements of urinary retention (the bladder does not empty arbitrarily) and urinary incontinence due to mechanical overstretching of the sphincter.

The mechanism of violation of the act of defecation is the same as that of urinary incontinence. Delayed bowel movements indicate bilateral damage to the pyramidal tract. Faecal incontinence can occur simultaneously with urinary incontinence.

Bilateral damage to the pyramidal tracts or peripheral spinal centers at the level of the sacral segments of the spinal cord causes a violation of sexual intercourse.

1. Questions for self-control:

1. How is the autonomic nervous system divided?
 - a) cortical;
 - b) striary;
 - c) pretty;
 - d) parasympathetic.

Answer : c, d.

2. Where are the higher vegetative centers located?
 - a) in the cortex;
 - b) in the thalamus;

- c) in the hypothalamus;
- d) in the medulla oblongata.

Answer: c

3. What vegetative samples do you know?
- a) dermographism;
 - b) sample of Romberg's test;
 - c) Stewart-Holmes test;
 - d) ortho-clinostatic test.

Answer: a, d.

2. Tests for self-control:

First level (with a single selected answer):

1. What belongs to the autonomic nervous system?
- a) hypothalamus;
 - b) cerebral cortex;
 - c) thalamus;
 - d) medulla oblongata.

Answer: a.

2. What departments is the autonomic nervous system divided into?
- a) sympathetic;
 - b) parasympathetic;
 - c) striate;
 - d) pallidary.

Answer: a, b.

3. Methods of studying the autonomic nervous system include:
- a) study of surface reflexes;
 - b) research of deep sensitivity;
 - c) study of dermographism;
 - d) study of statics and coordination of movements.

Answer: c

B. Tests for self-control with standards of answers.

1. Is research done to diagnose disorders of the animal nervous system?
 2. To diagnose a violation of the autonomic nervous system, is it investigated?
- a) muscle tone ;
 - b) coordination of movements;
 - c) reflexes;
 - d) pilomotor reaction of the skin;
 - e) clinorhthostatic test.

**Answer: 1- a, b, c.
2 - d, e.**

1. What is observed when the parasympathetic nervous system is irritated?
 2. What is observed when the sympathetic nervous system is irritated?
- a) muscle weakness ;

- b) tachycardia;
- c) bradycardia;
- d) violation of coordination of movements.

Answer: 1 - c

2 - b.

1. What are the most typical symptoms of parasympathetic irritation nervous system?
2. What are the most typical symptoms of sympathetic irritation nervous system?
 - a) tachycardia;
 - b) bradycardia;
 - c) increase in blood pressure;
 - d) decrease in blood pressure;
 - e) hyperemia of the skin;
 - g) paleness of the skin.

Answer: 1 - b, d, e.

2 - a, c, g.

B. Tasks for self-control with answers.

The patient received a closed craniocerebral injury. After some time, he began to complain of headache, sweating, palpitations. An objective examination revealed tachycardia - a pulse of 110 per second, fluctuations in blood pressure from 110/70 to 90/50 mmHg, hyperhidrosis of the distal parts of the limbs, red diffuse dermographism, asthenization.

Make a diagnosis and determine the topic of the lesion.

Answer: Post-traumatic vegetative-vascular dystonia.

Fire damage - hypothalamic zone.

A list of educational practical tasks that must be completed during the practical session.

1. To study dermographism.
2. To study the pilomotor reaction.
3. To study the hydrophilicity of the skin.
4. Investigate the Danini-Ashner reflex.
5. Study the clino-orthostatic test.

Literature:

Basic

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Additional

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ISW No. 4

Topic: Functional diagnosis of diseases of the nervous system.

Relevance of the topic: Electrophysiological methods of examination of the nervous system (both peripheral and central department) are extremely important in the diagnosis of various neurological injuries. Therefore, the ability to correctly apply and interpret one or another examination significantly expands the doctor's capabilities and creates favorable conditions for the appointment of a specific and adequate treatment of patients. It is impossible to diagnose tumors, brain injuries, inflammatory and vascular diseases, demyelinating diseases and a number of others without X-ray and liquefaction studies. Therefore, the wide implementation of these methods in medical practice ensures correct diagnosis, and hence, timely and adequate treatment.

Lesson goals:

To acquaint applicants with the main electrophysiological and X-ray methods of studying the nervous system and indications for their use.

Lesson content.

Radiological examination of the skull and spine

Conventional radiography of the skull and spine is an integral part of a detailed examination in cases of pathology of a traumatic, spondylogenic and neoplastic nature, but has a rather limited value in other diseases. The technique is relatively simple, and the obtained data can be interpreted by most radiologists. More important in neurology and neurosurgery are six special radiological methods, which currently make it possible to visualize most parts of the brain and spinal cord, as well as their vessels.

Computed tomography (CT). CT allows differentiation of epidural, subdural, and intracerebral hemorrhages, changes in the ventricular system with volume processes, and also detects tumors, abscesses, granulomas [when CT is performed after intravenous administration of contrast agents], areas of cerebral edema, foci of infarction, hydrocephalus, and atrophy brain The simplicity of this non-invasive procedure, the minimal risk for patients with large lesions, and the low dose of X-ray radiation revolutionized diagnostic neurology and neurosurgery.

NMR tomography. Modern use of nuclear magnetic resonance (NMR) allows visualization of brain lesions that are not detected by CT. The NMR-tomography method is non-invasive and does not involve the influence of ionizing radiation. This study provides the possibility of tissue delineation without contrast enhancement, and since bone tissue does not interfere, the method is particularly convenient for visualizing the basal structures of the brain, primarily in the posterior cranial fossa. The NMR method has already significantly expanded the diagnostic possibilities of neuroradiology, and in the future, NMR spectroscopy should become a method of determining metabolites in the brain. The high resolution of NMR-tomography in distinguishing between white

and gray matter determines its wide application in order to establish the localization of lesions in the white matter, for example due to demyelination. The method is also used to visualize the spinal cord, both on sagittal and transverse "slices".

Angiography. This method has been refined over the past 30 years until it has become relatively safe and extremely valuable for the diagnosis of arterial occlusions, aneurysms and vascular malformations, tumors, abscesses, and intracranial hemorrhages. But, after the discovery of CT and NMR tomography, it began to be used much less often. After local anesthesia, a needle or cannula is inserted percutaneously into the lumen of the brachial or femoral artery; it is possible to introduce a catheter, passing it through the aorta with cannulation of the main arteries in the neck. The introduction of a contrast agent allows visualization of the aortic arch, the initial sections of the carotid and vertebral blood supply systems, the passage of blood vessels through the neck region into the skull cavity and, with appropriate indications, the arteries of the spinal cord. Under optimal conditions, it is possible to clearly show cerebral arteries with a lumen diameter of up to 0.1 mm, as well as small veins of a similar diameter, vascular anomalies (angiomas, aneurysms), occluded arteries, slowing of blood flow due to increased intracranial pressure at volume processes and blockages of the sinuses of the dura mater and veins, the displacement of vessels by a volumetric process or the impossibility of filling intracranial vessels during brain death. Digital subtraction venous angiography, in which a contrast agent is injected into the brachial vein under pressure, is considered as an alternative or additional method, especially if the initial examination of the diameter of the lumen of large extracranial arteries is necessary.

Pneumoencephalography and ventriculography. The introduction of air into the subarachnoid space during a lumbar puncture, performed in the patient's sitting position, enables detailed visualization of the size and location of the ventricles, the subarachnoid space (superior spinal and cerebral) and, indirectly, the structures located between the ventricles and the meninges. However, this method is rarely used at present. Ventriculography, which was carried out by injecting air or a contrast agent directly into the lateral ventricles, also has mainly historical significance. CT and NMR tomography have practically replaced both pneumoencephalography and ventriculography.

Radioactive isotopes. Radioactive isotopes, such as technetium (brain scan), are sometimes used to diagnose tumors, voluminous processes of inflammatory genesis, viral encephalitis, and some vascular lesions, such as heart attacks in areas adjacent to the blood supply of the brain, the detection of which is difficult by other methods. Since this method is simple and non-invasive, the only limitation to its use is the high cost. The more significantly the blood-brain barrier is affected by the pathological process, the more clearly the affected area is determined when using these methods. In order to detect the displacement of the middle structures of the brain by a volumetric process, ultrasound can also be used.

Positron emission tomography

Positron emission tomography (PET) is an experimental research method currently available at the disposal of only a few centers. The procedure includes systemic administration of positron-emitting radionuclides of oxygen or ^{18}F -deoxyglucose (^{18}FDH) followed by computer tomography. Administration of labeled O_2 , CO_2 , and ^{18}FDH makes it possible to quantitatively assess regional oxygen consumption, blood flow, and glucose utilization. An examination of patients with cerebrovascular lesions, convulsive disorders, degenerations was carried out. With a stroke, it is advisable to perform PET in the acute stage in order to distinguish between viable and non-viable tissue. In patients with epileptic manifestations, the study with ^{18}FDH in the inter-seizure period allows to detect areas of reduced glucose metabolism in and around the epileptic focus, while during the seizure, an increase in glucose metabolism is registered. During the study of metabolic processes using ^{18}FDG , a decrease in glucose uptake in the striatum was also demonstrated in patients with Huntington's chorea, in whom no pathological changes were detected on CT.

Electromyography (EMG)

This technique is carried out during the clinical examination of patients with neurological

diseases in which the neuromuscular apparatus suffers, that is, with primary and secondary lesions of the skeletal muscles. Advanced EMG technique ("central EMG") provides a quantitative analysis of motor system function.

Electroencephalography (EEG)

EEG is part of the clinical examination of a patient with suspected brain damage; it is also used to assess the involvement of the central nervous system (CNS) in many diseases.

In addition to recording at rest, so-called tests with stimulation are usually conducted.

1. The patient is offered to make deep breathing movements with a frequency of 20 times per 1 minute for 3 minutes. Developing alkalosis and cerebral vasoconstriction can provoke characteristic signs of convulsive readiness and other changes.

2. A powerful light source (strobe) is placed in front of the patient's face, which flashes with a frequency of 1 to 20 in 1 s, while the patient's eyes are open, then closed. As a result, patients with photosensitivity may develop pathological discharges.

3. EEG is recorded after the patient is allowed to fall asleep naturally, or after oral or intravenous administration of sedative drugs. Procedures 1 and 2 are used more often, but recording during sleep is exceptionally effective in detecting changes, especially in cases of temporal lobe epilepsy and some other convulsive conditions. A frequent reason for nocturnal EEG registration is previous research on sleep disorders.

In order for the EEG to be most effective, it is necessary to observe certain conditions. The patient should not be under the influence of sedative drugs, remain for a long time without eating, since both sedatives and relative hypoglycemia lead to changes in the normal structure of the EEG. The same applies to states of mental concentration, hyperexcitability, and drowsiness, each of which contributes to suppression of the normal alpha rhythm and amplification of muscle artifacts. When examining patients with a possible diagnosis of epilepsy who are already receiving treatment for this condition, most clinicians prefer to conduct the first EEG recording against the background of continued medication.

Types of normal records. In a normal EEG in adults, somewhat asymmetric sinusoidal alpha waves (frequency 8-12 Hz, amplitude 50 μ V) are registered in the occipital and parietal regions. These waves rise and fall spontaneously and usually disappear quickly when the subject opens his eyes or focuses his attention on something. Faster waves with a frequency higher than 13 Hz and less high amplitude (10-20 μ V), called beta waves, are determined symmetrically in the frontal regions. Very slow waves (delta waves), sharp waves and other unusual fluctuations are absent in a normal recording. When healthy subjects fall asleep, the oscillation rhythm is symmetrically expressed and characteristic waves appear (sharp waves and sleep spindles in the parietal regions); if the sleep is caused by barbiturates and benzodiazepines, then an increase in high-frequency activity is determined, which is considered normal (see ch. 20). In case of excessive high-frequency activity, it should be clarified whether the patient is taking any drugs of these groups.

During stroboscopic stimulation, a normal EEG may show a reaction of the occipital cortex to light flashes, called an evoked reaction or, at a higher rate of their repetition, a reaction of learning the rhythm of photostimulation. The clinical significance of the evoked response of the occipital cortex lies in the fact that it expands the diagnostic capabilities of EEG in several ways: 1) it gives the researcher logical confidence that the subject at least perceives light, 2) if the evoked response is absent in one hemisphere, but is determined in the other, then this serves as a physiological confirmation of the presence of a lesion that prevents the normal conduction of impulses from the optic tubercle to the occipital lobe, which is localized on the same side, 3) if light flashes cause pathological waves, then this indicates increased excitability. If the EEG activation procedure is continued, true convulsive discharges may be provoked ("photoparoxysmal" reaction); in the case of even higher sensitivity, pronounced myoclonic twitching of the muscles of the face and upper limbs, generalized convulsions may appear. These phenomena should be distinguished from a purely muscular reaction, also of a myoclonic nature, which is normally caused by the contracting muscles of the scalp and is often visible during routine

EEG (photomyoclonus).

Types of pathological records. The most pronounced disturbance is the disappearance of the EEG curve and its replacement by "bioelectric silence of the brain", which means that the electrical activity of the cerebral cortex recorded from the scalp is less than $2\text{ }\mu\text{V}$ or absent. A similar isoelectric EEG can be induced by anesthetic doses of drugs such as barbiturates and by profound hypothermia (less than 70°F). However, in the absence of CNS depressant drugs or deep hypothermia, the "flat" type of recording (except for artifacts) in all leads is almost always the result of cerebral hypoxia, ischemia, or widespread cortical destruction. It is believed that if the patient does not have EEG activity, reflexes, spontaneous breathing and any muscle activity for 6 hours or more, one should talk about "irreversible coma". Such patients have extensive brain necrosis. It is impossible to restore the function of the central nervous system, in connection with which the patient can be considered dead, despite the preservation of vegetative (cardiovascular) functions, supported by mechanical devices, for example, respirators. None of the more than 900 patients examined over the past 18 years in a multidisciplinary clinic in Massachusetts was an exception.

Sometimes local areas of absence of EEG activity are observed in cases of large heart attacks, massive superficial brain tumors or in the presence of clots located between the cortex of the large hemisphere and the electrodes. These changes make it possible to precisely establish the localization of the pathological focus, but, of course, it is impossible to judge the nature of the process by them. In most cases, the sizes of the pathological foci are too small to be directly registered with the help of EEG, but the obtained curve can record pathological waves originating mainly from functionally rather than organically changed areas of the brain around the affected area. These pathological waves are slower and higher in amplitude ($50\text{--}350\text{ }\mu\text{V}$) than normal ones. Waves with a frequency of less than 4 Hz are called delta waves, from 4 to 7 Hz - theta waves; higher voltage and fast waves are designated as spikes or sharp waves. Sometimes fast and slow waves are observed simultaneously; when series of such waves suddenly, in the form of paroxysms, interrupt a relatively normal EEG recording, there is every reason to suspect epilepsy. Patients with petit mal (absence) seizures are characterized by the appearance of "peak-wave" complexes with a frequency of 3 Hz in all EEG leads at the same time, followed by their sudden disappearance at the end of the attack.

Neurological conditions with pathological EEG. In the following forms of neurological diseases, EEG is essential for making the correct diagnosis.

Epilepsy. All types of generalized epileptic seizures (major and minor) are accompanied by certain periodically recorded electroencephalographic changes. Pathological changes in the EEG are often detected also in more limited types of epileptic seizures (complex partial, myoclonic, focal and Jacksonian). The only exception is some deep foci in the temporal lobe, the amplitude of discharges from which when approaching the scalp is insufficient to manifest against the background of physiological EEG activity, especially in the presence of a pronounced alpha rhythm. It is possible to determine the localization of an epileptic focus in the medial part of the temporal lobe with the help of the zygomatic and sphenoidal leads; the changes revealed at the same time are sometimes the only confirmation of epileptic activity during EEG. Among other exceptions, in which it is sometimes not possible to register EEG changes during a seizure, focal seizures (sensory, Jacksonian, myoclonic) are included. Probably, this fact means that the neuronal discharge is very deep in localization, discrete, short and asynchronous to be transmitted by volumetric conduction through the skull and recording by EEG electrodes located at a distance of about 2 cm from the cortex of the large hemispheres. Electroencephalographic manifestations of small, some myoclonic and large seizures are closely correlated with the clinical forms of paroxysms and can be detected in the period between attacks. Artifacts due to motor activity during a "seizure" are usually indistinguishable from electrical brain activity. Differentiating psychogenic seizures from true epileptic seizures requires careful EEG analysis at the beginning of the seizure, when characteristic high-frequency activity can be detected, or immediately after the cessation of convulsive movements, when post-seizure slowing and suppression of waves must be determined

in the case of a disordered state of consciousness. A normal alpha rhythm in the post-seizure period in an "unresponsive" patient suggests a psychogenic pseudoepileptic attack.

It is important to note that 20% of patients with minor and 40% of patients with generalized epilepsy have a normal EEG between attacks. When treated with anticonvulsants, there is also a tendency to decrease the severity of EEG changes. In the other 30-40% of patients with epilepsy in the periods between attacks, EEG abnormalities are detected, but they are of a non-specific nature, therefore, the diagnosis of epilepsy should be made only on the basis of accurate interpretation of clinical data in combination with EEG changes.

Tumor, brain abscess and subdural hematoma. Approximately 90% of patients with clinically manifest intracranial volume formations also have EEG changes. These changes depend on the localization and form of the pathological process. In addition to diffuse disorders, focal and localized slow waves (usually of the delta range) are considered classical in such cases, sometimes epileptic activity with a decrease in amplitude and synchronization of a normal rhythm is determined. The highest frequency of changes in EEG is observed in more rapidly increasing in size, bulky formations (abscess, some metastases, glioblastoma), especially in the case of their supratentorial localization (in 90-95% of cases in the last two, 100% - in abscesses). Slower-growing tumors (astrocytomas) and primarily neoplasms located outside the large hemispheres of the brain (meningiomas, pituitary tumors) often do not show EEG abnormalities, despite the presence of clear clinical manifestations. Electroencephalographic changes have a clear lateralization in 75-90% of patients with supratentorial tumors and abscesses, focal EEG abnormalities in brain metastasis can be detected when the metastasis is not yet visible on CT. Normal EEG results, as well as CT scan, practically rule out supratentorial tumors or brain abscess. Meanwhile, the EEG is normal in 20-25% of patients with subtentorial tumors.

Vascular diseases of the brain. Both diffuse and local changes on the EEG caused by vascular lesions of the brain (heart attacks and intracranial hemorrhages) mainly depend on their localization and size, and not on the nature of the pathological process. Shown. that EEG should be performed for the differential diagnosis of vascular hemiplegia. When the lesion is localized in the basin of the internal carotid artery, the area of reduced normal activity or excessive slow-wave impulses is almost always clearly defined in the corresponding area. If hemiplegia is caused by a lesion of a small-caliber vessel, that is, a lacunar infarction localized in the deep parts of the hemispheres or the brain stem, then the EEG pattern is usually normal. Extensive hemispheric foci, in which a sharp depression of consciousness is noted, also cause the appearance of widespread, diffuse slow-wave activity of a nonspecific nature, the same as that determined in stupor or coma of any etiology. Changes begin after a few days, when brain swelling decreases, and focal activity (slow-wave activity or inhibition of physiological rhythms) may be noted. Infarcts of smaller sizes are accompanied by pronounced focal changes, which clearly indicate the lateralization of the focus, but do not allow its exact localization. Unlike tumors, the improvement continues, and after 3-6 months, in almost 50% of patients with acute cerebrovascular pathology, the EEG normalizes, despite the presence of a persistent neurological defect. In a similar situation, the prognosis is unfavorable. The subsequent detection of moderate or high-amplitude waves on the EEG, and especially spikes or sharp waves, indicates the presence of pathologically functioning tissue that may have epileptogenic properties. With an acute subarachnoid hemorrhage, the EEG can provide useful information about the side of its localization, while the severity of the changes depends on the degree of impact on the underlying brain tissue.

Brain injury. In the event of a stroke or rupture of the brain, changes similar to those in cerebrovascular pathology appear on the EEG. Diffuse changes often give way to focal changes, especially when lesions are localized on the lateral or upper side of the cerebral hemispheres. If there is no epileptic activity, the focal changes usually disappear after several weeks or months. Sharp waves or spikes sometimes appear after the disappearance of focal slow-wave changes. These disturbances or lack of EEG normalization usually precede the development of post-traumatic epilepsy. Therefore, performing an EEG in dynamics after a traumatic brain injury is

valuable for determining the prognosis of epilepsy.

Diseases that cause coma and disorders of consciousness. In almost all conditions accompanied by some disturbances of consciousness, the EEG has a pathological character. In hypothyroidism, the rhythms are of a normal configuration, but are usually slow. In general, the deeper the disorder of consciousness, the more the EEG picture is changed. With pronounced disturbances of consciousness, bilateral, high-amplitude slow (delta) waves are detected, which are more noticeable in the recording from the frontal regions. This applies to such different conditions as acute meningitis and encephalitis, sudden changes in gas composition, blood glucose content, water-electrolyte balance, uremia, diabetic and hepatic coma, and loss of consciousness in massive brain lesions. With hepatic coma, the severity of changes on the EEG corresponds to the stages of confusion, stupor or coma. In addition, paroxysms of bilateral synchronous large sharp "triphasic waves" are characteristic, although they can also be present in other metabolic encephalopathies against the background of renal and pulmonary insufficiency. Diffuse degenerative lesions of the cerebral cortex (for example, in Alzheimer's disease) are accompanied by relatively weak diffuse slow waves of the theta range (4-7 Hz). In more rapidly progressing diseases, such as subacute sclerosing panencephalitis (PSPE), Creutzfeldt-Jakob disease and, to a lesser extent, cerebral lipidoses, very characteristic, almost pathognomonic EEG changes are also observed, consisting of repeated complex bursts of activity in the form of acute and slow waves. A normal EEG in a patient with apathy, lethargy, depression or memory loss is one of the evidences in favor of the diagnosis of affective disorder or schizophrenia.

EEG can also help the doctor manage a comatose patient. According to the results of the EEG, one can suspect such causes of coma as hepatic encephalopathy (bilateral synchronous triphasic waves), intoxication with barbiturates or benzodiazepines (excessive rapid activity), clinically prolonged epileptic discharges occupying a large volume of the lesion, diffuse anoxia-ischemia (a sign of "flash-suppression" with repeated generalized complexes separated by periods with a very low amplitude of the EEG curve).

Other brain lesions. Many disorders of the functions of the nervous system cause only weak changes on the EEG or are not accompanied by changes at all. Similar examples are multiple sclerosis and other demyelinating diseases, although in their advanced stages the EEG is pathological in 50% of patients. Alcoholic delirium, disease. Wernicke-Korsakov and withdrawal syndrome seizures are accompanied by minor changes in the EEG, despite the dramatic nature of the clinical picture, and sometimes the EEG may not be changed at all. Some slow-wave activity usually accompanies a twilight state, which is referred to as hypokinetic delirium. It should be noted that neuroses and psychoses, such as manic-depressive disorders and schizophrenia, pathological conditions caused by hallucinogenic drugs such as LSD, and most cases of mental retardation are accompanied by minor or nonspecific changes.

Special application of EEG. Since the EEG provides information about the state and functioning of the brain, it is appropriate to use it for monitoring in the operating room when monitoring the viability of the brain during modern cardiac surgical interventions, which are becoming more and more extensive. For a long time, electroencephalographic equipment was used to determine the level of anesthesia. The use of simple equipment by anesthesiologists for monitoring the functions of the heart and brain in patients during surgical operations has not lost its importance even today.

Continuous EEG monitoring during carotid endarterectomy is a common practice these days - a manipulation performed on carefully selected patients suffering from stenotic or ulcerative lesions of the carotid artery. Characteristic changes in the EEG (especially a significant decrease in voltage) indicate the need for temporary anastomotic shunting to maintain sufficient cerebral blood flow to eliminate ischemic damage to the brain during surgery.

In neurosurgical operating rooms, EEGs can be recorded from the bare brain (electrocorticograms), while the focus of epileptic activity is more accurately localized than when recording from the scalp, which makes resection of functionally altered tissue possible.

Routine EEG is of diagnostic value in hysterical blindness. Similarly, a response to noise during daytime sleep can help confirm the presence of auditory perception in a patient simulating total deafness. Examining these responses may also be useful for evaluating hearing and vision in newborns.

Evoked potentials

The method of evoked potentials (EP) is a method of recording the electrical activity of groups of neurons in the spinal cord, brainstem, optic tubercle, and large hemispheres after stimulation of one or another afferent system by visual, auditory, or tactile influences. The amplitude of these potentials, recorded from the scalp using conventional electroencephalographic electrodes, varies from 0.5 and less to 20 μV . Due to their extremely small size, they can rarely be registered on an electroencephalography with ink sand against the background of the main electrical activity of the brain, which usually reaches an amplitude of 50 μV or more. Therefore, special equipment, including simple computers, is needed to extract from the main EEG curve the wave-like response that is of interest to the researcher. This technique is called "averaging" because the procedure involves repeating 100-1000 precisely timed stimuli and recording the electrical activity in a certain short interval after each stimulus. Further spontaneous activity on the EEG at each given moment of time after the stimulus can be negative, and at other moments - positive in its polarity, tends to be extinguished with a sufficient number of repetitions. On the other hand, the evoked response has stable temporal characteristics in relation to the stimulus and at a specific moment after the stimulus always appears as an electrical signal of the same shape. In this connection, the VP increases as repetitions occur, while the amplitude of the background curve decreases. It is important to use special amplifiers, to place the electrodes on the surface of the scalp with great accuracy, to give stimuli precisely on time and to minimize accompanying electrical artifacts. VP expand the possibilities of clinical neurological research of the corresponding afferent system, make it more sensitive and objective, but are not a more specific method of etiological diagnosis.

Visual evoked potentials. Visual evoked potentials obtained with the help of pattern alternation (VEP) have the longest history of clinical use. In the course of the study, patients are shown a reversible black-and-white checkerboard pattern projected on the screen. When the patient examines the alternating pattern, a characteristic wave-like impulse is formed in him, which can be registered from the scalp of the back of the head. Normally, this three-phase wave has a distinctive positive peak with a latency period of 95-115 ms (usually denoted as P100; Fig. 341-5) from the moment of pattern reversal. The duration of this latent period, the duration of the response and the peak amplitude are measured. The latent period is the most important clinical parameter. Research is carried out separately for each eye. Purely monocular changes indicate the presence of a conduction defect below the chiasm.

Many forms of lesions of the optic nerve fibers with changes in the ChPZVP. Glaucoma, compression of the optic nerve, chiasm or tract by various volumetric formations, degenerative lesions of the optic tract often cause a decrease in the amplitude and/or an increase in the latency of the response. With a significant damage to the visual system, it is not possible to register reactions to stimulation of one or both eyes. In clinical practice, optic neuritis, often associated with multiple sclerosis, is the most common cause of changes in the CSFV. With demyelination of optic nerve fibers, both in primary demyelinating disease and in the pathological processes listed above, a slowing of conduction along nerve fibers and an increase in the latency of the positive peak of ChPZVP (up to 115-200 ms) are detected. Indeed, almost all patients with optic neuritis, even after the restoration of visual acuity to normal, have characteristic changes in the LVEF, while no abnormalities are detected during a detailed ophthalmological examination. If in patients with multiple sclerosis, the LVEF is within the normal range, then no abnormalities are detected during neuro-ophthalmological examinations. When LVEF is pathological, a significant number of patients have no abnormalities in visual fields, visual acuity, pupillary reactions, and fundus.

Approximately 50% of patients with multiple sclerosis, in whom the visual function has

never been impaired, also find abnormalities on the part of ChPZVP, which indicates the high efficiency of this method in this disease. If the patient consults a doctor about the first episode of a neurological disease, in which the focus of the lesion is localized in the brain stem or spinal cord, then in the case of the detection of the pathological nature of the CPZVP or other does not clinically manifest the focus of the lesion in another department of the CNS (optic nerves), the diagnosis becomes more likely multiple sclerosis, which frees the patient from the need for a number of neuroradiological examinations.

Changes in visual acuity are not reflected on the CHPZVP until vision deteriorates so much that the patient is unable to distinguish a checkerboard pattern; patients with visual acuity of 20/200 and above can undergo examination. The only additional requirement is the patient's sufficient ability to interact and remain calm in a sitting position for 20 minutes while he examines the pattern. Newborns and children can also undergo this examination, but at the same time special methods are used.

Stem auditory evoked potentials. Trunk auditory evoked potentials (AEPs) are more difficult to study than AEPs, as they are much lower in amplitude, within 0.5 μ V. They are caused by sound clicks that stimulate one ear through an earpiece. The patient can be both asleep and in a comatose state. It should be noted only that excess movements and muscle artifacts make it difficult to get an answer.

Somatosensory evoked potentials. Somatosensory evoked potentials (SEPs) are evoked by small, painless electrical stimuli directed at large sensory fibers of mixed nerves of the upper and lower extremities. The afferent impulse is registered at many levels as it travels towards the center; series of waves can be recorded that reflect activity in the trunks of peripheral nerves that conduct pathways of the spinal cord, the nuclei of the thin and sphenoid fascicles, the structures of the pons and / or cerebellum, as well as the optic tubercle, thalamocortical pathways, and the primary sensory fields of the cortex of the large hemispheres (see Fig. 341-5). Damage to these conductors at any level leads to changes in subsequent waves, which allows to establish or confirm the localization of the pathological process, similar to the way it is done in the study of SSVp.

Angiography is a valuable method that provides an X-ray image of brain vessels after the introduction of an X-ray contrast agent into them. is carried out with the aim of clarifying the localization of the pathological focus, clarifying its nature and character.

Brief information about the methods, the principles on which they are based and the main indications for use are given below

Electromyography

Principles of the method: Assessment of the functional state of nerves and muscles. Determination of the electrical activity of muscles, lesions of the neuromotor apparatus (primary muscular, neural, anterior horn lesions, peripheral nerve lesions)

Indications for use: Hereditary myopathy, myotonia, radiculoneuritis, extrapyramidal hyperkinesia, mono-, polyneuritis, axonal, demyelinating neuropathies

Evoked potentials

Principles of the method: Stimulation of receptors of the sensory system (visual, auditory, somato-sensory) causes an electrical signal in the corresponding area of the cortex

Indications for use: Retrobulbar neuritis, multiple sclerosis, brainstem glioma, brainstem infarctions, spinal cord tumors, myelopathy

Electroencephalography

Principles of the method: The method of recording spontaneous electrical activity of the brain using electrodes attached to the surface of the head. Determination of the functional activity of the brain, localization of the pathological focus in the brain, monitoring of the pathological

process

Indications for use: Epilepsy, tumors, vascular, inflammatory, degenerative diseases of the brain, comatose states

Echoencephalography

Principles of the method: The method of echolocation inside the cranial space, which is based on the property of ultrasound to be reflected at the boundary of the environment endowed with different physical properties (skull bones, blood, cerebrospinal fluid, brain matter)

Indications for use: Tumors, abscess, edema, brain hematoma

Ultrasound dopplerography

Principles of the method: Non-invasive study of extracranial carotid and spinal vessels, as well as determination of the linear speed of blood flow and its direction. Study of the difference in the frequency of reflected ultrasound waves, which depends on the speed of blood circulation - the Doppler effect

Indications for use: Atherosclerosis, nonspecific aortoarteritis, deformation and stenosis of an aneurysm of extracranial vessels, angiodysplasia, extravasal compression of arteries

Craniography

Principles of the method Determination of the state of the skull as a whole: base of the skull, Turkish saddle, cranial sutures, facial skeleton

Indications for use: Craniocerebral trauma, voluminous intracranial neoplasms, hyperostosis of skull bones, neurinoma VIII and optic nerve, pituitary neoplasm

Brain angiography

Principles of the method Invasive method of imaging vessels using a series of craniograms during intra-arterial injection of a radiopaque substance

Indications for use: Congenital anomalies of vessels, arterial aneurysms, tumors, hematomas, abscesses, parasitic cysts

Spondylography

Principles of the method Determination of the walls and lumen of the spinal canal, intervertebral openings, shape, structure, contours of the vertebral bodies, their arches, spinous and transverse processes, as well as the condition of the intervertebral discs and joints

Indications for use: Sacralization of the V lumbar or lumbago of the I coccygeal vertebrae, spondylopathy, compression fracture of the vertebral body, tumors, spondylolisthesis, ankylosing spondylitis

Myelography

Principles of the method Contrast X-ray examination of the spinal cord (introduction of iodine-containing solutions through a lumbar puncture into the spinal canal)

Indications for use: Compression of the spinal cord or roots (intramedullary tumors, disc herniation, abscess, cyst)

Computed tomography

Principles of the method The use of X-rays, with the help of which we obtain sections of the brain and spinal cord in the axillary (horizontal) plane at different levels with subsequent processing on a computer

Indications for use: Hematomas, meningiomas, cysticerci, foci of calcification, ischemia, glial and metastatic tumors, cysts, herniated discs, hydrocephalus, brain atrophy, pathology of skull bones, vertebrae

Magnetic resonance imaging

Principles of the method: Use of nuclear magnetic resonance for a more detailed image of the brain and spinal cord in axial, frontal, sagittal projection in T1 and T2 modes

Indications for use: Multiple sclerosis, demyelinating diseases, pituitary adenoma, congenital brain anomalies, Arnold-Chiari anomalies, hydrocephalus, arteriovenous malformation, brain ischemia (in the first 48 hours), cerebral hemorrhage in the subacute period, dysplasias

Positron emission tomography

Principles of the method: Imaging of the brain (radioisotope diagnostics) using radiopharmaceuticals labeled with positron-emitting isotopes. Study of brain vital processes, including glucose metabolism and oxygen utilization, assessment of blood circulation and perfusion

Indications for use: Differentiation of malignant and benign tumors, Parkinson's disease, Huntington's disease

Questions for self-control:

1. Is the blood supply to the brain ensured?

- a) by the system of the internal carotid artery and the artery of Adamkevich ;
- b) vertebro-basilar and anterior spinal artery;
- c) internal carotid artery system and vertebral-basilar system.

Answer : c.

2 . What electrophysiological methods are used to diagnose hydrocephalus syndrome?

- a) EEG;
- b) Echo-EG;
- c) REG;
- d) EMG.

Answer: b.

3. a) Indications for spinal puncture?

- b) In which diseases are the symptoms of irritation of the meninges most often observed;
- c) Indications for angiography?

Answer:

- A) Symptoms of irritation of the meninges.
- B) They are most often observed in meningitis, subarachnoid hemorrhages.
- C) Suspicion of a volumetric process in the brain, aneurysms of its vessels, occlusive processes.

Tests of the second level (with several selected answers).

- 1. What rhythms on the EEG are considered the main ones?
- 2. What rhythms on the EEG are considered pathological?

- a) alpha rhythm ;
- b) beta rhythm;
- c) delta rhythm;
- d) gamma rhythm.

Answer: 1- a, b.

2 - c

1. What are the indications for prescribing REG?
2. What are the indications for the appointment of Echo-EG?

- a) cerebral atherosclerosis;
- b) hydrocephalus syndrome;
- c) stroke;
- d) fluid dynamics disorders.

Answer : 1 - a, c.

2 - b, d.

B. Tests for self-control with standards of answers.

1. Is a voluminous process of the brain observed on Echo-EG?

- a) "M-ekha" dislocation;
- b) hypertensive-hydrocephalic syndrome;
- c) hypertensive CSF syndrome;
- d) normotensive-CSF syndrome;
- e) there will be no "M-ekha" dislocation.

Answer: a, b.

B. Tasks for self-control with answers.

1. The patient received a closed TBI, after some time convulsive papades appeared.

On the ECG, there is a focus of pathological activity (high-amplitude waves in the alpha range, pathological waves) in the left parietal-temporal zone.

What syndrome has developed in the patient?

Answer : Post-traumatic epileptic syndrome.

2. The patient complains of constant headache, vomiting. On the external bottom - congestive nipples of the optic nerves. An X-ray of the skull shows an increase in finger depressions. During the puncture - an increase in the pressure of the cerebrospinal fluid: the liquid is transparent, protein-cell dissociation is determined.

What do auxiliary research methods indicate?

Literature:

Basic

1. Neurology: study guide / [I.A. Hryhorova, L.I. Sokolova, R.D. Gerasymchuk, A.S. Son, etc.] edited by I.A. Grigorova, L. I. Sokolova - 3rd edition - Kyiv, Medical University "Medicine", 2020 - 640 p.
2. Topical diagnosis of pathology of the nervous system. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.
3. Methods of examination of a neurological patient: teaching. manual / edited by L. I. Sokolova, T. I. Ilyash. - 2nd edition. - Kyiv: Medicine, 2020. - 143 p.
4. Emergency medicine. Emergency medical care: textbook / I.S. Zozulya, V.I. Bobrova, H.G. Roschyn and others / edited by I.S. Cuckoos - 3rd edition, trans. and additional - Kyiv. - VSV "Medicine", 2017. - 960 p.
5. Negrych T.I., Bozhenko N.L., Matvienko Yu.Sh. Ischemic stroke: secondary inpatient care: education. manual Lviv: LNMU named after Danylo Halytskyi, 2019. – 160 p.

Additional

1. Bozhenko M.I., Negrych T.I., Bozhenko N.L., Negrych N.O. Headache. Study guide.-K.: Medknyga Publishing House, 2019. - 48 p.
2. Davidson's Medicine: Principles and Practice: 23rd Edition: In 3 Volumes. Volume 1 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2020. - 258 p.
3. Davidson's Medicine: Principles and Practice: 23rd Edition: In 3 Volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 778 p.
4. Davidson's Medicine: Principles and Practice: 23rd Edition: In 3 Volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 642 p.

ISW No. 5

Topic: Diseases of the peripheral nervous system.

Actuality of theme :

Among the diseases of the nervous system, damage to its peripheral part occurs most often. Unlike the nerves of the central N.S. , peripheral nerves do not have strong bone protection and a blood-brain barrier. They are damaged over a considerable length and show increased sensitivity to the action of various exogenous and endogenous factors.

Specific goals:

- to know

- a) symptoms of damage to the lumbar sacral roots, sciatic and femoral nerves;
- b) symptoms of damage to the ulnar, radial and median nerves;
- c) etiology and clinical course of polyneuritis;
- d) paraclinical research, treatment and prevention of diseases of the peripheral nervous system.

be able:

investigate:

- a) brachial plexus functions;
- b) symptoms of sciatic and femoral nerve tension;
- c) symptoms of sciatic nerve damage;
- d) symptoms of femoral nerve damage;
- e) symptoms of damage to the ulnar, radial and median nerves;
- e) prescribe the necessary examinations and treatment for the thematic patients.

Questions for the lesson to be discussed:

- 1.. List the symptoms of tension of the sciatic and femoral nerves, know the method of their inspection.
2. What is the pathogenesis of tension symptoms?
3. What are upper and lower brachial plexitis?
4. Sanatorium-resort treatment: mechanisms of physical exercise, massage, balneo- and physiotherapy effects on the body.

New terms: symptom of Wasserman, Neri, Laseg, Matskevich, Brogard.

Questions or tests, diagnostic tests, role-playing games, etc. to determine the quality of students' mastery of the lesson topic:

Question

- A) The function of which fingers is affected by neuritis of the ulnar nerve?
- B) The function of which fingers is impaired with neuritis of the median nerve?

Answers

- A) 4 and 5 fingers of the bone.
- B) 1, 2 and partially third fingers.

Tests

- with a single selected answer (1 level)
- A. Duchenne-Erb upper plexitis shows the following symptoms:
- a) flexion-elbow reflex is lost.
 - b) the elbow flexion reflex increases.
 - c) there is an elbow extensor reflex
 - d) the elbow extensor reflex increases.
 - e) reflexes do not change. The answer is a)
- B. With neuritis of the femoral nerve, tension symptoms appear:
- a) Lasega
 - b) Neri
 - c) Brogard
 - d) Wasserman
 - d) Sokolyanskyi Answer - d)
- multiple choice (2 levels)
- A) Name the type of sensitivity disorder in plexitis.
- B) Name the type of sensitivity disorder in polyneuritis:
- a) root
 - b) conductive
 - c) plexal
 - d) mononeural
 - e) by type of stockings and gloves

Answers

- A. - c
- B. - b

When the peripheral nervous system is damaged, symptoms are observed:

- a) muscle hypertrophy
- b) hyperreflexia
- c) muscle hypotonia
- d) pathological reflexes
- e) hypotrophy

Answer: v.d.

Patient N., 24 years old, drank almost 300 ml in the evening. of vodka, fell asleep with his right hand at his side. Waking up in the morning, he noticed that his hand was not moving. Objectively: the hand in the elbow joint does not extend, the fingers of the bone do not extend, supination of the bone is not possible, the bone "hangs", the thumb cannot be opposed to others.

1. Name the diagnosis of the disease.
2. Prescribe treatment.

Answer:

1. Intoxication-traumatic neuritis of the right radial nerve.
2. Proserin, nicotinic acid, dibazole, vitamins B1, B12, furosemide, massage, HRT, myoton.

Literature :

Basic

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

1. Medical Books On-line Library (Neurology) – free download <http://medbookshelf.info/category/neurology/>
2. Clinical guidelines in neurology. (Order of the Ministry of Health of Ukraine No. 487 dated August 17, 2007) <https://zakon.rada.gov.ua/rada/show/v0487282-07#Text>
3. Ministry of Health of Ukraine <http://moz.gov.ua>
4. State Expert Center of the Ministry of Health of Ukraine www.dec.gov.ua/mtd/home/

ISW No. 6

Topic: Headache.

Relevance of the topic : Acquaintance with the problem of sleep disturbance is relevant due to the fact that it can be observed at any age and with any pathology. The acquirer must recall the physiology of sleep, its clinical phases and bioelectrical characteristics; study different types of sleep disorders, learn to determine adequate therapy according to the etiology of the disease. The ability to correctly establish a diagnosis and prescribe adequate therapy is essential for reducing the suffering of many patients.

Specific goals:

know:

1. The main types of headache.
2. Differential diagnosis of headache
3. Treatment of headache
4. Physiology of normal sleep
5. Phases of sleep
6. EEG characteristics of each of the phases
7. Clinical characteristics of each phase of sleep
8. What is insomnia
9. What is hypersomnia

be able:

1. Diagnose the main types of headache.
2. Conduct differential diagnosis of headache
3. Prescribe headache treatment
4. On the EEG, know the signs of the rapid phase of sleep.
5. On the EEG, find the slow phase of sleep
6. To be able to differentiate between insomnia and hypersomnia
7. Prescribe adequate therapy for sleep disorders

Tasks for independent work during preparation for the lesson

A list of the main terms, parameters, and characteristics that the student should learn when preparing for the lesson:

- Term: tension headache, post-puncture headache, hypertensive-fluidic headache, hypotensive-fluidic headache.

Theoretical questions for the lesson:

1. Classification of headache.
2. Clinical manifestations of the main forms of headache.

Practical works (tasks) that will be performed during the lesson:

1. Assign an examination to a patient for tension headache and migraine.
2. To carry out a differential diagnosis between migraine headache, tension headache and hypertensive cerebrospinal fluid headache.
3. Prescribe treatment to the patient for tension headache and migraine.

Topic content:

Classification of headache

Migraine.

Tension headache.

Cluster headache and chronic paroxysmal hemicrania.

Various forms of headache not associated with structural damage.

Headache associated with head injury.

Headache associated with vascular disorders.

Headache associated with nonvascular intracranial disorders.

Headache associated with the use of certain substances or refusal to take them.

Headache associated with infection.

Headache associated with metabolic disorders.

Headache or facial pain associated with pathology of the skull, neck, eyes, nose, sinuses, teeth, mouth, or other facial or cranial structures.

Cranial neuralgia, neuropathies and deafferentation pain.

Unclassified headache.

Two types are most common among all headaches: migraine - 38% and tension headache - 54%, as well as post-traumatic headache.

Migraine is a paroxysmal recurrent headache of a pulsating character, usually one-sided (hemicrania - pain in 1/2 of the head). It occurs in 2-6% of the population, mainly in women. Occurs at the age of 10 to 30 years.

In the pathogenesis of migraine, a hereditarily determined violation of vasomotor regulation of extra- and intracranial arteries is of leading importance. During an attack, 4 phases of vasomotor disturbances successively replace each other: spasm mainly of intracerebral and retinal vessels; dilatation of extracerebral arteries; edema of the vascular wall; reverse development of changes. In the first phase, an aura may occur, in the second - a headache. Disruption of the metabolism of serotonin and other biologically active substances (histamine, prostaglandins, tyramine, glutamate, etc.) plays an important role in the pathogenesis of migraine. Recently, the triggering factor of an attack is considered to be not biochemical, but neurophysiological changes.

According to the international classification, migraine is divided into 2 types: migraine without aura and migraine with aura. Precursors of migraine can be periodic syndromes of childhood: spasm-like pains in the stomach (abdominal migraine), paroxysmal dizziness ("vestibular migraine"), attacks of vomiting, tendency to pump, less often alternating paresis of the limbs.

Migraine without aura (simple migraine). Its main manifestation is a throbbing unilateral headache. More often, it does not occupy the entire half of the head, but, as a rule, the fronto-temporal or parietal-occipital region, less often it is bilateral, the side of the pain may alternate. The intensity of the pain is average or significant, at the end of the attack the pain is dull. During the attack, general hyperesthesia, intolerance to light, loud sounds develops. The patient wants to lie down in bed and not move, this relieves the pain, physical exertion intensifies it. In most patients, the attack is accompanied by nausea, often vomiting. The duration of attacks varies from 4 to 72 hours.

Migraine with aura. Aura is a focal neurological symptom that precedes a headache. Headache occurs immediately after the end of the aura or after a short light interval, less often - during the aura, especially a prolonged one. The most typical visual aura, manifested by a flickering scotoma, blurring of vision, a zigzag line in the homonymous fields of vision. It lasts 5-20 minutes, and then a headache attack occurs (ophthalmic, classic migraine). The second most frequent place is an aura in the form of paresthesias, which occur first in one finger of the hand, then pass to others, rise up the hand and spread to the face, tongue (this sometimes causes dysphasia, even with left-sided paresthesias). Rare types of aura include hemiparesis, motor aphasia, and ophthalmoparesis. Migraine with aura in the form of neurological disorders was previously called associated. In rare cases, usually in elderly men, the aura is not followed by a headache (dissociated migraine, "migraine without migraine"). Aura is caused by local ischemia. Unlike transient ischemic attacks, which are mistakenly diagnosed in these cases, the main and intracranial arteries are intact and the prognosis in most patients is favorable.

Of great interest is "familial hemiplegic migraine" associated with the pathology of the gene mapped on the 19th chromosome. It is characterized by a prolonged aura in the form of hemiparesis, paresthesia, and speech disorders. The duration of the aura varies from 2-3 hours to 3 days, and the headache develops during the aura, as in other cases with a prolonged aura.

Complications of migraine. These include migraine status and migraine stroke.

Migrainous status. Sometimes migraine attacks follow one another without a break, accompanied by repeated vomiting and dehydration. If the attack lasts more than 72 hours, migraine status is diagnosed. It requires hospitalization and emergency treatment, including corticosteroids.

Migrainous stroke (brain infarction). Recently, it has been proven that in rare cases, a migraine attack ends with the development of a brain infarction, which leads to the occurrence of a neurological deficit that persists for more than 3 days and is not always reversible. Brain infarcts in migraine are usually localized in the back of the brain.

Chronic daily headache. In some patients with migraine, a constant headache appears against the background of typical migraine pains, which differs in character from migraine (pain is not pulsating, diffuse, less intense, without nausea and vomiting). Its cause was considered to be a combination of migraine with another type of headache, most often psychogenic. Recently, it has been proven that the basis may lie in the abuse of medicines, such a headache is called an abuse headache.

The diagnostic criteria for migraine are given in the International Classification of Headache.

Diagnostic criteria for migraine without aura.

The presence of at least 5 attacks lasting from 4 to 72 hours (without treatment).

Cephalgia has at least 2 of the indicated signs: one-sided, pulsating, moderate or strong, aggravated by physical exertion.

Cephalgia is accompanied by at least one of the listed symptoms: nausea and/or vomiting, photo- and phonophobia.

It is also important to alternate the side of cephalgia, since a one-sided headache for a long time requires the exclusion of other diseases.

Another classification criterion that applies to all other types of primary headache is compliance with one of the following three conditions: either the anamnesis, somatic and neurological examination exclude the presence of diseases in which cephalgia is symptomatic, or these diseases are expected, but excluded by detailed examinations, or the patient has these diseases, but migraine attacks are independent and not related to them in terms of time of occurrence.

Diagnostic criteria for migraine with aura.

The presence of at least 2 attacks, characterized by at least three of the listed signs: there is one or more aura symptoms, completely reversible, which indicate focal cerebral dysfunctions of the cortex and / or trunk;

none of the aura symptoms lasts more than 60 minutes (but if there are more symptoms, a proportionately longer duration is allowed);

headache follows the aura with a light interval of less than 60 minutes, but may begin before the aura or simultaneously with it;

at least one of the aura symptoms develops gradually in more than 4 minutes or 2 or more symptoms develop simultaneously;

have one of the conditions listed above (migraine without aura).

Diagnostic criteria for abuse headaches (caused by chronic administration or drug overdose):

headache occurs after taking the drug daily for 3 months or more;

it is possible to accurately establish the minimum dose that brings relief;

headache is chronic (15 days or more per month) and disappears within 1 month after stopping the drug.

Ergotamine abuse tooth pain occurs at a dose of 2 mg per day orally and 1 mg rectally.

Analgesic overuse pain occurs when taking 50 aspirin per month (or an equivalent dose of another non-narcotic analgesic), or when taking combined analgesics (with caffeine, barbiturates, tranquilizers, etc.) in the amount of 100 tablets or more per month, or when taking narcotic analgesics.

Tension headache is one of the main forms of primary headache. It is manifested by cephalic episodes (several minutes - several days). The pain, as a rule, is bilateral, compressive in nature, of moderate or light intensity, does not increase with ordinary physical exertion. Sometimes photo and phonophobia is possible.

The term "tension headache" also corresponds to: GBN, muscle tension headache, stress headache, psychomyogenic headache, psychogenic headache, idiopathic headache.

Classification of tension headache

There are several types of tension headache, some of which in turn have subtypes:

episodic (occurs no more than 15 days within 1 month)

chronic (occurs more than 15 days a month)

In addition, both forms of tension headache are divided into "tension headache" and "tension headache without pericranial muscle tension."

Etiology and pathogenesis of tension headache

In modern medicine, tension headache is considered exclusively as a neurobiological disease. Presumably, not only central, but also peripheral nociceptive mechanisms are involved in the etiology of tension headache. The leading role in the pathogenesis of tension headache is played by increased sensitivity of pain structures, as well as insufficient function of the descending inhibitory pathways of the brain.

The main provoking factor of a tension headache attack is emotional stress. It has been proven that switching attention or positive emotions can reduce the intensity of a headache up to its complete disappearance. However, after a while the headache returns. Another provoking factor is the so-called the muscular factor, i.e. being in tension for a long time without changing the posture (forced position of the head and neck when working at a desk and driving a vehicle).

There are also factors that form a chronic pain pattern. One of these factors is depression. In addition to traumatic life situations, the development of depression is also facilitated by personality characteristics, you or other behavioral characteristics. Another factor of chronicity is drug abuse (abuse of symptomatic painkillers). It has been proven that in case of consumption of a large number of painkillers, chronic tension headache is formed twice as often. To treat drug abuse, it is necessary to cancel the drug that caused this complication as soon as possible.

Clinical picture of tension headache

Typically, patients describe a tension-type headache as a mild to moderate, non-throbbing, bilateral squeezing headache that squeezes the head in a "hoop." The intensity of such a headache does not depend on physical exertion, and is very rarely accompanied by nausea. It appears, as a rule, some time after waking up and continues throughout the day.

Diagnosis of tension headache

There are several criteria for diagnosing a tension headache:

Duration of headache from 30 minutes to 7 days

The presence of at least two of the following signs:

pain intensity does not depend on physical exertion;

bilateral headache;

mild or moderate intensity of pain;

the nature of the pain is not pulsating, but squeezing (squeezing the head with a "hoop");

absence of nausea and vomiting;

headache is not a symptom of another disorder of the body's functions;

increasing pain against the background of strong emotional stress;

pain relief against the background of positive emotions and psychological relaxation.

Since, in addition to the above signs indicating a tension headache, patients often complain of a feeling of discomfort and even burning in the back of the head, back of the neck and forearm ("coat hanger" syndrome), when examining the patient, it is necessary to examine the

cranial muscles . It has been proven that the most sensitive diagnostic method for detecting pericranial muscle dysfunction in patients with GBN is palpation. This dysfunction is revealed when pressing in the area of the frontal, masticatory, sternoclavicular-mammoid and trapezoidal muscles, as well as when palpating with rotational movements of the second and third fingers in the area of the same muscles. The presence of pericranial muscle dysfunction is taken into account in the future when choosing a treatment strategy. Hypersensitivity of the pericranial muscles during palpation means the presence of "chronic (or episodic) tension headache with tension of the pericranial muscles."

In addition, in addition to the signs listed above, manifestations of anxiety and depressive disorders in the form of low background mood, apathy or, on the contrary, increased aggressiveness and irritability often coexist. The degree of such disorders in tension headache can vary from mild to severe.

Differential diagnosis

To rule out an organic cause of tension headache (tumors, inflammatory processes, impaired blood circulation of the brain), a full set of neurological examinations is performed: EEG of the brain, ECHO-EG, if there are indications - CT scan or MRI of the brain.

The main difference between episodic tension-type headache and chronic tension-type headache is the number of days (days/month) in which this headache occurs.

Treatment of tension headache

In the treatment of tension headaches, neurologists use a complex approach. Firstly, it is necessary to normalize the emotional state of the patient, and secondly, to eliminate the dysfunction of the pericranial muscles. In addition, it is necessary to take measures to prevent drug abuse. The result of such treatment is the reduction of pain and muscle-tonic syndrome, prevention of transformation of episodic tension headache into chronic GBN.

The following groups of drugs are used as drug treatment for tension headaches:

antidepressants (selective serotonin reuptake inhibitors, selective serotonin and norepinephrine reuptake inhibitors);

muscle relaxants (tolperisone, tizanidine), NSAIDs (diclofenac, naproxen, ketoprofen);

drugs for the preventive treatment of migraine (in case of combination of tension headache with migraine)

Acupuncture, manual therapy, massage are used as non-medicinal methods of treatment for tension headaches.

Cluster headache. Synonyms: cluster headache, migraine neuralgia Harris, histamine cephalgia Horton, etc. This type of headache unites several previously distinct forms: migraine neuralgia, ciliary neuralgia, etc.

In the International Classification, 3 forms of cluster pain are distinguished depending on the frequency of their manifestation: with an undefined periodicity, episodic and chronic.

Together with cluster headaches, chronic paroxysmal hemicrania and cluster-like headaches are considered.

Cluster cephalgias are rare, men are affected 5-6 times more often, the onset of the disease is in 20-40 years. Etiopathogenesis is not known, it is assumed that vascular mechanisms are the basis of pain.

Episodic cluster headache.

This disease is characterized by attacks of very strong one-sided headache, which repeats daily (1-2, rarely 5-8 times) for several weeks or even months. After that comes a long remission (months and years). The intensity of pain and the duration of the attacks change during one cluster period (a series of attacks) from lighter and shorter to stronger and longer, and then the attacks

become light and disappear. The pain occurs suddenly without warning, is localized in the eye area, periorbital zone and in the temple, radiation to the ear, neck, and hand is possible. The nature of the pain is burning, and the force is so great that it wakes up sleeping patients. The duration of the attack (without treatment) is 15-180 minutes. At the beginning of the series, attacks more often develop at night, in the morning at the same time ("alarm clock" headache), but they can also occur during the day. Psychomotor excitement is noted during the attack. Attacks are accompanied by pronounced vegetative disorders, redness and lacrimation from one eye, nasal congestion, rhinorrhea, sweating in the face or forehead, ptosis, miosis. According to the modern classification, the diagnostic criteria for cluster episodic headache are the presence of at least 5 attacks of the nature and location described above and at least one of the listed vegetative symptoms, as well as a serial course of daily attacks.

Chronic cluster headaches occur in two variants: without remission from the moment of their appearance or transforming from an episodic headache. Attacks are shorter and less severe, but are characterized by high frequency (up to 20-30 per day) and lack of remission.

Chronic paroxysmal hemicrania is a rare type of paroxysmal one-sided headache, localized in the eye-frontal-temporal area (above and below the eye, with migraine - above the eye), of great intensity, drilling. Attacks last 10-30 minutes, repeat 10-20 times a day and are accompanied by vegetative symptoms from the eye and nose. They are distinguished from chronic cluster cephalgia by the predominance of women and the "dramatic" effect of indomethacin, which quickly relieves headache ("indomethacin" headache).

Various headaches not associated with structural damage. This group includes rare forms of non-organic headache. These include idiopathic shooting pain, which manifests itself as a sharp lightning (fraction of a second) severe pain in certain parts of the head, its pathogenesis is unclear.

Headache associated with hypothermia can be caused by any cold stimulus, even in the pharynx.

The headache associated with coughing and physical exertion (effort) is throbbing in nature, but is never accompanied by any other symptoms. By nature, this is a benign pain, but in some patients, the appearance of pain during tension can be caused by structural processes that occur with hypertensive syndrome (brain tumors, etc.). Therefore, headaches with coughing and physical exertion require a detailed examination, including a CT scan of the brain.

Diagnosis of headache. At the first stage of diagnosis, it is necessary to determine whether the headache is related to a structural lesion (organic brain disease). It is most important to rule out tumors, acute disorders of cerebral circulation, especially subarachnoid hemorrhage, hematomas, complications of acute TBI (epi- and subdural hematomas, etc.), inflammatory diseases of the brain, its membranes, paranasal sinuses, eyes, glaucoma, etc.

In the International Classification, "danger signals" in headache that cause suspicion of structural damage are highlighted:

Occurrence of headache for the first time after 50 years.

Sudden severe ("thunderous") headache (subarachnoid hemorrhage, first migraine attack, temporal arteritis).

"Tide" to the head (intracranial hemorrhage).

Increase in headache during coughing, straining, physical exertion (intracranial hypertension, migraine).

Increase in headache over time: over hours-days (meningitis, encephalitis), days-weeks (tumor, temporal arteritis).

Headache is the cause of night awakenings (tumor, attack of cluster headache, migraine).

In the morning, nausea, vomiting, hiccups, dizziness (tumor).

An indication of a structural headache is also the presence of focal neurological symptoms, changes in additional research methods, ineffectiveness of therapeutic effects, non-compliance of the headache with the criteria of the International Classification of Headaches.

In favor of a primary headache (migraine, tension headache, cluster and rare variants) is evidenced by the compliance of the headache with the diagnostic criteria, normal well-being and stable body weight, duration of the pain for more than two years and the absence of deviations from the norm during the examination (this sign is the most significant).

Scheme of examination of a patient with a headache:

- According to the indications, visualization of vessels by non-invasive methods (extra- and transcranial dopplerography) or with intra-arterial injection of a contrast agent (angiography) can be performed to clarify the diagnosis.

These studies more accurately than CT allow to establish the presence and localization of an aneurysm, occlusion of an artery, the state of the vascular bed, the displacement of vessels in the case of bulky processes, to detect vascular tumors, the peculiarities of its blood supply or avascular areas with a violation of the topography of vessels (cyst, abscess, some tumors, hematomas, infarction with perifocal edema).

Other special methods - evoked potentials, nystagmography, etc. - are used to finally specify the nature of the structural process.

At the second stage of diagnosis, after excluding the symptomatic nature of headaches, the type of primary headache is specified. Here, compliance of the headache with the diagnostic criteria of the International Headache Society is crucial. The given diagnostic criteria are very effective in distinguishing the type of headache. Next, it is necessary to assess the frequency of attacks, which is important for the choice of treatment tactics, for example, abortive (in case of rare migraine attacks) or preventive (in case of frequent attacks) treatment.

To work out the tactics of therapy, the choice of drugs and their dosages in patients with chronic pain, it is recommended to keep a diary, which reflects the type of headache, its intensity, duration and frequency of attacks (similar to the diary of a patient with epilepsy). The diary also displays previous and accompanying headache symptoms, provoking factors, response to stress, which drug and which dose stopped the attack. After clarifying the type of headache and its features, the patient is given a calendar in which the dates of the attacks are noted and their frequency is taken into account.

Sleep is a functional state of the brain of the entire human and animal body, which differs from non-sleep by specific qualitative features of the activity of the central nervous system and the somatic sphere and is characterized by the inhibition of the active interaction of the organism with the environment and the incomplete cessation (in humans) of conscious mental activity.

In newborn children, sleep and non-sleep periods are arrhythmic and occur chaotically, while the strongest stimulus that can disrupt sleep is hunger arousal. The child sleeps almost all the time, but his sleep is restless, shallow. The total duration of a newborn's sleep is 16-20 hours a day. At the end of the first month of a child's life, under the influence of natural environmental stimuli and based on the daily need for sleep, a daily rhythm of sleep and non-sleep is formed.

In order to create a rhythmic alternation of the latter in the child during the day, it is necessary to create special conditions for fast falling asleep and sound sleep during the hours provided for sleep and an active state during the hours provided for entertainment.

Sleep is a complex and heterogeneous state. There are 2 phases of sleep - slow (FPS) and fast (FSHS); the latter is sometimes called a paradoxical dream.

FPS is divided into 4 stages, which differ in EEG characteristics and awakening thresholds and objective indicators of deep sleep (1-nap; 2-mid-depth sleep; 3-4 stages — the deepest delta sleep)

The reticular formation of the brain stem, the mesencephalic hypothalamic and preoptic areas of the hypothalamus are important in the regulation of sleep.

Human sleep is characterized by the cyclicity of one cycle lasting 1.5-2 hours. During the night, 3-5 cycles are observed.

Delta sleep is the first 2 cycles, FSHS is maximal during 3-4 cycles (in the morning hours). On average, FPS is 75-80% of the duration of all sleep; FSS takes 20-25%.

During sleep, depending on the phase, the activity of the motor system, the vegetative-visceral state of the body, the activity of the endocrine system, and mental activity change significantly.

Changes in the organization of sleep are influenced by a variety of factors: biological, social, mental, physical, time zone changes, the Earth's magnetic state, and others.

Sleep disorders are divided into 2 groups: 1- sleep disorders or hypersomnia; 2-sleep disturbance or insomnia.

At the heart of the sleep disorder of the first group are cerebral disorders, which are accompanied by a deficiency of activating systems of the reticular formation of the brain stem.

Sleep disorders of the second group can be observed due to excessive activation of the system of the reticular formation, which increases, as well as a violation of the activity of the synchronizing systems of the brain (bulbar centers of the brain stem, nuclei of the Varolii bridge, preoptic zone of the hypothalamus, visual hills that create the FPS, as well as the limbic-reticular complex, which ensures a normal change in sleep and non-sleep processes.

According to the classification of M.M. Yakhno and O.M. Wayne (1979) hypersomnias are divided into the following subgroups:

Narcolepsy — imperative bouts of daytime sleepiness, accompanied by catalepsy (short-term muscle relaxation that leads to the patient falling), hypnagogic hallucinations, sleep disturbances with vivid, frightening dreams.

Idiopathic hypersomnia syndrome is characterized by daytime sleepiness, prolongation of night sleep, "night drunkenness" syndrome (feeling of tiredness, brokenness, light confusion of consciousness after a nocturnal state). This disease is observed in persons with a violation of the emotional sphere and moderate hypothalamic insufficiency.

And idiopathic hypersomnia with breathing disorders during sleep — episodic cessation of breathing, or apnea. They occur in functional and organic pathology of the brain, which leads to a violation of the central regulation of breathing.

Pickwick's syndrome — is accompanied by night apnea, intense snoring, cardiopulmonary failure, obesity, which leads to limitation of the mobility of the diaphragm and obstruction of the upper respiratory tract. Night apnea may be associated with neoplasms of the nasopharynx (adenomas, polyps). Daytime sleepiness is compensatory in nature.

Kleine-Levin syndrome is episodes of compulsive sleepiness lasting from several hours to several days. It is observed exclusively in young men of pubertal and post-pubertal age and is combined with psychopathological disorders, bulimia and polydipsia during the attack.

Syndrome of periodic pseudohypersomnia of hysterical genesis ("hysterical hibernation") - characterized by paroxysmal dream-like states, lasting from several hours to many days. As a rule, it occurs in connection with a psychotraumatic situation; it is more often observed in women who have other signs of hysterical disorders. At the same time, the EEG shows a pattern of autonomic activation (tachycardia, increased blood pressure, hyperthermia, etc.) and no sleep.

The syndrome of periodic pseudohypersomnia of epileptic genesis is rare and is characterized by paroxysmal sleep-related states. EEG shows clear epileptic activity in the form of "peak waves" following one another. This syndrome is a variant of convulsive forms of status epilepticus and can occur in the final phase of generalized epileptic seizures, during migraine paroxysms, hypoglycemic states, liver failure, etc.

Hypersomnia syndromes of a permanent nature - occur with neuroinfections, tumors, intoxications, when the hypothalamic-mesencephalic zone is involved in the pathological process, and the dynamics are determined by the etiology of the disease

Insomnia (insomnia)

Partial insomnia is the most common form of sleep disorder. It is manifested by subjective dissatisfaction with night sleep, its depth and duration. At the same time, difficulty falling asleep, waking up in the middle of the night, and early morning awakening are observed. This leads to neurotic and mental disorders, as well as various neurological and visceral disorders.

Complete insomnia is rare. Occurs with reactive or endogenous mental illnesses.

Pseudoinsonmias - patients complain of reduced or no sleep. At the same time, the main EEG characteristics of sleep are preserved. Significant emotional disturbances are noted.

Idiopathic hypersomnia is rare. At the same time, practically healthy people sleep several hours a day and at the same time do not complain about the lack of sleep at night.

Treatment of the above disorders is based on etiological, pathogenetic and symptomatic complex approaches to the disease. The prognosis depends on the cause of the sleep disturbance. Recently, Ivadal, Imovan, and Donormil have been used in 1 cap each. In 15 min. Before going to sleep, which ensure normal physiological sleep.

Sleep disturbance to one degree or another is noted in the majority of elderly people, about 25% of whom regularly take sleeping pills. Insomnia is a significant factor that helps to assess the quality of life of an elderly person.

Disruption of sleep and the process of falling asleep in old age is a consequence of the interaction of medical, psychogenic and psychosocial factors, which are combined with dysfunction of brain activity. Primary sleep disorders. The causes of primary sleep disturbance in an elderly person are sudden muscle contraction, anxiety, night snoring, and respiratory arrest. As a rule, apnea syndrome is mainly observed in obese men after the age of 50, and often in old age in women.

Restless legs syndrome is accompanied by a feeling of some discomfort and tingling in the legs, as a result of which there is an irresistible desire to move them. Such sensations are called dysesthesia and are manifested in a state of rest, when a person is resting and relaxed. The appearance of restless legs syndrome is observed at a young age, and it only worsens over the years, especially in people suffering from kidney failure, uremia, neurosis, arthritis, and during pregnancy. In old age, it is necessary to be more attentive to yourself, as the signs of this disease may indicate serious health problems. Secondary causes of disturbed sleep. Secondary disturbance of sleep and the process of falling asleep in old age is caused by neurological, somatic and mental diseases. Most often, such heart pathology as coronary disease, hypertension, arterial hypertension and angina pectoris leads to insomnia. Therapeutic correction of heart failure allows you to solve sleep problems. Attacks of bronchial asthma, obstructive pulmonary disease, various pain syndromes, polyosteoarthrosis, diabetes, thyrotoxicosis, prostate hyperplasia, anemia, and kidney failure are often sleep disruptors.

With somatic pathology, the patient in old age has superficial sleep with frequent awakenings, so the treatment is aimed at eliminating the underlying disease, and not at the use of sleeping pills.

Pharmacological causes of sleep disturbance:

- psychotropic drugs;
- hypotensive drugs;
- antiarrhythmic drugs;
- broncholytic and hormonal drugs;
- antibiotics;
- cardiac glycosides;
- antiparkinsonian and hypolipidemic drugs;
- anticancer agents;
- antitussive drugs.

Also, the use of some types of eye drops, diuretics, and antidiabetic drugs can cause disruption of sleep and the process of falling asleep.

Diagnosis of sleep disorders. In order to identify the main causes of insomnia, such parameters as the time of sleep onset, its duration, the number of night awakenings, the presence

and characteristics of dreams, and the quality of sleep and awakening are also evaluated. When making a diagnosis and correctly determining the causes of sleep disturbances, a strategy for possible correction of this phenomenon is determined, taking into account the individual characteristics of the state of health of each individual patient.

Treatment of insomnia.

Patients who suffer from insomnia are advised to go to bed only when there is a physical need for it, to get up at the same time in the morning, regardless of the duration of sleep, and to cancel daytime naps. It is also important to limit empty time in bed, avoid exciting experiences before going to bed, go to bed at the same time, exclude tonic drinks in the evening and do not overeat.

Drug therapy of insomnia.

Drug treatment of insomnia is indicated only for long-term sleep disturbances, while low doses of pharmacokinetic drugs are prescribed, the intake of which is short-term. After the post-therapeutic monitoring of the patient, as a rule, there is a gradual withdrawal of hypnotic drugs.

When prescribing tranquilizers to the elderly, their recommended dose is halved, and the course of therapy is prescribed to be minimal with timely breaks and a change of drugs. First-generation hypnotics are usually avoided in old age to avoid the risk of nocturnal respiratory depression. Treatment of secondary sleep disorders is carried out with the help of second-generation hypnotics, which are short, medium and long-acting. Short-term drugs are prescribed to facilitate the process of falling asleep, drugs of medium duration of action are used to eliminate frequent awakenings and deeper sleep, and stronger drugs are needed in order to improve the quality of sleep and eliminate early awakenings.

Side effects of insomnia treatment:

- decrease in muscle tone;
- trace drowsiness;
- neuromuscular weakness;
- respiratory depression;
- constipation;
- decrease in heart rate;
- arterial hypotension;
- disequilibrium;
- decreased concentration of attention;
- suppression of consciousness;
- senile psychoses.

Drug dependence in the elderly increases as a result of excessive alcohol consumption, but the use of drugs is quite safe.

Prevention of insomnia.

To prevent sleep disturbances, it is recommended to exclude the use of tea, coffee, chocolate and tonic drinks, reduce physical exertion, avoid overeating and excessive impressions before bedtime.

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