MINISTRY OF HEALTH OF UKRAINE

ODESA NATIONAL MEDICAL UNIVERSITY

Faculty of Medicine №2

Department of Neurology and Neurosurgery

APPROVED BY
Vice-Rector for Scientific and Educational Work
_____ Eduard BURIACHKIVSKYI
«_ _» _____ 2024

TEACHING MATERIAL
FOR ISW CLASSES ON THE ACADEMIC SUBJECT

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

Approved by:
Meeting of the Department of Neurology and Neurosurgery
Odesa National Medical University
Protocol No. 1 dated “26” 08 2024

Head of the Department  Anatoliy SON

Developers:
Anatoliy Son, PhD, DSci, Professor, Head of the Department
Alexander Stoyanov, PhD, DSci, Professor
Yuriy Goranskyi, PhD, Associate Professor
Gana Perkova, PhD, Associate Professor
Olena Lebed, PhD, Associate Professor
Vasyl Dobrovolskyi, PhD, Associate Professor
Yuliia Solodovnikova, PhD, Associate Professor
Eliza Babych, Assistant Professor
Iryna Hnatiuk, Assistant Professor
Kateryna Sarazhyna, Assistant Professor
Ksenia Yarovaya, Assistant Professor
Ihor Serbin, Assistant Professor

INDEPENDENT STUDENTS WORK

ISW No. 1

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

Relevance of the topic: The study of neurology took place in the universities of the Middle Ages and in the Renaissance. Modern directions of the development of neurology: differentiation of neurological science (creation of separate centers and scientific divisions for the study of cerebrovascular, demyelinating diseases, epilepsy, neuromuscular pathology, etc.) and integration with other sciences (vertebroneurology, neurosurgery).

Specific goals:

As a result of independent work on 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

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. 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. O. Ya. Kozhevnikov was an outstanding research scientist. This talented clinician had a deep knowledge of the morphology, histology and physiology of the nervous system. He is the author of original works in which many diseases of the nervous system are described. He has a classic description of special seizures, known in world literature as "Kozhevnikov epilepsy." VK Roth (1848-1916) was a talented neuropathologist who studied progressive muscle diseases. His monograph "On muscular tuberculosis" is a generalization of these studies. VK Roth systematized the forms of muscle atrophy known at that time. He was one of the first to study diseases of the nervous system of children. VK Roth was not only a talented scientist, but also a progressive public figure. VK Roth emphasized the need to prevent nervous diseases in children by improving education. The organizational talent helped him realize the dream of his teacher O. Ya. Kozhevnikov. He achieved the construction of the building of the Neurological Museum named after O. Ya. Kozhevnikov. Laboratories for the study of morphology, physiology, psychophysiology, etc. functioned in this museum. The Neurological Institute was established on the basis of the museum. One of the founders of child psychoneurology is G. I. Rossolimo (1860-1928). He was a talented clinician with remarkable abilities as a doctor-pedagogue. He has works on children's neuropathology, psychoneurology, and medical psychology, in which he drew the attention of advanced Russian intelligentsia, primarily doctors and teachers, to the issues of protecting children's psyche, preventing diseases of the nervous system in children. Such works include "Fear and Education", "Bad Habits and the Fight Against Them", "On the Question of Mental Disasters in Youth", "On Abnormal Elements in a Child's Character", "Art and Sick Nerves" and others. G. I. Rossolimo took an active part in the work of special congresses at which issues of education and training of blind, deaf-mute and mentally retarded children were discussed. In 1911, G. I. Rossolimo together with a group of his students organized the Institute of Child Psychology and Neurology, where, for the first time in our country, clinical and pedagogical conferences were held with the participation of doctors and teachers. In 1923, a textbook on nervous diseases was published under the editorship of G. I. Rossolimo. The famous neurologist V. O. Muratov (1865-1916) made many new contributions to the study of nervous and mental diseases in children. He studied children's cerebral palsy in detail and identified separate clinical forms of this disease. In addition, he studied the morphology of the brain.

The history of the establishment and development of the Odessa School of Neurologists begins on October 11, 1905, when the Department of Nervous Diseases was opened at the Medical Faculty of Novorossiysk University. At the origin of the development of this school stood scientists

known not only in Russia, but also outside its borders: M.M. Popov, O.E. Yanishevskyi, V.M. Obraztsov, who came from schools in St. Petersburg and Kazan. Their followers put a lot of effort into the development of the Odessa School of Neuropathologists, Psychiatrists and Neurosurgeons. One of the founders of this school was Academician Hryhoriy Ivanovich Markelov, a world-renowned neurologist. He raised a brilliant galaxy of outstanding scientists, neuropathologists, psychiatrists and representatives of related specialties, who successfully continued his work.

Professor Mykola Mykhailovych Popov (1854-1939) became the first head of the Department of Nervous and Mental Diseases. He headed the department until 1920. M.M. Popov graduated from the Medical and Surgical Academy in St. Petersburg, underwent advanced training in nervous and mental diseases in Leipzig and Paris, worked as a professor of the Department of Nervous and Mental Diseases at Warsaw and Kazan Universities. In 1909-1912, he was dean of the medical faculty of Novorossiysk University. In 1920, together with his student Professor O.E. Yanyshevskyi emigrated to Bulgaria, where in 1922, having withstood competition with German specialists, he created the Department of Nervous Diseases and Psychiatry at Sofia University.

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 Yanyshevskyi (1873-1936) - professor of the Department of Nervous and Mental Diseases (1916-1920). He graduated from the medical faculty of Kazan University. 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. He graduated with honors from the medical faculty of Kazan University. In 1904, he worked at the Department of Nervous and Mental Diseases of Novorossiysk University. 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 editorial office of "Neurological Herald" (Kazan). 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 of the Academy of Sciences of the Ukrainian SSR, honored scientist of the Ukrainian SSR, outstanding neuropathologist-vegetologist. He graduated with honors from the medical faculty of Novorossiysk University in 1906. All his scientific activities are 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. Khersonskyi, M.O. Gornyka, O.S. Lyvshina, I.I. Dobrovolskyi, 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. Vishnevska 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. Puchkovska (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 1913, he passed the exam at Novorossiysk University and received a doctor's diploma with honors.

Since 1917, L.L. Papadato worked as an assistant, resident and professor of the Department of Nervous Diseases of the Medical Faculty of Novorossiysk University. 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 Boris Ivanovich Sharapov (1897-1969) headed the department of nervous diseases from 1953 to 1956. He graduated from the medical faculty of Moscow University. At the Stalingrad Medical Institute, he headed the Department of Nervous Diseases (since 1939). Then, in 1944, he was appointed head of the clinic for nervous diseases of the Sochi Institute of Balneology. Then he moved to Moldova and from 1945 he headed the Department of Nervous Diseases of the Chisinau Medical Institute and at the same time the deputy director for educational and scientific issues.

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. Honored Scientist of the Moldavian SSR. 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. H.G. In 1923, Sokolyansky graduated from the medical faculty of the university in Rostov-on-Don. During the war (1943-1944), he headed the Department of Nervous Diseases of the Samarkand Medical Institute, and from 1944 to 1956 - the similar department of the Yaroslavl Medical Institute.

Just like B.I. Sharapov, H.G. Sokolyanskyi is a student of famous St. Petersburg 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, which was led by O.I., a student of the Academy of Medical Sciences of the USSR. Arutyunova, 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 chairman of the Odesa Scientific Society of Neurosurgeons (since 1994, the Association of Neurosurgeons of the Odesa Region), a member of the board of the All-Union Society of Neurosurgeons, a member of the editorial board of the magazines "Questions of Neurosurgery named after N.N. Burdenko" and "Ukrainian Neurosurgery Journal", a member of the Problem Commission of the Ministry of Health of the USSR on neurotraumatology.

Professor Yurii 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. Mirtovskyi, 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 Awarded the Great Commemorative, Gold, Silver and Bronze Medals of the VDNG of the USSR, Silver Medal of the VDNG of the USSR. 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.

Since 2014, the department has been named "Department of Neurology", its head was Tetyana Mykolaivna Muratova, Director of the Center for Reconstructive and Restorative Medicine (University Clinic) of ONMedU.

Modern directions of development of neurology:

- 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).

Recommended Books:

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., 2015. – 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: training. 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.rada.gov.ua/rada/show/v0487282-07#Text

ISW No. 2

Topic: Independent curation of patients with compilation of medical history.

Relevance of the topic: Curation of patients with independent diagnosis and appointment of treatment requires the use of the entire arsenal of knowledge and skills of students and must be carried out according to a strict scheme to prevent the occurrence of errors that can significantly harm both the patient and the doctor.

Specific goals:

to know: The scheme of writing a medical history

be able to: explore:

1. Anamnesis
2. Function of the nervous system
3. Formulate a diagnosis and carry out differential diagnosis

Lesson content.

Story outline

DISEASE HISTORY

Patient (name, surname):_____

Clinical diagnosis (neurological): _____

Faculty: _____

Course: _____

Group:_____

Supervisor (student name, surname): _____

Observation time:_____

Supervisor (teacher's name, surname):_____

II. Passport data: last name, first name, last name, age, gender, profession, home address, clinical diagnosis of the patient.

III. Patient complaints: mainly only those neurological complaints, due to which the patient was hospitalized.

IV. Medical history. The onset of the disease, the cause of the disease (infections, injuries, intoxication, tuberculosis, venereal diseases, cooling, psychophysical overstrains, rheumatism, hypertension, diabetes, and others). Development of symptoms of the disease, features of the course, influence of previous therapy, dynamic laboratory and paraclinical data (ECG, EEG, etc.).

IV. History of life. In the history (childhood infections, influenza, tonsillitis, tuberculosis, venereal diseases, craniocerebral and spinal cord injuries, operations). Inherited history, hypertension, diabetes. For women - menstrual cycle (regularity, pain, duration of menopause), pregnancy, childbirth, menopause. Marital status. Harmful habits (smoking, alcoholism, drug use, etc.).

V. General condition. Body temperature. Position in bed. Face expression. Constitutional features. Subcutaneous fat layer. Skin. Lymph nodes. A hundred joints. Pulse characteristics. Blood pressure. The limits of the heart. Heart activity and tones. Respiratory organs. The Digestive System. Genitourinary system.

VII. Neurological status.

I pair - olfactory nerve (smell of each nostril)

II pair - optic nerve. Visual acuity is checked in accordance with the table, the field of vision is checked by the perimeter or a control method, color perception occurs in accordance with the Rabkin table.

III pair - oculomotor, IV block and VI pair - abductor nerve are checked simultaneously. The position of the eyes, the size, movement of the eyeballs, and the size of the pupils and their uniformity, the quality of the direct and cooperative reaction of the pupils to light (live, lethargic, absent), the presence of the Argyle-Robertson symptom.

V pair - trigeminal nerve (act of chewing, tone of masticatory muscles, corneal and conjunctival reflexes, sensitivity on the face, soreness at the exit points of the trigeminal nerve).

VII para-facial nerve (functions of mimic muscles are checked; taste sensitivity on the front 2/3 of the tongue, hearing, lacrimation).

VIII para-auditory nerve (whisper voice is checked from a distance of 5-6 meters for each ear separately, Rinne and Weber test)

IX pair - glossopharyngeal and X pair - vagus nerves.

Speech sounds, the presence of swallowing and reflexes from the soft palate, the act of swallowing, the taste on the back third of the tongue, the movement of the soft palate during phonation, the position of the tongue and soft palate, the rhythm of respiratory movements and heart activity

XI pair - additional nerve (head position, level of shoulder elevation, head rotation, tone and appearance of m. sternocleidomastoideus and trapezius, fibrillation and fasciculation in them).

XII pair - hypoglossal nerve (tongue mobility, volume of tongue muscles, fibrillation, deviation) deviation of the tongue during its movement. Language articulation.

Motor functions.

Gait (spastic, ataxic fell, etc.) of patients. The volume and speed of active movements in all joints. Muscle strength (dynamometry and patient resistance according to a 6-point scale) in all muscle groups. Muscle volume (atrophy is manifested by studying and measuring the volume of the limbs with a tape). The volume of passive movements and muscle tone (hypotonia, hypertension, muscle dystonia). The phenomena of "cogwheel" and "folded knife". Presence of hyperkinesis and their type (chorea, athetosis, myoclonus, tremor, tics, and others).

Coordination of movements (finger-nose and knee-heel test, intentional tremor during movements, adiadochokinesis, Stewart-Holmes test, Babinsky test, Romberg's pose, hyper and dysmetria, asynergia).

Sensory functions.

Palpation of nerve exit points and nerve trunks. Symptoms of nerve and root tension (Neri, Laseg, Bekhterev, Wasserman, Matskevich, etc.). Zones of hypoesthesia (hypo- and anesthesia). Deep sensitivity: joint-muscular and vibrational sensitivity, feeling of pressure and weight.

Complex sensitivity: presence of astereognosis, violation of two-dimensional spatial and discriminative sensitivity. A sense of localization. Hyperpathies and paresthesias.

Meningeal signs (rigidity of the neck muscles, Kernig's sign and symptoms of Brudzinsky and Lesage for young children).

Reflective sphere.

Tendon reflexes (flexor-ulnar, extensor-ulnar, knee, Achilles), periosteal reflexes (carporadial). The level of reflexes, their symmetry, the presence of hypo-, hyper- and areflexia.

Cutaneous reflexes (upper, middle, lower abdominal, plantar and cremasteric in men).

Clonus of the feet and kneecaps. Symptoms of Babinski, Oppenheim, Rossolimo, Zhukovsky. Analogues of pathological reflexes on the hands. Reflexes of oral automatism (Marinescu-Radovichi, nasolabial).

Organs of the small pelvis. Delayed and paradoxical enuresis, imperative urination disorders. Frequent urination. Fixed

Vegetative functions. Sweating (general and local hyperhidrosis), pilomotor reaction. Dermographism (red, white, pink, raised). Asymmetry of skin temperature. Fat secretion.

Oculocardial reflex, tenderness of the solar plexus upon deep palpation, ichthyosis, brittle nails, hirsutism and baldness (alopecia), focal alopecia.

State of consciousness.

Mood. Thinking and intelligence. WARNING. Memory. Orientation in the environment, place, time and own personality. Emotions. Behavior. Delusions, hallucinations, symptoms of obsession and phobia. Sleep. Speech - (motor and sensory aphasia and their characteristics in the patient; slurred speech, dysarthria, stuttering, etc.).

Clinical and laboratory research.

The patient's medical history includes tests of blood, cerebrospinal fluid, urine, and feces; biochemical studies, X-ray examination data (chest X-ray, X-ray of the skull, spine, etc.); data of paraclinical examination methods (EEG, EEG, EMG EMG, CT, MRI and others) diagnoses of consultants (therapist, ophthalmologist, ENT and others).

Summary data on the disease syndrome and symptoms.

The student (supervisor) must systematize the patient's complaints and objective examination data into a certain neurological syndrome, and then explain the etiology and pathogenesis of this or that neurological syndrome.

Topographic diagnosis.

Based on the data obtained through an objective examination of the patient and the syndrome of the disease, the manager must mark (show) the localization (topography) of the damage to the brain and spinal cord, peripheral nervous system.

Differential diagnosis.

Differential diagnosis is carried out by comparing similar symptoms of different diseases, and then analyzing the signs on which these diseases are differentiated. After differential diagnosis, a final neurological diagnosis is made. Concomitant diseases unrelated to the main pathology are not taken into account.

Etiology, pathogenesis, pathomorphology. Modern data on the etiology, pathogenesis of this disease, as well as the main pathomorphological changes are presented.

Journal of the disease.

The disease log is filled in during patient observation, the dynamics of the disease, the effectiveness of medicines are displayed

Prognosis of the disease.

The prognosis of this disease is given in relation to life, recovery of working capacity, in general, and, in particular, for the controlled patient.

Epicrisis. Epicrisis contains the name and surname of the patient, age, profession, date of admission to the clinic, main complaints, data of objective examination and laboratory tests, which make it possible to make a correct diagnosis. The treatment of this neurological disease and its results, the course of the disease, recommendations for the future and the final clinical diagnosis are shown.

Appendices: a temperature graph, a scheme for researching fields of vision and sensitivity, drawings, schemes and a list of the studied special literature on this disease by the student who treated the patient.

The medical history must be signed by the student.

EXPLANATORY PART

History (from the Greek - hystori) - this document contains information about the health of the patient during his life, about heredity and factors that in one way or another can affect the health and complaints and the objective condition of the patient immediately after admission to the hospital and during his tenure there.

The doctor's conclusions about the form of examination, diagnosis and treatment, as well as their results, must be presented in the medical history. In addition, it reflects the general somatic status, which can play a significant, and sometimes the main role in diagnosis. Special attention is paid to the neurological status, which solves the question of medical tactics and the effectiveness of the treatment. The medical history is an important medical and legal document that needs to be correctly and clearly drawn up.

The medical history of a neurological patient begins with the passport part, which must include columns that require filling:

The full name should be written legibly, according to documents (passport, identity card), and in the absence of documents - from the words of the patient. If the patient was delivered in an unconscious state, then from the words of relatives, acquaintances, accompanying persons. The indicated passport data are necessary to substantiate the identity of the patient, and may have a certain value in diagnosis.

Many diseases of the nervous system occur at one age or another. For example, tumors of the pineal gland occur more often in children, multiple sclerosis affects mainly young people (16-25 years old), gray matter sclerosis affects the elderly, etc.

There are diseases specific to a certain gender. Yes, small chorea mainly affects girls. Due to certain, often unexplained circumstances, certain diseases occur more often in representatives of a certain nationality (Ashkenazi Jews suffer from Mediterranean anemia or periodic disease, and, on the contrary, the native inhabitants of Tajikistan practically do not suffer from multiple sclerosis).

The profession of the patient and the nature of work determined by it can also help in solving the question of diagnosis. It is known that livestock breeders suffer from brucellosis more often. Poisoning with carbon disulfide, lead, arsenic, and other industrial toxins occurs in persons of certain professions. If the patient is on old-age or disability pension, it should be emphasized who he worked for before retirement or disability and what disability group he has.

The home address is recorded from documents, from the words of the patient, or accompanying persons. It should be clarified whether the patient lives at the place of registration or at another address. It is necessary to write down the phone number (if available), as well as the surname and phone number of relatives (children, parents, brothers, sisters), neighbors, close acquaintances, so that, if necessary, you can invite them to solve emerging problems (deterioration of the condition, the need to solve the issue about operative treatment or sudden death of the patient).

The patient's residence in other geographical latitudes or stay abroad can be of great importance, because some diseases are somewhat dependent on geographical conditions: for example, in areas with a humid climate, rheumatism is noted more often than in dry areas; tick-borne encephalitis affects people living in taiga areas; hypertension and its complications in the form of cerebral hemorrhage do not occur among residents of high mountain areas.

The date and time of the disease are important for acute and subacute onset, poisoning, injuries, surgical diseases, various forms of comatose states, acute and fulminant forms of meningococcal meningitis or Landry-type ascending myelitis, acute intracranial hematoma, etc.

The date and time of seeking medical help can also affect the diagnosis, conflict situations also arise in cases of untimely medical care, especially with adverse consequences of the disease.

Diagnosis of the institution that referred the patient. It is necessary to record the diagnosis according to the direction (polyclinics, ambulance teams, etc.). Or note that the patient was admitted without a referral, delivered by a passing vehicle or brought by passers-by. If delivered by accompanying transport, then the names of the accompanying persons, the number of the vehicle, note the place from which and under what circumstances the patient was taken should be recorded.

A clinical diagnosis is a diagnosis made in a hospital after examination and clarification.

The main diagnosis is the disease for which the patient was hospitalized and for which treatment was carried out. For neurological patients, the basic diagnosis includes the concept of nosological form of the disease, as well as the topical location of the focus (for example: transverse myelitis at the level of the Th7 segment of the spinal cord, or a stroke (ischemic, hemorrhagic) in the area of the internal capsule, right/left) hemisphere.

Accompanying diagnosis. This is a pathology on the part of other organs and systems, which is currently not leading, does not determine the general condition of the patient and is not the reason for hospitalization.

Complications of the disease (for example: urosepsis or swelling and dislocation of brain structures).

If the patient received surgical assistance, the name of the operation, date and time of its performance are recorded.

In the medical history of neurological and neurosurgical patients, first describe the somatic status, which is accepted at the Department of Internal Diseases Propaedeutics, and then the neurological status.

ANAMNESIS

The basis of the analytical part of the diagnostic work of a doctor of any specialty is a thorough study of the anamnesis.

Anamnesis (from the Greek anamnesis - memory) is a set of information that is reported to the patient and his environment and is used in establishing the diagnosis and prognosis of the disease. The very process of collecting this information is called anamnesis collection and takes place by questioning the patient. In some cases, the doctor has to interview not only the patient, but also his close relatives, co-workers, surrounding or accompanying persons in those cases when the patient is in an extremely serious condition or if the patient is deaf and dumb, has a speech and writing disorder as a result of a brain disease, mental disorders.

Anamnesis is of great importance both for recognizing the disease and for solving the question of its possible consequence. Therefore, the collection of anamnestic information is one of the most important parts of a comprehensive examination of patients. The most outstanding clinicians of domestic medicine M.Ya. Mudrov, S.P. Botkin and others attached great importance to the collection of anamnesis, and G.A. Zakhar'in scientifically substantiated and developed the method of questioning the patient and brought it to the level of art.

COMPLAINTS

This section describes complaints on the day of the patient's curation. It is very important for a novice neurologist to develop and approve the systematics of clinical examination of a neurological patient. It is necessary to learn to conduct detailed, purposeful and in-depth questioning of the patient about his illness, to be able to listen to complaints in order to systematize them later. When talking with the patient, it is necessary to understand his experiences, to assess the correctness and consistency of the presentation of the story about his illness. You should learn about the feelings that bother the patient, and not be skeptical of his complaints.

The survey must be conducted carefully, detailing facts that may sometimes seem random, minor, unimportant. Depending on individual and intellectual characteristics, some patients describe their complaints in detail, others express themselves uncertainly, focusing on secondary or random facts. The patient does not always manage to express subjective feelings accurately. It is characteristic of some patients to exaggerate their experiences, sometimes to treat the manifestations of the disease inadequately, to overlook or not pay attention to those facts and details that are important to the doctor. Leading questions of the doctor should contribute to the correct, consistent presentation of the patient's feelings.

It is undesirable to conduct a survey of the patient in such a form that may cause an incorrect answer, one should also not impose one's vision of the symptom or disease on the patient, which often leads the curator himself to errors in diagnosis, especially in cases where secondary symptoms or symptoms at a "distance" » currently prevail over the immediate symptoms.

LIFE HISTORY OF THE PATIENT

This section displays all periods of the patient's life, starting with the age of the parents: during this pregnancy, how was the delivery, what complications were observed during pregnancy or delivery, how was feeding (breast or artificially), when did he start walking, talking, how was childhood, what diseases he was suffering from at that time. It is also necessary to specify living conditions, development during the school period: when he started studying, was it easy or difficult to study, what was his character in childhood and youth. They determine the time of onset of puberty (for women - the menstrual cycle, peculiarities of sexual life, pregnancy, childbirth, miscarriages, abortions), from what age one is married. Family composition, health status of family members. Living conditions, food. Beginning of employment, profession, job description, change of employment conditions due to illness. Work regime, working conditions. It is necessary to clarify what could have preceded and what the development of the disease is related to, whether there are similar patients in the family and close relatives or in the surrounding (this means contact with infectious patients). It is necessary to find out the allergic history, bad habits and their abuse.

When collecting anamnesis, it is necessary to carefully find out whether parents and other family members had the same diseases.

History of this disease

It is necessary to establish whether the patient is suffering from a disease of the nervous system for the first time or it is a recurrence of the disease. If this is the first manifestation of the disease, then it is necessary to clarify the period of development of the real disease, its course, from which sensations the disease began and what has changed during the entire period of development of the disease. If there is a relapse, it is specified when and where the disease started, when and under what circumstances the relapse occurred, what contributes to the development of the relapse, its frequency and in what periods of time it develops, the duration of the course of the relapse.

All changes in the clinical course of the disease from the moment of occurrence to the day of curation of the patient are clarified. When asking about the disease, it is necessary to find out when and how the disease developed, to determine the nature of development - sudden, gradual, fast or slow, the time of appearance and sequence of development of individual signs of the disease, subsidence or disappearance of earlier symptoms and the appearance or layering of new ones; highlight the main symptoms of the disease (pains, paralysis, convulsions, hyperkinesis). They increase in intensity or weaken, have a constant, paroxysmal, progressive or regressive character, which is the sequence of symptoms.

In neurological patients, paroxysmal states, epileptic seizures, crises, fainting are specified in detail, with the determination of the time of appearance, their form and frequency, dependence on external or other factors. It is necessary to find out the indicators that directly or long preceded the disease (infections, intoxications, physical and mental overstrain, etc.).

It is determined how the disease progressed before the patient was cured and whether there were deteriorations or relapses. It is clarified whether previous laboratory tests, treatment and their results, conclusions of the doctors who performed the treatment were carried out. In the same section, it is necessary to describe the history of concomitant diseases, if any, and their impact on the main disease.

At the very beginning, it is necessary to find out when and from what the disease began, what were the first sensations or manifestations (symptoms) of the disease. In neurology and neurosurgery, these symptoms are of great importance, as they are most often immediate, primary focal symptoms. During the development of the disease, the primary symptoms may be overlapped by symptoms in the neighborhood or symptoms at a distance.

It is necessary to establish the nature of the development of painful phenomena (sudden, gradual, rapid or slow growth of symptoms), the time of appearance and the sequence of development of individual signs of the disease. The main symptoms of the disease (pain, paralysis, convulsions, atrophy, hyperkinesis) should be identified, specify how long ago they developed, whether they increase in intensity or, on the contrary, weaken, are paroxysmal or constant, and what is the sequence of symptoms.

In particular, it is necessary to ask in detail about paroxysms, epi-attacks: crises, fainting, the time of their development, the form and frequency of their manifestation and duration, factors contributing to their occurrence. It is also necessary to identify immediate and long-preceding factors (infections, intoxications, injuries, etc.). It is necessary to find out how the disease progressed before the current examination of the patient and whether there were deteriorations or relapses.

GENERAL CONDITION.

Examination of the patient (inspectio). One of the main, sometimes the most important methods of direct examination of the patient. Examination began to be used at the beginning of the development of medicine and until the 19th century remained almost the only method of direct examination of the patient. Since then, examination has been the most important part of diagnosis, being a very valuable and reliable method of clinical research for doctors of any specialty.

Observation allows the doctor to catch the most subtle features of the body shape, posture, gait, condition of the skin and mucous membranes, judge the general development of the patient, facial expression and gaze. With the help of a general examination, the doctor develops a general idea about the patient's body.

The pathological signs noted by the doctor during the first examination not only provide significant help in asking questions during the collection of anamnesis, but sometimes allow making the correct diagnosis at "first glance" (characteristic changes of the facial skull in acromegaly, muscle atrophy in myodystrophy, muscle atrophy and shortening of the limbs in childhood poliomyelitis, violent movements in Huntington's chorea, the presence of a characteristic rash in infectious diseases, vascular "stars" in cirrhosis of the liver, etc.).

By examination, a number of characteristic signs indicating congenital anomalies, disorders of various body functions and anatomical changes in organs can be detected. Known combinations of these signs make up certain clinical symptoms and syndromes characteristic of certain diseases.

By synthesizing a number of received visual impressions during the examination of the patient, the doctor makes a general conclusion about the patient, which plays an important role in the prognosis of the disease.

The introduction of numerous instrumental research methods and the improvement of old ones lead to the fact that modern doctors pay little attention to direct examination without sufficient reason. Meanwhile, none of the most advanced devices can replace the eyes of an experienced doctor and give an idea of the patient as a whole.

In order to use all the possibilities inherent in the examination, the doctor must train in this method, constantly conduct examinations in compliance with certain rules, always strive for their consistency and completeness.

About the outstanding Russian psychoneurologist V.M. Bekhterev, who perfectly mastered the methods of clinical examination, contemporaries said that "he could make a diagnosis as soon as the patient entered his office."

The inspection is carried out in daylight or with daylight lamps that give the illuminated object a white color tone.

The inspection technique is simple. The patient is always examined from top to bottom. The subject's position should be such that the examined parts of the body are fully illuminated. Thus, it is better to examine the torso and chest in an upright position, and the abdomen in vertical and horizontal positions.

Doctors neglecting the technique and procedure of the examination miss the most important signs that give the key to the diagnosis of diseases. First, a general examination of the patient is carried out, which makes it possible to identify symptoms of general importance, and then - a special local examination of areas of the body, organs and systems.

A general examination, applied to doctors of any specialty from the first moment of his meeting with the patient, can be carried out according to the following scheme:

1) appearance (habitus)
2) gender of the patient;
3) age;
4) general condition of the patient;
5) head, face, neck;
6) torso (chest, stomach, back);
7) bones and joints;
8) state of nutrition;
9) skin, mucous membranes and hair;
10) genitals.

The general condition of the patient is judged by the following signs: state of consciousness and psyche, position of the patient, posture, gait. Some diseases are accompanied by disorders of consciousness, expressed in varying degrees - from blackout to coma.

Position of the patient. There are active, passive and forced positions.

An active position may not always be a criterion for the severity of the disease. Thus, with a brain injury in the so-called "light interval", when the patient develops a state of euphoria with a psychomotor component (patients are talkative, overly mobile, deny painful sensations), an impression is created of a satisfactory state of the patient, and after some time he falls into somnolence and sleepiness. Activity is preserved in the initial stages of severe incurable diseases, when individual sensitivity to pain sensations, timidity of surgical interventions, incredulity also affect the patient's indications.

The passive position of the patient is observed in cases of extreme weakness, loss of consciousness.

The forced position is typical for the patient when the painful sensations are weakened or stopped. For example, a forced sitting position with so-called "ortopnoea" reduces shortness of breath. Symptom of pneumonia - shortness of breath decreases when lying on the affected side, and with a rib fracture - on the healthy side. The position on the side with the head thrown back and the legs brought to the stomach, bent at the knee joints is observed in cerebrospinal meningitis. A forced standing position is observed with intermittent lameness, sometimes with angina attacks. Antalgic posture is characteristic of patients with radicular pain.

The lying position on the back occurs with severe abdominal pain (acute appendicitis, perforated ulcer of the stomach, duodenum, peritonitis). A forced lying position on the stomach is observed in patients suffering from a tumor of the tail of the pancreas, peptic ulcer disease (when the ulcer is located on the back wall of the stomach). With exudative pericarditis, severe pain in the abdominal cavity caused by tumor pressure on the solar plexus, a forced knee-elbow position, etc., is observed.

Posture. By the posture of the patient, by the way he holds himself, in many cases it is possible to judge the general tonus, the presence of strength, the development of the muscular system, the correctness of the nervous functions, often - about the profession, intelligence and habits.

Gait together with posture reflect the well-being and mood of the patient. Specific gait and posture are observed in Parkinson's disease and parkinsonism, in some other diseases of the nervous system (hemiplegia, spinal tuberculosis, neuropathies, myelitis, Huntington's chorea, Friedreich's disease, sciatica, etc.). Diseases affecting the joints (rheumatism, rheumatoid arthritis, gout, blood circulation disorders in the vessels of the lower extremities, surgical bone diseases) also complicate and change gait and posture.

During a general examination, the doctor's attention is fixed, first of all, on the open parts of the body - the head, face, neck, hands. In the further course of the study, other areas of the body, trunk and limbs are examined.

Head. The change in the size and shape of the skull has a certain diagnostic value. An excessive increase in the size of the skull is found in hydrocephalus (hydrocephalus). An increase in protruding parts of the head is observed in acromegaly. An abnormally small size of the head - microcephaly - is found in craniostenosis, and if mental retardation is present at the same time, then these are hereditary forms of diseases. A square shape of the head, flattened on top, with prominent frontal bumps indicates congenital syphilis or transferred rickets. A narrow and tall, so-called "tower" skull can be a symptom of hereditary and degenerative forms of diseases.

The position of the head is also of diagnostic importance in many diseases of the nervous and muscular systems, upper parts of the spine (myositis, myodystrophy, torticollis, cerebrospinal meningitis, consequences of transmitted tick-borne encephalitis, spondyloarthritis, arthrosis, etc.). Shaking movements of the head are observed mainly in the case of damage to the extrapyramidal system of the brain. Pulsation of the head is caused mainly by insufficiency of the aortic valves (Musset's symptom). Scars on the head can indicate an epileptic disease.

The patient's face often tells the doctor a lot and provides valuable diagnostic and prognostic data. In addition to various pathological, mental and somatic conditions, age, gender and physique affect facial expression. A mobile, expressive face at a young age seems to freeze

until old age. Feminine facial features in men and masculine facial features in women with endocrinopathies have a known semiological significance.

The following facial changes may have diagnostic value:

1. Puffiness of the face is observed due to:

a) general edema in diseases of the kidneys, heart, tumors of the mediastinum;

b) local stagnation with frequent attacks of suffocation and coughing (bronchial asthma, emphysema);

c) compression of the lymphatic channels with a large effusion in the pleural cavity and pericardium, with a tumor of the mediastinum, compression of the superior vena cava, jugular veins.

2. "Corvisar's face" is characteristic of cardiac decompensation. It is swollen, yellowish-pale with a bluish tint. The mouth is constantly half-open, the lips are cyanotic, the eyes are dull and stuck together.

3. A febrile face (faciesfebrilis) is characterized by hyperemia of the skin, shiny eyes, and disturbed facial expression. A febrile face with various infections has some features. Thus, with croupous pneumonia, the febrile blush is more pronounced on the side of the inflammatory process in the lungs. In case of typhoid fever, general hyperemia of a swollen face is observed, sclera are injected ("rabbit eyes"). In febrile tuberculosis patients, burning eyes on an emaciated, pale face with a limited blush attract attention. With septic fever, the face is swollen, motionless, pale yellow, blurred vision.

4. Change in facial features and its expression in various endocrine disorders:

a) acromegalic face is a person with an increase in its parts (nose, chin, brow ridges, cheekbones, prognathism).

b) myxedematous face - evenly swollen (mucosal edema), lack of facial expressions, reduction of eye slits, smoothed contours, hair loss on the outer halves of the eyebrows. The presence of blush on a pale background resembles a doll's face.

c) an intensely red, moon-shaped, shiny face with the appearance of a beard and mustache in women is observed in Itsenko-Cushing disease.

d) with hyperthyroidism: exophthalmos, glittering eyes, expression of fear, moisture and hyperemia of the skin of the face.

5. When the aortic valves are affected, a pale color of the skin of the face is observed, and with decompensated defects of the bicuspid valve - cyanosis of the face, lips, tip of the nose, and ears.

6. A lion's face with nodular thickening of the skin under the eyes and above the eyebrows and an enlarged nose is observed in leprosy.

7. Parkinson's mask - amemic face in patients with parkinsonism, characteristic of patients with chronic encephalitis and with disorders of cerebral blood circulation against the background of atherosclerosis.

8. The face with meningitis is concentrated, sometimes with a menacing expression, often the pupils are evenly dilated, while strabismus and ptosis may be observed.

9. The face of a "wax doll" - slightly puffy, sharply pale with a yellowish tint and as if the skin is translucent - occurs in Addison-Birmer anemia.

10. Sardonic face, "sardonic laughter" (risus sardonicus), in which the mouth is widened, as in laughter, and the forehead forms folds, as in sadness, observed when straightening (spasm of facial muscles in the form of a bitter smile).

11. A cachectic face - wasted, pale, with an earthy hue, is a sign of cancer, especially of the organs of the abdominal cavity.

12. The face of Hippocrates (facies hippocratica) is a change in facial features associated with collapse in severe diseases of the abdominal organs and described by Hippocrates: sunken eyes, a pointed nose, the skin of the face is deathly pale with a bluish tint, sometimes covered with large drops of cold sweat. Currently, this term is used to describe the appearance of a patient who is in a preagonal state.

13. Myopathic face (facies miopatisa), which is characterized by a smooth forehead, insufficient closing of the eye slits, sluggish and often thick lips ("tapir lips"). Patients cannot press their lips into a tube, whistle, put out a match, when smiling, the corners of the mouth do NOT rise up, but simply stretch to the side - a symptom called "transverse laughter".

14. Typical face with chorea: frequent blinking, rapidly changing grimaces, tongue protrusion.

15. The face with hydrocephalus is pointed to the chin with a wide, large head.

16. In Crouzon's disease, the tower skull is combined with diastosis of the front part of the skull.

17. With exanthematic diseases, children have an erythematous, scarlet, scaly face.

eyes An examination of the eyes and their appendages makes it possible to detect a number of important symptoms. Swelling of the eyelids ("bags" under the eyes) is one of the most visible manifestations of general swelling and is the first sign of acute nephritis.

It is necessary to pay attention to the color of the eyelids (dark color in case of Based's disease, tuberculosis, Addison's disease), to the presence of xanthomas, which indicate a violation of cholesterol metabolism.

An enlarged eye slit with eyelids that do not close is noted in facial nerve palsy. Persistent drooping of the upper eyelid (ptosis), converging or diverging strabismus are important symptoms of CNS damage. Narrowing of the eye slit is observed in myxedema with swelling of the face. Exophthalmos can be an individual feature, but it also occurs in severe myopia, Based's disease, retrobulbar tumors, tumors of the small wing of the main bone, endocrinopathy of the Devik type. Sunken eyeball (enophthalmos) is typical for myxedema, and is also one of the characteristic features of the peritoneal face. Unilateral depression of the eyeball, which is accompanied by narrowing of the eye slit, drooping of the upper eyelid and narrowing of the pupil, is defined as Horner-Claude-Bernard syndrome. The shape of the pupils, their uniformity, reaction to light, accommodation and convergence, "pulsation" are of great diagnostic importance in a number of diseases of the nervous system.

Oscillating movements of the eyeballs (nystagmus) are also symptoms of CNS damage. Indicators of pathology are the yellowish color of the sclera, their injection, hemorrhages in the sclera.

The nose can draw attention to itself with a sharp increase and thickening (acromegaly), distortion (trauma), change in shape, for example, with rhinoscleroma. A saddle-shaped, crushed nose occurs as a result of transferred gum syphilis, deformation of the soft tissues of the nose is observed in lupus.

Mouth. When examining the mouth, its shape is recorded (symmetry of the corners, constantly open or closed mouth), color of the lips, eruption of blisters on the lips (herpes labialis), cracks on the upper lip, cheilosis, cheilitis. The mucous membrane of the oral cavity is examined (presence of aphthae, pigmentation, Filatov-Koplik spots, thrush in infants, hemorrhages, cheek bites, which can often be observed in patients with epilepsy). Pronounced changes in the gums can be observed in a number of diseases (scurvy, pyorrhea, acute leukemia, diabetes, etc.), as well as in intoxications (mercury, lead).

When examining the teeth, various anomalies should be noted (irregularities in their shape, orientation, size); the absence of a significant number of teeth, which is of great importance in the etiology of a number of diseases (hereditary forms, diseases of the digestive tract).

The tongue is not a "mirror of the stomach", as it was believed before, but rather indicates the general condition of the patient. Disorders of his movements and atrophy are associated with damage to the caudal group of cranial nerves and the brain stem. Enlargement of the tongue is characteristic of myxedema and acromegaly. Sometimes an increase in the tongue is observed with glossitis. The appearance of the tongue can indicate a number of diseases:

1) clean, red and wet - with peptic ulcer disease;

2) crimson - for scarlet fever;

3) dry, covered with cracks and a dark brown coating in severe intoxications and infections;

4) covered with plaque in the center and near the root and clean at the tip and at the edges - in case of typhoid fever;

5) Gunter's tongue - with pernicious anemia;

6) the so-called mother-of-pearl tongue, covered with a thick white coating - with croupous pneumonia;

7) bluish-white, so-called porcelain tongue - with rheumatism;

8) "lacquered" tongue with smoothed nipples - in stomach cancer, chronic colitis, pellagra, sprue, ariboflavinosis;

9) with "abdominal catastrophes" and acute pancreatitis, the tongue becomes dry;

10) local thickening of the epithelium (leukoplakia) occurs in smokers;

11) raspberry tongue with smoothed papillae - with B-avitaminosis.

When examining the tongue, local pathological processes and changes can also be detected (ulcers of various etiologies, aphthae, scars (the presence of a tongue bite during epiattacks should be noted).

Examination of the pharynx by an ENT specialist is performed using mirrors, reflectors, a spatula, etc., but the duties of a doctor of any specialty include a simple examination of the pharynx, tonsils, and soft palate. Absence of movements of the soft palate or its unilateral movement is observed when the caudal group of cranial nerves is affected. Plaque on the tonsils may indicate diphtheria.

When examining the neck, it is necessary to pay attention to the pulsation of the common carotid arteries (occlusion, tumor in the area of the bifurcation of the carotid artery, insufficiency of the aortic valves, thyrotoxicosis), to the swelling and pulsation of the external jugular veins (insufficiency of the tricuspid valve, tumor of the mediastinum), to the increase in lymph nodes (tuberculosis, lymphadenosis, lymphogranulomatosis, lymphosarcomatosis, cancer metastases), for diffuse or partial enlargement of the thyroid gland (thyrotoxicosis, simple goiter, adenoma, soft tissue tumor).

When examining the chest, it is possible to detect: its deformation, general exhaustion, the development of collateral blood circulation with insufficient blood circulation in the basin of the superior vena cava, systemic damage to the lymph nodes.

Abdominal examination is performed with the patient in a vertical and horizontal position. They pay attention to the size and shape of the abdomen, the movement of the abdominal wall, the organs of the abdominal cavity, as well as the properties of the abdominal wall itself. The abdomen increases with obesity, flatulence, ascites, echinococcosis, ovarian cysts, the development of a large tumor (in the latter case, asymmetry of the abdomen is noticeable). When examining the abdomen in a vertical position, protrusions are more visible (hernias, navel, protrusion of the lower part of the abdomen in splanchnoptosis, ascites, large ovarian cysts), sagging of the thick abdominal wall - in obesity, edema. Abdominal asymmetry is observed with a significant increase in individual organs of the abdominal cavity: tumors, osmotic exudate, sharp swelling of an isolated intestinal loop, especially with twisting of the intestine (Wal's symptom), as well as with hernias and tumors of the abdominal wall itself. When examining the abdomen in a horizontal position, it is possible to detect peristalsis of the stomach in the case of stenosis of the gatekeeper or movement of the intestine in the case of stenosis, and the peristaltic area periodically becomes dense during palpation. Respiratory movements of the abdominal wall stop with acute peritonitis, sometimes there is asymmetry of respiratory movements with local peritonitis. Pulsation of the front wall of the abdomen is observed with an aneurysm of the abdominal vessels, aorta; with tumors located above the aorta; pulsation of the liver - with insufficiency of the tricuspid valve. When examining the navel, it can be noted that it is retracted with obesity, protrudes with ascites, and is smoothed with flatulence; it can be bluish with hemorrhage in the pancreas or with rupture of the tube as a result of an ectopic pregnancy. Retraction of the abdominal wall is noted in cachexia, long-term debilitating diseases, tubmeningitis, lead colic due to the spastic state of the

intestines and abdominal muscles. The presence of a network of enlarged and blood-filled subcutaneous veins of the abdominal wall allows you to recognize difficulties for the outflow of blood in the portal vein of the liver (the so-called "medusa head" - caput medusae).

When examining the skin of the abdomen, pay attention to rashes, striae, postoperative scars, the location of which allows judging the nature of surgical interventions.

When examining the back, various types of curvatures (scoliosis, kyphosis, kyphoscoliosis, lordosis), deformations and restriction of movements in different parts of the spine can be detected. You should pay attention to the position of the shoulder blades (wing-shaped shoulder blades are characteristic of myodystrophy). One- or two-sided tension of the back muscles indicates the presence of a pain syndrome in the area of the lumbar spine (symptom of the legs).

When examining the limbs, bones and joints, attention is paid to the configuration of the bones, the configuration and shape of the joints, the volume of movements in the joints, as well as to the tremor of the fingers, hands, feet or the entire limb (and sometimes all four limbs), which is observed in thyrotoxicosis, parkinsonism, alcoholism, neuroses. The so-called "dead finger" is observed in Raynaud's disease. It is possible to detect a number of changes on the part of the nails (change in shape, color; atrophy, hypertrophy; formation of white spots, transverse depressions, ridges), which have diagnostic value. By examination, characteristic changes of the terminal phalanges of the fingers of the hand in the form of Heberdon nodes and tympanic sticks, characteristic of bronchiectasis, are revealed. Movement disorders may be associated with joint diseases or damage to the nervous system (paralysis, atrophy, contractures). Unilateral swelling of the limb indicates a violation of venous blood flow (vein compression, thrombophlebitis, phlebothrombosis). Varicose veins are easy to see on the lower legs and thighs. Lameness (claudicatio intermittents) is characteristic of damage to the roots of the cauda equina of the spinal cord and obliterating sclerosis of the arteries of the lower extremities. High arching of the foot in combination with the cock-shaped position of the first toe, the so-called "Friedreich's foot", is a symptom of Friedreich's disease. Various deformations of the foot indicate osteodysplasia, etc.

The state of nutrition is also determined by the examination. The development of the subcutaneous fat layer can be normal or increased or decreased to varying degrees. Uneven deposition of the fat layer indicates obesity. A decrease in overall fatness can be associated with constitutional features (asthenic type), starvation, and debilitating diseases. The extreme degree of reduced overall nutrition is cachexia.

Examination of the skin plays an important role in determining the general condition of the patient. They pay attention to the color, elasticity, moisture of the skin, various rashes and scars. The color of the skin depends on the degree of blood supply to the skin vessels, the quantity and quality of the pigment, as well as its thickness and transparency. Pale color of the skin is associated with insufficient filling of skin vessels with blood (spasm of skin vessels of various origins, their desolation during acute bleeding, accumulation of blood in dilated vessels of the abdominal cavity during collapse, aortic valve disease, rupture of a large artery). The cause of paleness of the skin is also anemia, with some forms of anemia the pale color of the skin takes on a characteristic shade: yellowish - in pernicious anemia, earthy - in cancerous anemia, greenish - in chlorosis, ashen or brown - in the development of malaria and the color of "coffee with milk" - with subacute septic endocarditis. The reasons for the pale color of the skin can be its low transparency or significant thickness in completely healthy people. Redness of the skin can be temporary (febrile illnesses, overheating of the body, increased vasomotor activity) or permanent (in persons exposed to the effects of external temperature for a long time, as well as suffering from erythema).

Bluish coloration of the skin (cyanosis), as a rule, is due to hypoxemia in insufficient blood circulation, chronic lung diseases. Cyanosis is a pathological symptom observed in various diseases accompanied by circulatory disorders, as well as in metheglobinemia and sulfhemoglobinemia. Yellow coloring of the skin and mucous membranes is associated with impaired liver function. Dark brown or brown coloration of the skin is observed in adrenal insufficiency.

Very different skin rashes appear during allergic reactions, including to medicines. Skin peeling, scars, sores, ulcers, bedsores, inflammatory processes of the skin (furunculosis, lymphangitis, etc.) are of diagnostic importance; parasitic skin diseases.

Hair cover. During the examination, pay attention to the distribution of hair, its density, loss, gray hair (that is, color).

When examining the external genitalia, attention is paid to developmental anomalies, underdevelopment of secondary sexual characteristics, cryptorchidism, etc.

When examining the area of the anus, it is possible to detect the loss of the mucous membrane or the entire rectum, hemorrhoidal nodes, fissures, fistulas, dermatomes, condylomas.

Habitus (lat. Appearance, physical appearance, physique, posture) - the appearance of a person, the idea of which is based on general indicators characterizing his physique (height, weight, proportionality of individual parts of the body, degree of development of muscles and subcutaneous fat layer, character hair cover), as well as on the morphological features of individual parts of the body. Habitus of a healthy person is determined by heredity, sex, age, diet, profession and other factors. Certain features of the appearance are also related to the functional state of the glands of internal secretion. In some diseases, the patient's habitus acquires an important diagnostic value ("moon-shaped" face, peculiar deposition of fat, phenomena of hirsutism, giant growth, gray-earthy color of the skin, suffering facial expression, etc.). Habitus can reflect abnormalities caused by damage to the nervous system (ataxic gait - with damage to the cerebellum or the posterior columns of the spinal cord (spinal tuberculosis), limb shortening with muscle atrophy - after poliomyelitis in childhood, etc.). In other words, everything that emerges from a general overview is habitus.

Further, the examination is carried out by systems (as in the departments of propaedeutics of internal diseases): the respiratory system, the circulatory system, the digestive system, the urinary system, the blood system, and the system of the glands of internal secretion.

RESEARCH OF THE NERVOUS SYSTEM. MENTAL STATE.

Contact with the patient. State of consciousness. Orientation in time, place, space. Contact with others: contact, inaccessible, easy or difficult to communicate with others, with the doctor. Mental development (correspondence to his age and education. Attitude towards his illness.

Assessment of the severity of the condition and life prospects associated with the disease (critical, unjustified exaggeration of fear). Emotional sphere. Behavior during the examination: facial expressions, gestures, manner of presentation of anamnestic information, complaints (sequential, disorderly, tendentious, with theatrical affectation). Mood. Feeling of well-being Irritability. Fatigue. Inattention. Exhaustion. Lightheartedness. Sleep. Delirium. Hallucinations (visual, auditory, olfactory, tactile). Hypnogogic hallucinations. Illusions. Oneiroid state. Hypochondriacal state. Obsessive phenomena. Attention and memory (for near / distant events).

Amnesia: retrograde and antegrade.

The general condition of the patient: satisfactory, moderate, severe, very severe, extremely severe. These conditions are described in the same way as at the department of propaedeutics of internal diseases.

State of consciousness: it is clear when the patient is correctly oriented in the surrounding space, time and place, easily enters into language contact, correctly and consistently explains his feelings and anamnestic information.

Disorders of consciousness:

1) delirium - a type of clouding of consciousness, in which the patient has visual hallucinations (verbal - verbal - hallucinations are also possible), acute sensory delirium, various affective disorders. A prognostically unfavorable sign is the transition of delirium into muttering (muttering) delirium, or into "professional" delirium, in which there is a deeper clouding of consciousness. Mumbling (muttering) delirium - chaotic, disorderly excitement, usually in bed; indistinct mumbling with the pronunciation of individual sounds, syllables or words. At the height

of the violation, choreiform hyperkinesis or a symptom of carphology (picking) develops - senseless grasping movements or small movements of the fingers. Clinical signs of sopor and coma often develop after convulsive delirium. "Professional" delirium. In the picture of excitement, automated motor acts prevail over the influx of hallucinatory images. Patients perform the usual movements for their profession. For example, the patient, who is a janitor by profession, "sweeps" the sidewalk with an imaginary broom, etc. Disorientation in the environment and the lack of reaction to the environment are the same as in obsessive delirium;

2) stupor - the initial phase of loss of consciousness, when inhibition, lethargy, drowsiness, insufficiently clear orientation, decreased activity are observed. As the stun increases, sopor may develop. The development of this type of obscuration of consciousness should be considered a prognostically severe clinical sign. The lightest degree of unconsciousness is called obnubilation (from the Greek - cloud). The patient's consciousness is, as it were, overshadowed by a cloud, covered by a fog. Patients are lethargic, unhurried, restless and slow to answer questions. As the stupor increases, somnolence (hibernation) develops;

3) sopor is a state of deep stupor, when the elements of consciousness are preserved and the patient can be brought out of this state by strong sound, light, and pain stimuli or get an appropriate reaction; 5) the comatose state is characterized by loss of consciousness, lack of active movements, suppression of reflexes, loss of sensitivity, lack of reactions to pain, sound, light irritations and violation of autonomic and visceral functions. There are four degrees of coma: a) light, in which somnolence and loss of consciousness are observed, but awakening is possible and swallowing, breathing, blood circulation and thermoregulation are preserved; b) medium, in which consciousness is impaired, reflexes and sensitivity are lost, swallowing is impaired, but breathing, blood circulation and thermoregulation are preserved; c) severe, when consciousness, swallowing, heart activity are impaired; d) borderline coma, in which consciousness is lost, all vital functions (swallowing, breathing, blood circulation) are absent, thermoregulation is disturbed, all voluntary movements are lost, pronounced muscle hypotonia, areflexia, mydriasis. EEG without oscillation.

The position of the patient can be active, passive, forced. At the same time, it is necessary to describe the state of the head - with meningitis, tumors of the posterior cranial fossa, the head is in an extended position. A bent position of the head - with reduced intracranial pressure. The turning of the head to the side is observed in case of damage to the cerebellar hemisphere, in case of stenosis or aneurysm of the carotid or vertebral artery, in case of spastic torticollis and damage to the accessory nerve.

Posture of the patient: flexor, opisthotonus (sharply stretched trunk), etc.

Facial expressions and facial expressions are important symptoms that you should always pay attention to. It is necessary to note whether suffering, pain, fatigue, apathy, fright or anxiety, joy and other internal experiences are not reflected on the patient's face; whether there is no rapid change in facial expressions associated with emotional experiences; mask-like appearance of the face (amymia in parkinsonism); hypermimia, often observed with hyperkinesis (small chorea, Huntington's chorea), sometimes with aphasia, when the patient compensates for the lack of speech with facial expressions; Paramimia (facial expressions do not correspond to the experienced affect), observed in mental illnesses. Violent crying and laughter belong to expressive disorders of facial expressions (with pseudobulbar paralysis). Hemimimia, also called emotional paresis, is observed in the case of damage to the optic tubercle, when voluntary movements are preserved in the muscles of the face on the opposite side of the focus, but paresis is detected during emotions.

Cerebral symptoms.

General brain symptoms include headache, dizziness, vomiting, stupor, cardiac disorders.

Headache - localization, character (intense long-lasting, attack-like), dependence on the position of the head and time of day (night, morning, until the end of the working day); in infants - tension of the fontanel, percussive symptoms of divergence of cranial sutures (symptom of a cracked pot).

Dizziness - a feeling of rotation of the body or objects in combination with nausea and vomiting, which do not depend on food intake, but may depend on the position of the head and body, and may also depend on the intensity of the headache (systemic vertigo) or a feeling of instability and stupidity (non-systemic vertigo).

Vomiting - frequency, dependence on food intake, frequency and intensity of headache attacks, head position and other conditions. At the same time, it is necessary to pay attention to the nature of changes in cardiac (tachy, bradycardia, arrhythmia) and respiratory function (tachy, bradypnea, periodic breathing according to the Cheyne-Stokes, Biot, Kussmaul type - in terminal conditions).

Noise in the head - localization, character (constant or intermittent), whether noise is heard during auscultation of the head.

Membrane symptoms

This is a symptom complex consisting of general brain and focal symptoms caused by irritation or damage to the membranes of the brain, increased intracranial pressure and changes in cerebrospinal fluid (general brain symptoms, see above). Vomiting with membrane symptoms more often has a "founting" character. Some irritations (light, auditory, tactile) can increase membrane symptoms.

Stiffness of the muscles of the back of the head: in a patient lying on his back, when passively bending the head to the chest, the examiner feels tension - stiffness of the muscles of the back of the head, and it is not possible to bring the head to the chest.

Kernig's symptom: the patient, who is lying on his back, bends his leg at a right angle in the hip and knee joints, and when trying to straighten the leg, there is a sharp tension in the leg flexors, as a result of which the leg cannot be straightened.

Brudzinsky's symptom: upper - when bending the head and trying to bring the chin to the sternum, the legs in the knee and hip joints bend in response; medium - when pressed in the pubic area, the legs bend in the hip and knee joints in response; lower - when one leg is bent (as in Kernig's symptom), the opposite leg is bent in response in the knee and hip joints.

The "lying dog" pose: bending the legs and bringing them to the stomach of the patient lying on his side. Lesage's symptom (hanging) in children: bending the legs and bringing them to the stomach when lifting the child under the armpits. Functions of cranial nerves and their disorders

Anatomically, 12 pairs of cranial nerves are distinguished, which are clearly visible at the base of the brain and are divided into three purely sensitive (I, II, VIII), 6 motor (III, IV, VI, VII, XI, XII) and 3 mixed (V, IX, X) pairs of cranial nerves. Some cranial nerves contain vegetative fibers, especially III, VII, IX and X pairs.

The first two of the cranial nerves (olfactory and visual) are different from the others, they are modified parts of the brain, carried to the periphery, while the others are similar to the spinal nerves, but their structure and functions are very original. Cranial nerves are further divided into a caudal group: IX, X, XI and XII pairs; bridge cerebellar angle group: VII, VIII, XIII pairs (sometimes V and VI pairs belong to this group due to the close exit of their roots from the brain stem); group of oculomotor nerves: III, IV and VI pairs; front, or oral, group: I and II pairs. Studies of the functions of cranial nerves require a special technique.

I pair - olfactory nerve (n. Olfactorius). Smell research is carried out with the help of soft aromatic substances (valerian drops, perfume, etc.) separately for the right and left halves of the nose. Substances such as ammonia, which irritates the receptors of the trigeminal nerve, cannot be used for this study. Disorders of smell are manifested in the form of anosmia - loss of smell, hyposmia - reduced perception and hyperosmnia - aggravation of smell (in pregnant women, hysterics). False olfactory sensations and hallucinations can be observed with irritation and damage to the cortical part of the olfactory analyzer. False olfactory sensations and hallucinations are sometimes observed with irritation and damage to the cortical part of the olfactory analyzer and can be the initial phase (aura) of an epileptic attack.
II pair - optic nerve (n. Opticus). Visual acuity (visus) of each eye is determined using Syvtsev's

table. Visual acuity is normally equal to 1. Decreased visual acuity, that is, amblyopia, can be observed with optic neuritis, with primary or secondary (after stagnation) atrophy of the optic nerve. In the case of a complete damage to the optic nerve, blindness occurs in the given eye - blindness - with the loss of the pupil's direct reaction to light and the preservation of the sympathetic reaction. With lesions in the region of the chiasm, optic tract, subcortical visual formations (external geniculate body), Graziole's bundle, and the cortical part of the visual analyzer of the occipital lobe, loss of visual fields is observed. When the crossed fibers are damaged in the region of the chiasm, the so-called temporal, or bitemporal, hemianopsia occurs, and the external, uncrossed fibers – binasal hemianopsia. These hemianopsias are also called variable, since in one eye the right field of vision falls out, in the other - the left field of vision. Hemianopsia of the same name, or homonymous, occurs when the optic tract, optic tubercle, internal capsule, and occipital lobe are affected. Quadrant hemianopsia, or scotomas, may be observed with lesions in the area of the Graziole bundle and cortical divisions. Visual field measurements are performed on the perimeter of each eye separately. Normally, the field of vision for white has the following limits: outer -90 °, inner and upper - 60 °, and lower - 70 °. In addition to the above, the fundus is examined and color perception is determined.

III pair - oculomotor nerve (n. Oculomotoris). Symptoms of damage to the oculomotor nerve: ptosis, divergent strabismus, restriction of movement of the eyeball up, in and down, double vision - diplopia when looking straight ahead, paralytic mydriasis, paresis of convergence and accommodation, loss of pupillary reaction to light and convergence.

IV pair - block nerve (n. Trochlearis). When the block nerve is damaged, paresis of the upper oblique muscle of the eye develops. Characteristic symptoms: restriction of movement of the eyeball downwards and outwards, diplopia when looking down.

V pair - trigeminal nerve (n. Trigemenus). Movements in the lower jaw are studied, deviations of the lower jaw to the side are revealed when the motor portion of the trigeminal nerve is affected. Pain, paresthesias and areas of impaired sensitivity on the face are determined, as well as soreness under pressure in the area where the branches of the trigeminal nerve exit: fissurasupraorbitalis, forameninfraorbitalis, foramenmentale.

The following types of sensitivity disorders are distinguished - peripheral, in which the innervation of the skin of the face corresponds to three branches, and segmental (bulb type, or Zelder's zone) in the case of damage to the descending nucleus of the trigeminal nerve. When the trigeminal nerve is damaged, the lower jaw deviates in the direction of the lesion, the tone and strength of the masticatory muscles are reduced, and the temporal and masticatory muscles atrophy. A sensitivity disorder is observed: pain, paresthesias (on the side of nerve damage) on half of the face according to the hemitype. When the upper branch (n. Ophthalmicus) is affected, the pain is localized in the area of the eyes, forehead, and temple; middle branch (n. maxillaris) - in the upper jaw; lower branch (n. mandibularis) - in the area of the chin and lower jaw. The trigeminal nerve provides sensitivity to the mucous membranes of the eye, nose, paranasal sinuses, oral cavity, tongue, and half of the facial skin. When it is damaged, the following reflexes are lost: corneal, conjunctival, mucous membranes of the nose, masseter reflex and mandibular reflex. Pains in the face of uncertain nature are called prosopalgia.

VI pair - abductor nerve (n. Abducens). Convergent strabismus, restriction or absence of outward movements of the eyeball, and diplopia when looking in the direction of the affected muscle are observed when the abductor nerve is damaged.

When examining the oculomotor nerves, they establish the uniformity and width of the eye slits, determine the amount of eyeball movements (up, down, inward, outward), determining whether there is a weakness of the eye muscles, which causes divergent (strabismusdivergens) or convergent (strabismusconvergens) strabismus. These squints are evaluated horizontally.

When the brain stem is restricted in the tentorial foramen (fissure of Bish), there is a vertical divergence of the eyeballs (Hertwig-Mozhandi syndrome). Determine whether there is paralysis of gaze: up, down, to the sides, as well as paresis of convergence and accommodation.

Pupils can be round, oval, irregular in shape: narrowed - miosis, dilated - mydriasis. The pathology is evidenced by: external ophthalmoplegia - a syndrome of damage to the large cell nucleus of the III pair; internal ophthalmoplegia – a syndrome of damage to the small cell nuclei of the III pair (Yakubovich-Edinger-Westphal and Perl nuclei); mydriasis - immobility of the pupil, violation of accommodation.

With simultaneous damage to the external and internal muscles of the eye, complete ophthalmoplegia occurs: complete immobility of the eyeballs, ptosis, strabismus, paralytic mydriasis, immobility of the pupil, accommodation disorder, exophthalmos.

VII pair - facial nerve (n. Facialis). For a considerable distance, the companion of the facial nerve is the intermediate nerve n. Intermedius (Wrisbergi), also called the XIII cranial nerve, which contains centripetal sensitive, or rather - taste, and centrifugal secretory salivary fibers. Facial nerve paralysis can be peripheral or central.

When the nucleus or the root of the facial nerve is damaged, peripheral paralysis of facial muscles occurs, prosopaplegia - on the side of the lesion. If sensitive or pyramidal pathways are involved in the focus, the alternating Miyar-Gubler syndrome develops. When cortico-nuclear pathways are affected, central paralysis develops, which is manifested by damage to the lower part of the facial nerve on the opposite side of the focus; the eyebrow reflex is preserved and even strengthened.

Peripheral paralysis of the facial nerve is characterized by the absence of folds when wrinkling the forehead, the inability to close the eyelids, when the upward movement of the eyeball is visible (Bell's phenomenon); lagophthalmos; inability to frown; lowering and smoothing of the nasolabial fold; inability to grind teeth, whistling, loss of eyebrow reflex.

Damage to the root of the facial nerve in the area of the bridge-cerebral angle is usually combined with deafness, while dry mouth may be felt (hyperacusis will not occur due to deafness). During processes in the area of the bone canal to the branching of the stony nerve, at the same time as paralysis of the facial nerve, dry eyes, a disorder of taste and salivation, hiperacusus (damage of n. Stapedii) are noted; after the branching of the stony nerve, the symptoms will be the same, but instead of dry eye, there is, on the contrary, increased lacrimation. In the case of damage below the branches of n. Stapedii is observed paralysis of facial muscles, lacrimation, taste disorder and salivation. The taste is examined on the front two thirds of the tongue by applying a drop of salty, sweet, sour. Loss of taste is called agesia.

VIII pair - auditory nerve (n. Acusticus &Vestibulo-cochlearis). Under the general name n. Acusticus unites two completely independent sensory nerves carrying different functions - pars cochlearis and pars vestibularis. Pathology of the hearing aid and hearing research is considered in detail in the course of otolaryngology.

In neurology, hearing loss is designated as hypoacusis, hearing loss as anacusis or surditas, and increased perception as hyperacusis. It is always important for an ENT specialist and a neurologist to distinguish hearing loss depending on pathological processes in the middle ear (tympanic membrane, auditory ossicles) and "neural" deafness or deafness (organ of Corti, cochlear nerve and nucleus). The first case is characterized by a decrease in hearing to low tones and preservation of bone conduction; for the second - loss of perception of high tones and loss of bone conduction.

The following cochlear functions are investigated: hearing acuity on the right and left (whether the patient hears a whisper at a distance of 5 m, or hears the sound of a tuning fork located near the ear canal, on low and high tones); Rinne's test - the ratio of bone and air conductivity (after examining the air conductivity with a tuning fork in the auditory canal, the tuning fork is placed on the mastoid process); Weber's test (a tuning fork is placed on the examinee's crown). When the sound-conducting apparatus is damaged, the sound is strongly perceived by the affected ear - the so-called "lateralization" of sound to the affected side; and with damage to the nervous system - "lateralization" to the healthy side.

The vestibular apparatus performs the function of orientation of a person in space. As a result of its damage, balance disorders, nystagmus, dizziness, vomiting occur. When examining

vestibular functions, caloric and rotational tests are performed, the presence of nystagmus is revealed, which can be horizontal, vertical, positional nystagmus, large- or small-sweeping, smooth or tonic, constant or paroxysmal.

Dizziness in disorders of vestibular functions are systemic ("objects rotate" from right to left or left to right, or the patient himself "rotates" along an axis in one direction or another), the conditions that provoke dizziness are specified.

IX pair - glossopharyngeal nerve (n. Glossopharyngeus). Mixed nerve. It is related to both somatic and autonomic innervation. Its motor portion is very small, it innervates only one silo-pharyngeal muscle, which raises the pharynx during swallowing. The motor nucleus shared with the X pair is located in the middle part of the medulla oblongata and receives bilateral cortical innervation.

Sensitive fibers start from the upper and lower jugular nodes. The dendrites of the cells of these nodes branch out in the back third of the tongue, soft palate, pharynx, pharynx, front surface of the epiglottis, auditory tube, and tympanic cavity. Axons from the lower node penetrate the medulla oblongata and end in the taste nucleus, and axons from the upper node, which carry general sensitivity, end in another nucleus - n. Alaecinereae.

In case of damage, the following is observed: loss of taste (ageusia) on the back third of the tongue, which is detected by applying solutions (salty, sweet) with a pipette to the root of the tongue; anesthesia of the mucous membrane of the upper half of the pharynx; a minor disorder of swallowing, which is rare, because the main innervation of the pharyngeal muscles is carried out by the vagus nerve.

Unilateral shutdown of the parotid gland (glandulae parotis) can lead to slight dryness of the mouth, which is usually compensated by the activity of other salivary glands.

X pair - vagus nerve (n. Vagus). It is related to somatic and vegetative-visceral innervation. With a unilateral lesion, there is a hanging of the soft palate on the side of the lesion, a deviation of the tongue (uvula); during phonation, it turns out that the voice acquires a nasal tone. As a result of unilateral vocal cord paralysis, the voice becomes hoarse - the so-called dysphonia. The pharyngeal reflex is lost on the affected side of the pharynx, which is determined by touching the spatula to the mucous pharynx.

With a bilateral lesion of the X nerve, there is sloppiness, pouring of liquid food through the nose due to paralysis of the soft palate, loss of voice up to complete aphonia. Paralysis of the epiglottis causes dander when eating. Swallowing disorders occur - dysphagia. Tachycardia and bradypnea may be observed. Complete bilateral damage to the X nerve leads to death (cease of cardiac activity and breathing).

XI pair - accessory nerve (n. Accesorii). Damage to the accessory nerve is manifested by difficulty in raising the shoulders and turning the head to the healthy side, the impossibility of raising the shoulder above the horizontal line. At the same time, the lowering of the shoulder and the departure of the lower edge of the scapula on the side of the nerve lesion are noted.

XII pair - hypoglossal nerve (n. Hypoglossus). With peripheral paralysis, atrophy of the tongue is observed on the side of the lesion with atrophy and thinning of the tongue muscles. Damage to the nucleus is accompanied by fibrillar and fascicular contractions of the muscles of the tongue. With a bilateral lesion, paralysis of the entire tongue develops, anarthria, and intake of liquids and food becomes difficult. A unilateral lesion of the corticonuclear pathway causes a deviation of speech to the side opposite to the focus of the lesion, since the nucleus of the hypoglossal nerve is connected only with the opposite hemisphere of the brain.

Symptom complexes of motor disorders that occur as a result of damage to the caudal group (IX, X, XII) of cranial nerves is called bulbar or pseudobulbar paralysis.

Bulbar paralysis occurs as a result of damage to the nuclei or roots of the caudal group. At the same time, speech disorders (dysphonia, aphonia), hoarseness of voice, swallowing disorders, choking on food, pouring of liquid through the nose, dysarthria or anarthria are observed. All these disorders are fully manifested in a bilateral process; they are characteristic of peripheral paralysis.

Therefore, atrophy of the tongue, fibrillar twitching in it, a reaction of regeneration, extinction of the pharyngeal reflex are typical here.

With a unilateral lesion of the corticonuclear pathway, the loss of functions of the caudal group is extremely minor. The only thing that is noted is the deviation of the tongue to the weak side.

With bilateral damage to the corticonuclear pathways, the entire symptom complex of bulbar disorders is presented in full, as with bulbar localization of the process, and is called pseudobulbar paralysis.

Violations of functions in both cases will be the same, but as with any central paralysis, in the absence of atrophy, reflexes of oral automatism are revealed.

MOTOR FUNCTIONS AND THEIR DISORDERS.

Voluntary movements arise as a result of the implementation of those programs and plans that are formed in the motor-functional systems and are aimed at satisfying certain needs of the body.

The study of motor functions must begin with an examination of the executive organs of movement - muscles. The configuration and relief of the muscles are determined (the musculature of the same areas of the trunk and limbs is compared). By measuring the muscles with a centimeter tape, the degree of expressiveness and localization of trophic disorders (atrophy, hypertrophy) are revealed. They pay attention to the condition of the spine (scoliosis, kyphosis, lordosis), the shape of the hands (hanging hand, monkey or clawed hand), feet (horse's foot, Friedreich's foot), the location of the shoulder blades and waist ("wasp waist") in myodystrophy, on the shape of the chest ("tower-like") with atrophy of the pectoral muscles. Define pseudohypertrophy of muscles in myopathies. During the examination, the presence of fibrillary twitches (damages to the cells of the anterior horns, cells of the nuclei of motor cranial nerves) is revealed.

Study of active movements. First, they find out whether the patient can actively move the limbs in all joints, these movements are carried out in full. When movement restriction is detected, the doctor uses passive movements to rule out local lesions of the bone-joint apparatus (ankylosis) and other processes that prevent movement. This kind of "immobilization" does not belong to the category of paresis or paralysis. Movements in the mandibular joint are studied. Bending, stretching, turning and tilting the head to the sides. Raising the shoulders up. Raising the arms to a horizontal position, then higher. Flexion and extension in the elbow joint. Flexion and extension in the radiocarpal joint, pronation and supination of the forearm. Flexion and extension of the fingers, adduction and adduction of the fingers, opposition of the 1st finger to the 5th. Flexion and extension in the hip joint, hip adduction and abduction. Extension and bending of the knee joint. Extension and bending in the ankle joint. Abduction and adduction of the foot. Flexion and extension of the toes. Movements of the spine: rotational inclinations of the trunk forward, backward and sideways. Walking on heels and toes.

The study of passive movements (carried out when active movements are disturbed) allows to differentiate between paresis or paralysis of the limbs and ankylosis, contractures, and other causes that prevent movement. Investigate the volume of passive movements in the same sequence as active movements.

Detection of paralysis in the absence of voluntary movements is not difficult. It is more difficult to detect paresis. First, they pay attention to reducing the amount of active movement, for example, when bending and extending a limb in one or another joint. However, this method of research has only indicative value and does not determine the degree of paresis.

The second method of detecting paresis is the study of the force of muscle contraction. In this way, you can get an idea of the degree of paresis and the formula for its distribution. This technique has been widely used in the clinic. There are devices with which you can quantitatively measure the force of muscle contraction, they are used in scientific research. Medical experience has shown that the force of contraction of various muscle groups can be determined not only by special technical devices, but also by the so-called manual method. Opposing some elementary,

arbitrary movement carried out by the patient, the researcher determines the degree of effort sufficient to stop this movement, which is how muscle strength is measured. For example, the strength of the forearm flexors is determined with full active flexion in the elbow joint. The patient is asked to hold his hand in this position with all his might. Grabbing the lower part of the forearm with the right hand and resting the left one on the middle of the patient's shoulder, the doctor tries to straighten the arm at the elbow. The results of the study are evaluated according to a five-point system: full muscle strength - 5 points, slight decrease in strength (flexibility) - 4, moderate decrease in strength - 3, the possibility of full range of motion after the removal of resistance - 2, safety of movement - 1. a muscle strength of 4 points indicates mild paresis, 3 points - moderate paresis, 1-2 points - deep paresis. The results of the study of muscle strength are recorded in the medical history. In some cases, electromyography is performed.

Determining the strength of individual muscle groups can be supplemented by observation of motility as a whole (walking, transition from a lying position to a sitting position, getting up from a chair, etc.). To detect paresis of the limbs, the Barre test (of a paretic limb) is used - the arm stretched forward or raised up in a lying patient is gradually lowered, the leg raised from the bed is also gradually lowered, while the healthy limb must be kept in the extended position.

Muscle tone is a stretch reflex (myostatic reflex), which provides reflex tension, resistance, elasticity of muscles and the posture of the limbs, trunk, and head. Muscle stretching causes a reflex tonic response. With normal tone, this tension is small, but clearly noticeable. With hypotonia, passive movements are performed without adequate resistance. For example, when bending the arm in the elbow joint, the wrist and hand can be brought to the shoulder joint. Palpation reveals muscle weakness. With increased muscle tone (hypertension), passive movements meet significant resistance, sometimes even difficult to overcome. This resistance has a peculiar limit. It is expressed only in the first moments of passive flexion and extension, then the obstacle seems to be removed and the limb moves freely - this is called a symptom of a folding knife. The muscles are tight to the touch. This type of hypertension is characteristic of damage to the pyramidal pathway.

Changes in muscle tone are also observed when the cerebellar and extrapyramidal systems are affected. With cerebellar muscle hypotonia, the muscles are weak, there is excessive excursion in the joints. With lesions of the extrapyramidal system, the study of passive movements can reveal discontinuity, as it were, gradualness of muscle resistance to passive stretching (gear wheel symptom). Plastic hypertension - increase in muscle tone evenly in flexors and extensors. Catalepsy - freezing of a limb in a given position.

PHYSIOLOGICAL REFLEXES

Reflexes are studied and their nature, uniformity, asymmetry is revealed, and when they increase, the reflexogenic zone is determined. In clinical practice, reflexes are divided by the location of receptors into superficial (skin, from mucous membranes) and deep (tendinous and periosteal). When describing reflexes, the following gradations are used: live reflexes, hyporeflexia, hyperreflexia (with an extended reflexogenic zone), areflexia.

Tendon and periosteal (periosteal) reflexes are caused by percussion with a hammer on tendons or periosteum, the response is manifested by the motor reaction of the corresponding muscle. To obtain tendon and bone reflexes on the upper and lower limbs, it is necessary to evoke them in the appropriate position, favorable for the reflex reaction (absence of muscle tension).

Brow reflex (periosteal) - eyelids close when percussion on the inner edge of the brow arch.

The mandibular reflex (periosteal) is caused by a hammer blow on the chin (the patient's mouth is slightly open), in response the masticatory muscles contract, the lower jaw rises up.

A reflex from the biceps muscle of the shoulder (tendon) is caused by a blow of the hammer on the tendon of this muscle, in response the forearm is bent (the patient's arm is bent at the elbow joint without tension).

Reflex from the triceps muscle of the shoulder (tendinous) - a blow of the hammer on the tendon of this muscle (the patient's arm should be bent at the elbow joint) causes extension of the forearm.

Metacarpal-radial (carporadial) - (periosteal) is caused by percussion of the styloid process of the radius. In response, there is flexion and pronation of the forearm and flexion of the fingers of the hand.

Ulnar reflex (periosteal) - flexion and pronation of the forearm upon percussion of the styloid process of the ulna.

The scapular reflex (periosteal) is caused by percussion with a hammer on the inner edge of the scapula, in response, the shoulder is adducted and rotated outwards.

The knee reflex is caused by a hammer blow on the tendon of the quadriceps muscle of the thigh below the kneecap, in response, the leg is extended.

The Achilles reflex is caused by percussion with a hammer on the Achilles tendon, in response, plantar flexion of the foot occurs as a result of contraction of the calf muscles.

The costal-abdominal reflex (periosteal) is caused by percussion with a hammer along the edge of the costal arch inward from the nipple line. In response, the abdominal muscles on the corresponding side contract.

Hyena-Triumphov's pubic reflex (periosteal) is caused by percussion with a mallet on the pubis, in response, the muscles of the abdominal wall on the corresponding side contract.

In addition to deep reflexes, superficial (skin) reflexes are studied in the clinic.

The corneal reflex is caused by touching (carefully!) cotton wool to the cornea. The eyelids close reflexively.

The conjunctival reflex is caused by irritation of the conjunctiva, and the eyelids close in response.

The pharyngeal reflex is caused by irritation of the back wall of the pharynx with a spatula, vomiting or coughing movements appear in response.

Sneeze reflex - sneezing reflexively occurs when the mucous membrane of the nose is irritated by cotton wool.

Abdominal reflexes - the upper one is caused by a line irritation of the abdominal skin parallel to the costal arch, the middle one - by the same irritation in the horizontal direction at the level of the navel, the lower one - parallel to the inguinal fold. In response, the abdominal muscles on the corresponding side contract.

If the abdominal wall is lax (for example, in women who have given birth many times or obese people, especially the elderly), it is recommended to stretch the skin of the abdomen by hand before examining the abdominal reflexes.

The cremaster reflex is the contraction of the muscle that raises the testicle when the inner surface of the skin of the thigh is irritated.

Anal reflex. When applying injections near the anus, its circular muscle (external sphincter of the anus) is shortened.

Plantar reflex - plantar flexion of the foot and fingers when the middle of the sole is irritated.

Distant reflexes

Blinking reflex - contraction of the circular muscles of the eyes during sudden illumination of the eyes or rapid movement of the hand in front of the patient's eyes.

Pupillary reflexes - a direct reaction to light when the eye is brightly illuminated is manifested by a narrowing of the pupil; conjugal - with light irritation of one eye - in the form of narrowing of the pupil is also observed in the opposite eye.

The reaction of the pupils to convergence and accommodation - the patient is asked to fix his gaze on the doctor's hammer or fingers, which is first placed at a distance of half a meter, and then, when approaching the bridge of the nose, the convergence of the eyeballs (convergence) and the narrowing of the pupils (accommodation) are noted.

The symptom of Argyle-Robertson consists in the absence of a direct reaction of the pupils to light while preserving it during convergence and accommodation (observed in spinal tuberculosis or pseudo tabetic syndrome). The distorted Argyle-Robertson reflex (or the reverse) consists in the absence of a reaction to convergence with the preservation of the reaction of the pupils to light (diphtheria polyneuropathy, epidemic encephalitis).

Pathological reflexes

Pathological reflexes are fairly constant and practically very important signs of damage to the central motor neuron. Depending on the reflex response, pathological reflexes are divided into flexor and extensor.

PATHOLOGICAL REFLEXES.

On the upper limbs

Rosolimo's reflex - bending of the fingers of the hand in response to rapid tactile stimulation by the researcher's fingers on the palmar surface of the terminal phalanges of the patient's 2nd-4th fingers. Similar to the Rossolimo reflex, the symptoms of Venderovich, Wartenberg, Bekhterev-Jacobson, Zhukovsky, etc. are caused.

Babinski's reflex - extension of the big toe when the sole is irritated.

Oppenheim's reflex - extension of the big toe with sliding-pressing movements of the thumb and forefinger on the crest of the tibia.

Gordon's reflex - extension of the big toe and fan-like separation of the other toes when the calf muscles are compressed.

Schaefer's reflex - extension of the big toe in response to compression of the Achilles tendon.

BENDING PATHOLOGICAL REFLEXES. On the lower limbs

The Rossolimo reflex is a quick plantar flexion of all toes in response to intermittent hammer blows on the toe pads.

Bekhterev's reflex - flexing the fingers and moaning when the heel is hit with a hammer.

Bekhterev-Mendel reflex - bending of the toes when tapping with a hammer on the back of the foot.

Zhukovsky's reflex - plantar flexion of the toes when struck with a hammer on the middle of the sole.

PATHOLOGICAL REFLEXES OF THE FACE

Corneal-mandibular reflex - the movement of the lower jaw in the opposite direction in response to the touch of the cotton wool to the cornea of the eye.

Corneal-mental reflex - contraction of the chin muscles when the cornea is scratched with cotton wool.

ADDUCTOR REFLEXES

Hirschberg's adductor reflex - adduction of the foot when the inner side of the sole is irritated.

Marinesko's adductor reflex - adduction and rotation of the foot inward with a line irritation of the inner edge of the foot.

Pierre-Marie adductor reflex - adduction of the opposite leg when percussing the tibia.

Embrace reflex

Protective reflexes (reflex-spinal automatism) are also characteristic of central paralysis. They represent involuntary complex tonic synergies in a paralyzed limb, arising in response to gross irritation of receptors of the skin or deep tissues.

Bekhtereva-Marie-Fois reflex - can be caused by a sharp passive plantar flexion of the toes, in response to reflex flexion of the leg in all large joints.

Remaku reflex - plantar flexion of the foot and fingers in response to line irritation of the skin of the front surface of the thigh.

Abdominal reflex of Astvatsaturov-Barre - in response to a single stroke irritation of the abdominal wall, involuntary bending of the leg in all joints and contraction of the abdominal muscles occurs simultaneously.

Cervical tonic reflexes arise in response to irritation associated with a change in the position of the head in relation to the body.

Magnus-Klein reflex - when turning the head to face the paralyzed limb, the extensor tone in the muscles of the arm and leg increases, and when turning the back of the head, the flexor tone of the muscles of the limbs increases; flexion of the head causes an increase in flexor, and extension of the head - extensor tone in the muscles of the limbs.

Gordon's tonic shin reflex - stiffening of the shin in the tonic phase when the knee reflex is triggered

Westphal's postural reflex - stiffening of the foot when it is moved to the dorsiflexion position.

Thevenar-Fois reflex - slow extension of the lower leg after passive bending in the knee joint in a patient lying on his stomach.

Yanyshevsky's grasping reflex - on the upper limbs, an involuntary grasping of objects that come into contact with the palm. On the lower limbs, the grasping reflex is manifested by increased bending of the fingers and foot when the sole is irritated.

Distal grasping reflex - an attempt to grasp an object that is shown to the patient at a distance (observed in frontal lobe damage).

PSEUDOBULBAR REFLEXES

Fronto-chin reflex Marinesko-Radovich - contraction of the chin muscles when the palm is irritated by a line.

Toulouse-Wurp reflex - protrusion of the lips when percussion on the upper lip.

Bekhterev's reflex - contraction of the mental muscles when percussing the chin.

Astvatsaturov's nasolabial reflex - pulling the lips with the proboscis when percussion with a hammer on the back of the nose.

Distance-oral reflex - pulling out the lips with the proboscis when the hammer is suddenly approaching them.

"Bulldog" reflex - tonic clenching of the jaws in response to the spatula touching the gums or lips.

Pseudobulbar reflexes include violent laughter and crying.

Clonus

Foot clonus - in response to an active shock-like dorsiflexion of the foot, rhythmic flexion and extension of the foot occur in the ankle joint. At the same time, the leg should be bent at a right angle in the knee and hip joints.

Clonus of the kneecap - rhythmic oscillatory movements of the kneecap when it is sharply and actively moved downward by the researcher's thumb and forefinger.

Neurogenic contractures

Contractures are persistent tonic muscle tension that causes limitation of limb movements or individual muscle groups. They are distinguished by their form - flexion, extensor, and pronator contractures; by localization - contractures of the hand, foot, mono-, para-, tri- and quadriplegic, by the method of detection - persistent and immobile in the form of tonic spasms (hormetonia); according to the time of occurrence after the development of the pathological process - early and late contractures; in connection with pain - protective-reflex, antalgic; depending on damage to various parts of the nervous system - pyramidal (hemiplegic), extrapyramidal (pallidonigral), spinal, with damage to peripheral and facial nerves.

Late Wernicke-Mann hemiplegic contracture, the upper limb is brought to the trunk, the shoulder is lowered, the forearm is bent, pronated, the fingers of the hand are in a bent position; the lower limb is adducted, the thigh and lower leg are extended, the foot is in plantar flexion and turned inward.

Early Davydenkov's contracture (hormetonia) is characterized by periodic tonic spasms in all limbs that occur as a result of various irritations.

Recommended Books.

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

2. Topical diagnosis of nervous system pathology. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.

3. Methods of examination of a neurological patient: teaching. Guide / edited by L. I. Sokolova, T. I. Ilyash. K., 2015. – 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: training. 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. Tutorial. - K.: Medknyga Publishing House, 2019. - 48 p.

2. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 1 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2020. - 258 p.

3. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 778 p.

4. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 642 p.

Electronic information resources

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

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

ISW No. 3

Topic: Tumors of the brain and spinal cord. Brain abscess.

Relevance of the topic : Tumors of the brain and spinal cord, brain abscess require timely diagnosis for the purpose of their effective treatment, therefore every doctor should know their clinical symptoms.

Specific goals:

As a result of independent study of this topic, students should:

know:

- Classification of brain and spinal cord tumors.
- Focal symptoms of CNS tumors.
- Cerebral symptoms that arise in connection with increased intracranial pressure (hypertensive syndrome).
- Features of the pathogenesis and course of headache in brain tumors.

be able:

- Suspect a CNS tumor.
- Diagnose the main symptoms of brain damage.
- Correctly use different methods of additional examination of the patient.
- Correctly interpret fluid dynamics tests and changes in the composition of cerebrospinal fluid.
- Use symptomatic means of treatment of brain tumors.
- Differentiate extra- and intramedullary tumors of the spinal cord

Topic content:

Classification (topical and pathomorphological). Clinic: cerebral, focal and dislocation syndromes. Differential diagnosis of brain and spinal cord tumors. Extra- and intramedullary tumors. The diagnostic value of ophthalmoscopy, cerebrospinal fluid examination, EEG, echo-encephaloscopy, angiography, ventriculography, MRI-CT tomography, spondylography, myelography and other methods in brain and spinal cord tumors. Principles of surgical and conservative treatment of brain and spinal cord tumors. Sources of abscessation. Clinic, diagnosis, differential diagnosis.

BRAIN AND SPINAL BRAIN TUMORS. According to various authors, brain tumors occur in 4-6 people per 100,000 population. They make up 6% of all human oncological diseases and are the cause of 1-1.5% of cases of pathological examinations. According to some authors (Babchyn I.S. 1973), brain tumors make up 4-5% of all organic brain diseases. Etiology and pathogenesis. The main theory of brain and spinal cord tumors is the polyetiological, dysontogenetic theory. According to this theory, hereditary factors, dysembryogenesis, injuries, carcinogenic effects, viral infection, intoxication, radiation, etc., play an important role in the development of tumors.

I. Classification. There are several classifications of tumors.

The latest histological classification was adopted by the WHO in 1993 on the basis of modern immunohistochemical and immunocytochemical studies, which made it possible to take into account the peculiarities of the histogenesis of tumors and their cytoarchitectonics. According to this classification, all tumors are divided into 10 groups:

1. Tumors from neuroectodermal tissue a) astrocytoma; b) oligodendrogliomas; c) ependymomas; d) glioblastoma; e) medulloblastoma; f) angioreticulomas;
2. Tumors of cranial and spinal nerves.
3. Tumors of the brain (meningiomas).
4. Lymphomas and tumors of hematopoietic tissue.
5. Tumors from germ cells.
6. Cysts.
7. Tumors of the Turkish saddle area.
8. Tumors growing from surrounding tissues.
9. Metastatic tumors.
10. Unclassified tumors.

Tumors of neuroectodermal origin are the most common (50-60% of cases). Tumors of the membranes make up 20% of all brain tumors. Brain tumors According to the clinical classification, tumors are divided into extracerebral (meningiomas, neurinomas) and intracerebral (gliomas). In relation to the cerebellar tent, tumors are divided into supratentorial and subtentorial.

Characteristics of tumors.

1. Astrocytoma (15%) is a benign, slow-growing tumor, which is more often located in the frontal, temporal, parietal lobes of the brain or in its stem.

2. Oligodendroglioma (8%) is a benign, slow-growing intracerebral tumor that differs little from brain tissue and has petrifications that are clearly visible on a craniogram. It is localized in the hemispheres of the brain.

3. Ependymoma (3%) – grows in the area of the ventricles (near the foramen of Monroe, in the central canal of the spinal cord, in the IU ventricle).

4. Glioblastoma (15%) is a very malignant intracerebral tumor located in the temporal lobe, often growing through the corpus callosum into both hemispheres. It grows quickly, causes pronounced intoxication, metastasizes through the CSF.

5. Medulloblastoma (4%) is an extremely malignant tumor localized in the cerebellum, in the left ventricle or in the brain stem in children under 10 years of age. Quickly metastasizes through the CSF.

6. Angioreticulum (2%) – benign, slow-growing, contains cysts of various sizes, well separated from brain tissue. It is often localized in the cerebellum.

7. Meningioma (15%) is a benign tumor that grows from the membranes outside the brain tissue. There are basal and convexity meningiomas. Basal ones are located in the area of the Turkish saddle or in the area of the main bone, and convex ones are often located parasagittally.

8. Neurinoma (8%) is a benign tumor growing from the membranes of the spinal cord (more often 8 or 10 pairs).

9. Pinealoma is a benign tumor that grows in the brain stem or in the back of the ventricle.

10. Pituitary adenoma (10%) – grows from the adenohypophysis. It can be hormonally active or inactive. Hormonally active, it produces prolactin, growth hormone, and adenocorticotropic hormone, causing the corresponding clinic.

11. Craniopharyngioma is a tumor growing from the remnants of Rathke's pocket. It is congenital, benign, has many cysts, petrifications.

12. Metastatic tumors (8%) are most often metastases of lung, breast, kidney, stomach cancer. The main route of metastasis is hematogenous.

Diagnostics.

1. The patient's complaints depend on the localization of the tumor, its size, the effect on the ventricular system and brain matter. The main complaints are headaches, heaviness in the head, nausea, vomiting, that is, there are symptoms of intracranial hypertension. In addition, there may be complaints reflecting focal symptoms.

2. Anamnesis. The disease, as a rule, begins gradually, when the symptoms of CSF hypertension, or focal symptoms, increase over a long period of time. Sometimes the first manifestations of the disease appear acutely from hypertensive or focal symptoms.

3. Objective examination data Clinical manifestations of tumors are determined by the following factors: 1. direct action of the tumor on the brain tissue adjacent to it; 2. an increase in intracranial pressure due to an increase in the contents of the skull; 3. displacement of some areas of the brain in relation to others. There are 3 groups of clinical syndromes: 1. Hypertensive or cerebral syndrome. 2. Focal syndrome. 3. Dislocation syndrome. The clinic of the tumor can manifest either focal or general brain symptoms, which depends on its location and effect on the liquid circulation. If the tumor is located at a distance from the cerebrospinal tract, focal symptoms come to the fore in the clinic. When the tumor is localized near the cerebrospinal fluid, the first symptoms of hypertension syndrome appear. Hypertensive or general cerebral syndrome, which is caused by an increase in intracranial pressure, is manifested by the following symptoms: 1. a headache that disturbs the morning, has a distending character, increases with coughing, decreases after vomiting; 2. vomiting that occurs at the height of headaches, not related to eating; 3. congested optic nerve discs, which lead to a decrease in visual acuity and subsequent atrophy of the optic nerve discs; 4. changes in pulse and blood pressure are late symptoms of CSF hypertension; the pulse often slows down, blood pressure first increases, then decreases; 5. epinaives, which are a manifestation of hypertension syndrome, and when the tumor is localized in the cortex, it is the first manifestation of it; 6. changes in the psyche, manifested by apathy,

inhibition, disorientation; 7. damage to cranial nerves; 8. changes in the cerebrospinal fluid, which are manifested by an increase in its pressure and protein-cellular dissociation; 9. changes on the craniogram (osteoporosis of the back of the Turkish saddle, increased finger depressions and vascular pattern). Focal symptoms are caused by the direct effect of the tumor on the brain.

Tumors of the frontal lobe. Their psyche is characteristic (decreased attention, intelligence, criticism, depression, euphoria, disinhibition, aggressiveness). When the anterior central gyrus is affected, central monoparesis and paralysis occur, often epi-seizures of the type of Jacksonian motor attacks. Attacks that begin with turning the head and eyes in the opposite direction are possible. When the tumor is localized in the left hemisphere, motor aphasia and agraphia occur. Frontal lobe tumors are characterized by symptoms of oral automatism, grasping phenomena, hyposmia or anosmia (loss or reduction of smell more often in olfactory meningiomas. Frontal apraxia, ataxia, astasia, abasia are possible.

Tumors of the parietal lobe. With this localization, the sensitivity is mainly disturbed. Disorders of complex types of sensitivity appear (autotopagnosia, anosognosia, violation of the body scheme, astereognosia). When the posterior central gyrus is irritated and damaged, there are sensitive Jacksonian attacks with a feeling of rubbing or tingling in the hand or leg and loss of superficial cortical types of sensitivity. When deep sensitivity is disturbed, afferent paresis of the arm or leg occurs on the opposite side. When the tumor is located on the left side, there are disorders of counting, writing, and reading. Apraxia occurs with tumors of the supramarginal gyrus.

Tumors of the temporal lobe. Epinauses with auditory, olfactory, gustatory, less often visceral hallucinations, as well as sensory and amnestic aphasia, homonymous hemianopsia, pseudocerebellar ataxia, vestibulo-cortical dizziness, psychomotor automatisms are characteristic. Tumors of the occipital lobe are rare and are accompanied by simple visual hallucinations (photopsia), loss of opposite fields of vision (homonymous hemianopsia), color vision disorders, visual agnosia (mental blindness). Tumors of subcortical nodes. With this localization, hyperkinetic-hypothalamic syndrome, amyostatic or parkinsonism syndrome, as well as capsular syndrome (hemiplegia, hemianesthesia, hemianopsia) occur. The rapid development of hypertensive and dislocation syndromes associated with the close location to the cerebrospinal tract is characteristic of tumors of this location. Cerebellar tumors cause disturbances in its functions (muscular hypotonia, balance and coordination disorders, asynergy). Headache in the occipital region, forced position of the head, early development of hypertension syndrome are characteristic.

Brain stem tumors. These are gliomas (benign) and sarcomas (malignant) or metastases in the trunk. With trunk tumors, alternating syndromes occur (cranial nerve damage on the side of the process and hemiparesis of the opposite limbs). Respiratory and cardiovascular disorders are often associated with trunk tumors. Tumors of the bridge-cerebellar angle (neurinomas) of the VIIIth pair). Slow growth, early onset of focal symptoms, late development of hypertensive syndrome are characteristic of neuroma. The first symptoms of neuroma are tinnitus and progressive hearing loss, dizziness and nystagmus, later lesions of V, VI, VP and XIII pairs of cranial nerves and cerebellar symptoms appear. When the tumor grows to the back, the IX-XII pairs of cranial nerves are affected. X-rays reveal expansion of the internal auditory canal and osteoporosis of the top of the pyramid of the temporal bone. Bitemporal hemianopsia, visual impairment with optic disc atrophy, hormonally active adenomas cause endocrine disorders (acromegaly, Itsenko-Cushing syndrome, adiposogenital dystrophy), as well as metabolic disorders (carbohydrate, protein). X-ray of the sella turcica and computed tomography of the brain in pituitary adenoma. Symptoms at a distance (dislocation syndrome). This is a violation of the functions of the part of the brain that is located directly next to the tumor (these are the so-called symptoms in the neighborhood). With tumors, symptoms from those parts of the brain that are significantly distant from the tumor (remote focal symptoms) are frequent. Dislocation symptoms include wedging symptoms. The most common are: - temporal-tentorial - axial occipital wedge. These symptoms arise due to the fact that the pressure increases in the area of tumor growth and

the mass of brain tissue shifts to those areas where the pressure is lower. As a result, the parts of the brain closest to the natural openings begin to wedge into these openings, compressing those parts of the brain that are located in these openings (brain stem, corpus callosum). The wedge syndrome is accompanied by disorders of consciousness, breathing and cardiovascular activity, which leads to a fatal outcome. The appearance of such syndromes depends on the location of the tumor, its histological structure, and growth rate.

4. Data of additional examination methods

If a tumor is suspected, the patient should undergo the following tests:

1. Detailed neurological examination.
2. Ophthalmological examination (fundus, visual fields, visual acuity).
3. Otoneurological examination of hearing, vestibular function.
4. Craniography in two projections, which, if necessary, is supplemented with additional images with special layouts. On a craniogram, in addition to signs of intracranial hypertension (osteoporosis of the back of the Turkish saddle, increased finger impressions, increased vascular pattern, expansion or non-union of skull sutures, osteoporosis of the top of the pyramid of the temporal bone), local hyperostosis, local thinning of bones, bone defects, expansion of natural openings (optic canal nerve, internal auditory canal), calcified areas and tumors.
5. Echoencephalography (EchoEG) reveals the displacement of the middle structures in the opposite direction or the expansion of the ventricles with hemispheric localization of the tumor.
6. Electroencephalography (EEG) reveals a focus of pathological activity (slow waves, delta-theta activity) in the area of the tumor.
7. Computer and magnetic resonance imaging (CT and MRI) are the most informative methods that make it possible to precisely localize the tumor and determine its size. When using these methods, angiography, pneumoencephalography, and ventriculography became necessary only to clarify some details required for surgical intervention.
8. Lumbar puncture does not have a significant diagnostic role, if only because in conditions of sharply expressed intracranial hypertension it can cause wedge syndromes.

III. Differential diagnosis. Differential diagnosis is carried out with many organic diseases of the brain, namely: a) vascular diseases of the brain, b) brain abscess c) cerebral arachnoiditis d) multiple sclerosis e) benign intracranial hypertension. a) Vascular diseases of the brain, such as ischemic or hemorrhagic strokes, TIAs begin acutely against the background of cerebral atherosclerosis, hypertension, coronary heart disease with irregular rhythm, diabetes. In the anamnesis of such patients there are hypertensive crises, transient ischemic attacks. The neurological status is dominated by focal symptoms, the symptoms of intracranial hypertension are absent or slightly expressed. On CT or MRI, foci of reduced or increased density are detected in vascular pathology (depending on the nature of the stroke). The most difficult is the differential diagnosis with ischemic thrombotic stroke, when the symptoms grow slowly. In this case, only a detailed neurological analysis, observation of the dynamics of the disease, repeated examination help in making a diagnosis. b) Brain abscess. In the anamnesis of a patient with an abscess, there is a focus of infection (otitis, sinusitis, osteomyelitis). It is necessary to take into account the rapid development of an abscess within 2-3 weeks, the presence of membrane symptoms, symptoms of an infectious syndrome, inflammatory changes on the part of the blood. A decisive role is played by MRI, CT, which reveal a round volume surrounded by a capsule. c) Cerebral arachnoiditis. Risk factors and causes of the development of arachnoiditis are taken into account: acute infections such as influenza, tonsillitis, rhinosinusitis, otitis, traumatic brain injury, a history of meningitis or meningoencephalitis. The following clinical signs are important: gradual onset of asthenoneurotic syndrome with the addition of irritative symptoms (epinapadas) and hypertensive syndrome. The course of the disease is taken into account, when periods of improvement are replaced by exacerbations. CT scan or MRI make it possible to objectify the fibrous or atrophic process, the presence of cysts, the nature of hydrocephalus. d) Multiple sclerosis. The presence in the clinical picture of the disease of symptoms of a multifocal process with damage to different parts of the

brain, mostly remitting or remitting-progressive course, the presence of clinical dissociation phenomena, foci of demyelination in the brain and spinal cord on MRI is taken into account. e) Benign intracranial hypertension occurs in women with excess body weight, with hypo- and hyperthyroidism, Itsenko-Cushing syndrome, dysmenorrhea, etc. Hypertensive syndrome does not progress, but, on the contrary, tends to decrease.

IV. Complication of the disease. The main complications are dislocation and wedging of the brain, which is manifested mainly by trunk symptoms in the phase of clinical decompensation.

V. Tactics of treatment. Treatment of brain tumors can be surgical followed by radiation therapy and medication. A radical method of treatment is surgical removal of the tumor. Some tumors (stem, thalamus) are not amenable to radical surgical intervention, and therefore only partial removal of the tumor (basal meningiomas) or palliative surgery with the aim of establishing cerebrospinal fluid drainage is possible. For tumors with infiltrating growth or metastasis, only decompression trepanation is performed to reduce intracranial pressure. Almost all extracerebral tumors are removed without an incision in the brain. Pituitary tumors can be removed through the nose and main sinus, small neurinomas of the VIII pair - through the labyrinth of the temporal bone. Radiation therapy complements surgical treatment (if the operation was not radical) and is carried out when, according to histological examination, it is found that the tumor is sensitive to radiation therapy. Radiation therapy is carried out by radiotherapeutic (remote gamma therapy, X-ray therapy, proton beam irradiation - in tumors of the base of the skull, brain stem, metastases, medulloblastomas) or radiosurgery (when a radiopharmaceutical is injected into the tumor; for example, in pituitary adenomas, craniopharyngiomas). Medical treatment is mainly symptomatic and aimed at reducing intracranial pressure (dehydrating and diuretic drugs: Lasix, mannitol, hypothiazide, magnesium sulfate, albumin), headache (analgesics), elimination of convulsive syndrome (anticonvulsant drugs).

Tumors of the spinal cord

Tumors of the spinal cord include neoplasms growing from its parenchyma, vessels, roots and membranes. Tumors of the spinal cord occur 6 times less often than brain tumors, and are observed in patients mainly aged 20-60 years. Spinal tumors are divided into primary and secondary. The group of primary tumors includes neoplasms originating from the brain substance (intramedullary tumors) and those growing from the membranes of the brain, roots, vessels (extra-medullary tumors). Extramedullary tumors occur much more often (in 80% of cases of all spinal neoplasms) than intramedullary tumors. Extramedullary tumors can be both subdural and epidural. Most extramedullary tumors are subdural. Sometimes there are tumors, part of which is inside the dural sac, and part is outside the dura mater, these are subdural-epidural tumors, as well as epidural-extravertebral tumors. Extramedullary tumors of the spinal cord include: 1) meningiomas (arachnoidendothelioma) originating from meninges or their vessels (occurring in 51.7% of cases); 2) neurinomas developing from Schwann cells mainly of the posterior roots of the spinal cord (occurring in 48.3% of cases); 3) hemangiomas, vascular tumors consisting of cavernous cavities filled with blood; 4) lipomas, which usually exist together with spina bifida or other dysraphic signs. The last two types of tumors are relatively rare. Extramedullary tumors are located mainly in the thoracic region of the spinal cord and in the region of the horse's tail. Intramedullary neoplasms of the spinal cord are represented mainly by gliomas. The basis of their classification is a histogenetic feature. Secondary tumors of the spinal cord are metastatic or grow into the spinal canal from the adjacent abdominal and thoracic cavities. The source of metastases is mainly cancer of the lungs, mammary, thyroid and prostate glands. Primary and secondary (metastatic) vertebral tumors, as well as neoplasms located in the spinal canal, should be distinguished from spinal neoplasms: lymphogranulomas, nonspecific granulomas, tuberculomas, cholesteatomas, parasitic cysts, which can cause the clinic of compression of the spinal cord or its roots.

Clinic. In the clinical course of extramedullary tumors of the spinal cord, it is customary to distinguish 3 stages:

And - root pain;

II — Brown-Sécard syndrome;

III — complete compression of the spinal cord.

Initially, radicular pain and paresthesias appear, usually on one side. Depending on the localization of the tumor, it can be a syndrome of cervical, intercostal, lumbosacral neuralgia. The pain is often stronger when lying down than when standing and moving. Tumors located on the front or anterolateral surface of the spinal cord may not cause pain. Increasing, the tumor begins to squeeze the corresponding half of the spinal cord, which leads to the development of Braun-Sécar syndrome. In the future, symptoms of transverse damage to the spinal cord are revealed. The intensity of pain at this stage may decrease. During the percussion of the spine, tenderness is felt at the level of tumor placement (symptom of the spinous process). This sign is typical for epidural localization of the focus. Tumors in the area of the horse's tail are characterized by a slow course. The main clinical symptom for many years can be increasing pain in the area of the perineum, buttocks and lower limbs, which increases during coughing and sneezing, lying down and sitting (position syndrome).

Intramedullary tumors are manifested by a gradually progressive syndrome of damage to the gray matter — dissociated disturbances of sensitivity and flaccid segmental paresis. In the future, conductive symptoms are observed - pyramidal paresis and sensitivity disorders, the border of the latter falls from top to bottom. In the case of an extramedullary tumor, radiographs show pathological changes in the vertebral bodies (osteoporosis, compression, etc.), Ellsberg-Dyke syndrome (incorrect shape of the legs of the vertebral arch and asymmetry of the distance between them and the spinous processes). Contrast (descending and ascending) myelography is used to determine the level of spinal cord tumor location. The clinic of spinal cord tumors at the level of different segments has its own characteristics. For tumors of the upper cervical level (C, —C1V), pain in the neck and nape of the neck, neck muscle tension, incorrect head position, spastic tetraparesis, conductive sensitivity disorders are characteristic. Damage to the C1V segment is accompanied by paresis of the diaphragm, manifested by hiccups, shortness of breath, difficulty coughing and sneezing. If the tumor is localized at the level of the cervical thickening, atrophic paresis of the upper limbs is observed in combination with spastic paresis of the lower limbs. Radical sensitivity disorders and pain may occur in the hands. For the CV111 — Th lesion, Bernard — Horner syndrome (narrowing of the eye slits, miosis, enophthalmos) is characteristic. Disorders of the functions of the pelvic organs in the case of tumors of cervical localization are usually absent for a long time and have the character of imperative calls for automatic emptying of the bladder. Tumors of the thoracic region cause conductive disorders of sensitivity, lower spastic paraparesis, dysfunction of the pelvic organs. Hands remain intact. Radical pain has a girdling character, imitating diseases of internal organs. Segmental disorders can be manifested by loss of abdominal reflexes, which helps to determine the level of damage. In the case of tumors of the upper lumbar segments, spastic paresis of the lower limbs is observed in combination with atrophy in their proximal parts, radicular pain in the innervation zone of the femoral nerve. In the presence of tumors of the epiconus, radicular pain in the lumbar region, saddle-like hypoesthesia, flaccid paresis of the gluteal muscles of the back surface of the thigh, lower leg, and foot occur. Disorders of sphincter function appear early in the form of urinary and fecal incontinence. Tumors of the cerebral cone are characterized by early and pronounced dysfunction of the bladder, rectum, and genitals. Paralysis of the lower limbs is absent, tendon reflexes are preserved. In the perineum there are dissociated sensitivity disorders in the form of horseman's pants. Bedsores often appear in the lumbosacral region. Additional research methods. In the diagnosis of spinal cord tumors, the study of cerebrospinal fluid and conducting lycodynamic tests are of great importance. A spinal cord tumor is characterized by an increase in protein in the cerebrospinal fluid with normal cytosis. In the case of some tumors (neurinoma of the horse's tail, ependymoma of the terminal thread), a particularly high protein content and its involuntary precipitation in the test tube are observed. If reactive arachnoiditis develops near the tumor in the cerebrospinal fluid, a small pleocytosis (20 — 40 cells) may occur, which is also likely in the presence of subdural malignant neoplasms.

Often, the cerebrospinal fluid is xanthochromic due to hemolysis of erythrocytes from compressed veins of the spinal cord or vessels of the tumor itself. To detect partial or complete blockage of the subarachnoid space, fluid dynamics tests help: - artificially increasing the pressure of the cerebrospinal fluid above the tumor by compressing the vessels of the neck, tilting the head forward (Poussep's test), - pressing on the abdomen (Stukey's test). The degree and speed of pressure increase in the subarachnoid space is determined manometrically during a lumbar puncture. The absence or insufficient increase in pressure indicates a violation of the patency of the subarachnoid space. A complete block is also characterized by a rapid and sharp (to zero) decrease in cerebrospinal fluid pressure when a small amount of fluid is removed. During liquefaction tests, the symptom of Rozdolsky's liquefaction shock (increased pain in the affected area of the root) and conduction paresthesias may occur. After a lumbar puncture, wedging syndrome is often detected (a sharp increase in conduction disorders up to the development of complete transverse compression of the spinal cord), the basis of which is the increased pressure of the tumor, which has shifted in the distal direction, on those areas of the spinal cord that are located below. Cervical shock syndrome and wedging syndrome are also of great diagnostic value, as they are pathognomonic for tumors of the spinal cord, especially extramedullary localization. Intramedullary tumors can exist for a long time without significant changes in cerebrospinal fluid. To determine the block of the subarachnoid space and the level of the tumor, contrast myelography should be performed, if there is a suspicion of a tumor of the spinal cord, the examination of the patient must necessarily begin with an X-ray of the spine in order to rule out diseases that can lead to compression of the spinal cord, as well as in order to identify bone changes characteristic of spinal tumors. Often, radiological changes in the presence of metastases in the spine lag behind clinical manifestations, namely radicular pain, and are revealed only at a later stage. NMR is a valuable method for detecting the pathological process, which allows you to clearly determine the boundaries and localization of the tumor in the early stages.

Diagnosis and differential diagnosis. It is often necessary to make a differential diagnosis with: a) neurological manifestations of osteochondrosis of the spine, b) ischemic myelopathy, c) tuberculous spondylitis, d) spinal arachnoiditis, e) anomaly of the cranio-vertebral transition, e) multiple sclerosis. Neurological manifestations of osteochondrosis arise against the background of degenerative-dystrophic changes in the spine, pain syndrome and static-dynamic disorders often recur after physical exertion or hypothermia, gradually decrease or disappear after treatment. The course of the disease is long, periods of exacerbation alternate with remissions. CT and MRI data are diagnostically important, allowing to rule out a volumetric process. Ischemic myelopathy. It is especially difficult to differentiate cervical myelopathy from extramedullary tumors due to the similarity of the clinical picture, course, multifactorial pathogenesis, discogenic compression of the spinal cord, and the possibility of impaired spinal blood circulation due to a bulky process. MRI is used for diagnosis. Tuberculous spondylitis. It is manifested by spinal, reflex, radicular and radicular-spinal syndromes that occur as early or late complications of tuberculous spondylitis. These syndromes appear acutely or gradually at the same time as the spinal cord injury or many years after the onset of the disease. For differentiation, laboratory diagnostics of tuberculosis and the activity of the process, X-ray of the spine, CT or MRI are carried out, which make it possible to objectify changes in the bodies of the vertebrae, the configuration of the discs, determine the condition of the spinal cord, the nature of the deformation of the spine, etc. Spinal arachnoiditis. This is an autoimmune, diffuse or limited membranous-ligamentous process of traumatic or inflammatory etiology. It is clinically manifested by local or diffuse pain, compression spinal syndrome with moderate paresis and pelvic disorders. The course is relapsing. The diagnosis takes into account the causative factor, the results of a lumbar puncture, and a contrast examination of the subarachnoid space. MRI, which reveals the ligamentous process and deformation of the roots, subarachnoid cysts, is of decisive importance. Anomalies of the cranio-vertebral transition are clinically manifested by radicular, spinal-trunk, syringomyelitic syndromes that increase in intensity, periodically compensate or, on the contrary, worsen. Multiple sclerosis (mainly spinal or cerebro-spinal forms). Cerebral symptoms and a remitting course are important for diagnosis.

For the final diagnosis, an MRI examination of the brain and spinal cord is required to detect foci of demyelination. During topical diagnostics, the upper border of the tumor can be determined by the localization of radicular pain and sensory disturbances, symptoms of the spinous process and CSF shock, persistent conductive sensory disturbances. It is necessary to take into account that the level of hypoesthesia is usually lower than the tumor, firstly, due to the eccentric placement of conductors of superficial sensitivity in the spinal cord, secondly, because the fibers of superficial sensitivity, before passing into the spinothalamic path of the opposite side, pass 2 — 3 segments on their sides; in addition, there is an "overlap" of adjacent segments. Therefore, the upper border of the tumor is localized 2-3 segments higher than the border of anesthesia. The lower border of the tumor is much more difficult to determine. The reduction of tendon and axial reflexes, the arcs of which pass through the squeezed segments, as well as the level of protective reflexes, are important . Descending and ascending myelography carried out according to the indications allows to determine the upper and lower borders of the tumor. In the diagnosis of tumors, the determination of its primary or secondary nature is important for prognosis and treatment. The main clinical sign of metastases of malignant tumors in the spine is pain that does not disappear at rest and in any position of the patient, resistance to treatment. The blood picture often does not show characteristic signs, the hemoglobin content may remain high, and the ESR may be low. Neurological pain has the character of secondary radiculitis without gross disturbances of sensitivity and motor functions, in the extreme case until the moment of a compression fracture or compression of the spinal cord by a growing tumor. Metastases are usually localized in the spinal canal. The development of spinal symptoms often occurs against the background of previous severe pain. In the anamnesis, there may be indications for surgery for cancer, and in their absence, clinical and X-ray examinations contribute to the detection of the primary tumor. The diagnosis of other forms of spinal disease is usually made on the basis of X-ray and NMR data and is confirmed by appropriate laboratory tests. Secondary tumors are always malignant and within a year or even a few months can lead to a syndrome of complete transverse damage to the spinal cord. Secondary tumors are localized, as a rule, extradurally. In the diagnosis of secondary tumors of the spinal cord, a detailed history, a thorough examination of the internal organs, repeated clinical blood tests and especially X-ray of the spine are important.

Differential diagnosis of a spinal cord tumor depends on the stage of the process. Neurinomas and meningiomas in the radicular stage should be differentiated from diseases of internal organs (pleurisy, duodenal and gastric ulcers, cholecystitis, nephrolithiasis, etc.), as well as from radicular syndromes of osteochondrosis. Tumors of the spinal cord, which give the clinic of an increasing transverse lesion of the spinal cord, are differentiated from the spinal form of multiple sclerosis. Suspicion of acute myelitis or epiduritis usually arises if the tumor is complicated by necrosis as a result of impaired spinal blood circulation. A thorough study of the history (presence of infection), onset of the disease (prodromal period with general infectious symptoms, increased body temperature), examination of the cerebrospinal fluid is important for the correct diagnosis. It is quite difficult to differentiate intramedullary tumor from syringomyelia. It should be borne in mind the slower (years) increase in spinal symptoms in the case of syringomyelia, especially pronounced trophic disorders in the case of less significant lower spastic paraparesis and pelvic disorders, dysraphic status, absence of signs of spinal cord compression and changes in cerebrospinal fluid. 4/. Data of additional examination methods. The diagnosis of the tumor is based on the appearance in the clinic of symptoms of gradual compression of the diameter of the spinal cord. To confirm the diagnosis, carry out a lumbar puncture with examination of the cerebrospinal fluid to detect protein-cellular dissociation and blockade of the subarachnoid space; radiography of the spine (on radiographs, the expansion of the intervertebral opening, osteoporosis of the base of the vertebral arches and expansion of the spinal canal, destruction of the vertebral bodies are revealed). Myelography with contrast makes it possible to clarify the presence of a tumor and determine its level. The most informative are computerized spinal tomography and MRI of the spinal cord.

Complication of the disease. The most frequent complications are bedsores, urosepsis due to long-term pelvic disorders.

Tactics of treatment. The only effective method of treatment of spinal tumors is their surgical removal. Extramedullary tumors and some intramedullary tumors (ependymomas, astrocytomas) are radically removed. In the case of malignant tumors, after their removal, the following X-ray therapy or chemotherapy is carried out.

The examination of work capacity is carried out taking into account the histological nature of the tumor (benign, malignant), the surgical intervention performed and its results (complete or partial removal of the tumor), the presence of relapses, repeated operations, postoperative periods, the nature and severity of impaired functions (motor defect, coordination, visual disorders , epi-attacks, etc. Indications for referral to MSEC are: a) unfavorable clinical prognosis in non-operated patients, in particular with metastatic tumors, b) operated patients with severe or moderately severe functional impairment and limitation of working capacity, c) recurrence or continued tumor growth, complications after operations, d) paraneoplastic lesions of the nervous system in tumors not of brain localization, complications of radiation and drug therapy.

Dispensary. Secondary prevention of disability involves early tumor diagnosis, timely surgical and (or) radiation treatment, medical rehabilitation in the postoperative period, dispensary monitoring of operated patients, creation of optimal working conditions for working patients. Tertiary prevention involves early detection and treatment of patients with recurrence or prolonged tumor growth, rational employment of the disabled. VIII. Rehabilitation. The rehabilitation process is better for extracerebral tumors, as the recovery of functions is much faster, already within a year after the operation. Medical rehabilitation is carried out mainly in the rehabilitation department, in the polyclinic, in the sanatorium. The scope and methods of rehabilitation depend on the nature of the functional impairment. Drug therapy (nootropics, biostimulants), a complex of kinesiotherapy methods, classes with a speech therapist, plastic surgery of a postoperative skull defect, etc. are used. Professional and social rehabilitation is also carried out (teaching disabled people self-care skills, providing them with technical means, psychological correction).

Clinical protocol for providing medical care to patients with brain abscess.
Appendix to the order of the Ministry of Health No. 317 dated 06-13-2008

A brain abscess is a focal accumulation of pus in the brain substance, separated by a capsule or its elements. A brain abscess appears on a CT scan (MRI) as a predominantly round formation with clear contours, limited by a capsule, the visualization of which is enhanced by the administration of contrast agents.

Clinical manifestations of a brain abscess depend on its localization, size, number, spread of perifocal edema, degree of brain dislocation, intoxication-septic manifestations, and concomitant diseases.

The most typical for brain abscesses is the triad of symptoms: headache, focal neurological deficit, increased body temperature, which is observed in more than 50% of patients.

Headache of varying intensity is the most constant symptom and occurs in 80% of patients.

Focal neurological symptoms depend on the location of the abscess/abscesses, the spread of perifocal edema, and the presence and degree of brain dislocation. Focal symptoms have great topical and diagnostic value. In case of brain abscesses, the focal symptomatology more often has the character of shedding, but it can also have the character of irritation, especially in the initial stages of the development of the abscess.

Against the background of the development of intracranial hypertension and dislocation of the brain, consciousness is disturbed from stupor to coma, craniobasal and trunk symptoms, nausea, vomiting appear. With their appearance, primary focal symptoms are eliminated.

Meningeal symptoms are observed quite often. Their increase to an extreme degree may indicate the breakthrough of the abscess into the ventricular system.

An increase in body temperature is observed in 30-50% of patients. The temperature can be from subfebrile to hectic. In some patients, when a thick abscess capsule is formed, it may be normal.

In the diagnosis of brain abscesses, it is important to identify purulent-septic diseases of the internal organs and skeleton in the anamnesis; infectious diseases, including HIV-AIDS; penetrating TBI; inflammatory lesions of the soft tissues of the head and face, bones of the brain and facial skull; inflammatory processes of the paranasal sinuses, middle ear, pars petrosa of the temporal bone, surgical interventions performed on this occasion. The absence of the above-mentioned data in the anamnesis does not exclude the presence of a brain abscess.

The above indicates the absence of symptoms pathognomonic for brain abscesses.

Additional methods of radiation and laboratory diagnostics are important.

Radiography of the skull allows in some cases to detect calcification of the abscess capsule, the presence of free gas in patients who did not undergo surgical interventions and did not have a penetrating TBI. Among the indirect signs of abscess, attention should be paid to signs of osteomyelitis of the bones of the skull and inflammatory lesions of the paranasal sinuses, pars petrosa of the temporal bone.

ECHO-EG reveals a lateral dislocation in the supratentorial location of fairly large brain abscesses.

Brain CT scan is an informative method for diagnosing brain abscesses. CT allows to detect an abscess, its localization, size, number, assess perifocal edema, dislocation changes. Contrast-enhanced CT allows to more clearly visualize the capsule of the abscess. In some cases, it is difficult to differentiate an abscess from brain tumors, metastatic brain lesions, and some vascular malformations.

MRI of the brain, especially with contrast enhancement, is the most informative method for diagnosing brain abscesses, and CT is superior in this regard. MRI allows not only to detect an abscess, its localization, size and number, but also to differentiate it from encephalitis, tumors, metastases, and vascular malformations.

Lumbar cerebrospinal fluid examination with a formed abscess reveals slight pleocytosis and an increase in the level of proteins.

Lumbar puncture in suspected abscess is contraindicated in the presence of signs of intracranial hypertension.

General blood tests reveal changes characteristic of the inflammatory process - leukocyte pleocytosis, a shift of the formula to the left, an increase in ESR.

Bacteriological studies are carried out by taking a biopsy directly during surgical intervention from the abscess cavity. It is also advisable to carry out bacteriological studies in the presence of purulent extracerebral foci, which may be the cause of an abscess. These studies make it possible to determine the causative agent of the process, its sensitivity to antibiotics.

Conditions under which medical assistance should be provided

Patients with brain abscess are subject to examination and treatment in the neurosurgical department.

Diagnostics

Diagnostic measures include:

1. General examination, history.
2. Neurological examination.
3. X-ray of the skull in 2 projections.
4. X-ray of the lungs.
5. ECHO-EG.
6. CT of the brain, CT of the brain with contrast enhancement or MRI of the brain, MRI of the brain with contrast enhancement.
7. General analysis of blood and urine.
8. Biochemical examination of blood, coagulogram.
9. Blood group and RH factor.

10. Examination by an oculist.
11. Review of ENT.
12. Review of the therapist.
13. Bacteriological studies of extracerebral inflammatory foci (if available).
14. Bacteriological studies of abscess contents (after surgery).

Treatment

The presence of a brain abscess is an indication for hospitalization in a neurosurgical department. Treatment is carried out under the supervision of a neurosurgeon and an infectious disease specialist. In the vast majority of cases, the presence of a verified brain abscess is an indication for urgent surgical intervention.

Conservative treatment is carried out mainly:

- at the stage of abscessing encephalitis;
- with small sizes (up to 2.5 cm) foci with no dislocation changes, deep and periventricular localization;
- with multiple abscesses of deep localization without dislocation;
- with multi-chamber (cellular) abscesses of deep localization without mass effect.

Conservative treatment consists of antibacterial therapy with broad-spectrum antibiotics according to existing modern treatment schemes, anti-edema therapy. They use immunostimulating and resorbing agents. In the presence of accompanying meningitis, a study of cerebrospinal fluid with determination of individual sensitivity is indicated. When indicated, it is possible to administer antibacterial agents endolumbarly.

In the absence of an effect from conservative treatment for 4 weeks, deterioration of the patient's condition, increase in the volume of the focus or dislocation changes, the issue of surgical intervention is decided. The absolute indication for surgical intervention is the presence of an abscess accompanied by mass effect and dislocation changes.

Surgical treatment:

- puncture method - there are no contraindications. It is indicated for the severe condition of patients of various ages, especially in children, with a significant increase in intracranial pressure, with a deep periventricular location and localization in functionally important zones. The pus is evacuated with mandatory washing of the abscess cavity with a solution of antibiotics,
- the method of drainage of the abscess cavity is the most effective method of surgical treatment, used mainly for supratentorial localization in cases where the size of the abscess allows the installation of a drainage system. The use of inflow-outflow systems for irrigation of the abscess cavity with a solution of antibiotics and evacuation of pus is shown;
- total removal of the abscess is indicated in cases where the first two methods of intervention were ineffective, in the case of recurrence of the abscess, in contact abscesses, and in cases where the density of the abscess capsule does not allow a puncture and its effective drainage. With multi-chamber abscesses, surgical intervention can be carried out in two stages:

The first stage - puncture of the largest abscess - reduces its mass with subsequent removal of the abscess.

In case of multiple abscesses accompanied by dislocation syndrome, the intervention is carried out on the largest focus mainly by the puncture method.

During surgery, it is mandatory to carry out a bacteriological examination of the contents of the abscess with the determination of sensitivity to antibiotics.

Each surgical intervention is accompanied by the use of antibiotic therapy with the use of broad-spectrum antibiotics to determine individual sensitivity.

A contraindication to surgical intervention is the extremely serious condition of the patient with vital disorders.

It is unacceptable to puncture the brain through a purulent wound (purulent otitis media, frontitis, etc.).

When brain abscesses form in HIV-infected patients, the treatment should be complex with the use of specific anti-inflammatory and antiretroviral therapy.

Efficacy criteria and expected results of treatment

Improvement of general condition, regression of neurological symptoms.

With timely diagnosis and effective treatment, the overall mortality is 0-10%. In the absence of CT and MRI diagnostics, the mortality rate can increase to 40-60%. In the long-term period, neurological deficits are noted in 45% of patients, epileptic seizures in 27% of cases, motor disorders in 27%.

Test tasks for self-control.

1. At what level is the tumor of the spinal cord localized when there are scapular pains, conduction disorders of sensitivity, spastic paralysis of the lower limbs, urinary disorders of the central type?

A. Upper cervical segments
B. Segments C_5-C_8 I T_I
B. Thoracic department
G. Lumbar section
D. Sacral segments (Answer B)

2. In a 30-year-old patient with a brain tumor, during a lumbar puncture, the cerebrospinal fluid is transparent and flows out under pressure. Suddenly, the condition worsened, there was a severe headache in the occipital lobe, vomiting, dysarthria, rotator nystagmus, breathing and pulse disorder. Pathological foot reflexes are caused on both sides.

What causes the deterioration of the patient's condition?
A. hypertensive CSF syndrome
B. hemorrhage in the lateral ventricles of the brain
B. dislocation of the brain stem in the area of the brain legs
G. wedging of the cerebellar tonsils into the large occipital foramen
D. hemorrhage in the medulla oblongata (Answer D)

Problems for self-control with answers.

1. Patient D. Complains of pain in the sternal region of the spine, weakness of the right leg. The pain appeared 6 months ago, and 2 months later - weakness in the leg, which is getting worse. Objectively: ChMN without pathology. The average abdominal reflex is absent. On the right leg, the knee and Achilles reflexes are strengthened, the pathological reflexes of Babinski and Oppenheim are evoked. Muscle tone is strengthened, according to the pyramidal type. Pain and temperature hyperesthesia on the left from level D_9. Decreased articular-muscular sensation on the right leg. Soreness of paravertebral points on the right side at level D_{9-11}.

Make a diagnosis.

Answer. The patient suffers from a partial lesion of the spinal cord (Brown-Secard syndrome) due to the extramedullary growth of a tumor of the spinal cord at the level of D_{8-11}.

2. Patient K. Complaints of headache for four months, mainly in the morning, vomiting. Recently, the headache has increased significantly, especially when changing the position of the body. Objectively: Anosmia, congested optic nerve discs on the left, frontal ataxia, Yanishevsky's reflex. Reduction of criticism, memory. On the craniogram, there are signs of hypertensive CSF syndrome. EchoEG – displacement of midline structures (M-echo) from left to right by 4 mm. What is the patient's diagnosis?

Answer. The patient has a tumor of the basal surface of the left frontal lobe of the brain.

Recommended Books.

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

2. Topical diagnosis of nervous system pathology. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.

3. Methods of examination of a neurological patient: teaching. Guide / edited by L. I. Sokolova, T. I. Ilyash. K., 2015. – 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: training. 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. Tutorial. - K.: Medknyga Publishing House, 2019. - 48 p.

2. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 1 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2020. - 258 p.

3. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 778 p.

4. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 642 p.

Electronic information resources

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

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

ISW No. 4

Topic: Parasitic diseases of the nervous system, prion infections.

Relevance of the topic: Parasitic diseases of the nervous system have a mechanical and toxic effect on brain tissue, which causes degenerative changes in nerve elements and reactive inflammatory changes in blood vessels, membranes and glia. Clinically, these diseases resemble brain tumors, meningitis, etc. Therefore, knowledge of this pathology, the ability to prescribe appropriate examinations for the patient help in making a diagnosis and carrying out the correct therapy. Toxoplasmosis is a parasitic disease caused by protozoa. This damages the nervous and lymphatic system, eyes, skeletal muscles, myocardium, etc. Toxoplasmosis in humans occurs everywhere, on all continents and in all climate-geographical zones. The prevalence of the disease, as a rule, is higher in territories with a hot climate, as well as among the rural population. The great polymorphism of the clinical manifestations of the disease, its extreme prevalence, require good knowledge and skills from doctors of any specialty in order to carry out timely diagnosis and treatment.

Specific goals:
should: know:

1. What is a cysticerc.
2. What conditions are necessary for the development of cysticercosis?
3. Morphology and pathomorphology of cysticercosis.
4. Clinical symptoms of cysticercosis.
5. Differential diagnosis of cysticercosis.
6. Morphological and physiological data on echinococcosis.
7. Ways of spreading echinococcosis.
8. Clinical symptoms of echinococcosis.
9. Treatment of echinococcosis.
10. Prognosis in case of echinococcosis.

be able:
Diagnose cysticercosis and echinococcosis.
Carry out differential diagnosis between cysticercosis, echinococcosis and brain tumors.
Assign an examination plan.
Prescribe treatment to patients.

Topic content:

Echinococcosis is a parasitic disease that rarely affects the nervous system. Infection occurs when echinococcus eggs - the larval stage of a small tapeworm (Taenia echinococcus) - live in the intestines of dogs and wolves, enter the human gastrointestinal tract. Pathomorphology. There are two main forms of echinococcus - solitary and racemous. In the first case, there are single cysts, often reaching very large sizes (up to 5-6 cm in diameter or more), with racemose forms of echinococci in the brain and spinal cord tissues located in clusters of bubbles, around which there are pronounced reactive changes. A connective tissue capsule is formed around the echinococcus, surrounded by a shaft of inflammatory brain tissue, foci of softening and hemorrhages appear. Inflammatory changes are also present in the membranes in the area of the bladder.

Clinical manifestations. The clinical picture of echinococcosis of the brain consists of a hypertensive syndrome and focal symptoms that resemble the manifestations of a brain tumor.

Hypertensive syndrome is headaches, dizziness, vomiting, congestive discs of the optic nerves, general epileptic seizures. The nature of focal symptoms depends on the localization of the parasite. Most often, cortical epileptic seizures with subsequent development of paresis occur in those limbs in which there were convulsions. Characteristic mental disorders are dementia, delirium, depression. Eosinophilia is usually found in the blood. In the cerebrospinal fluid, a small pleocytosis with the presence of eosinophils and a slight increase in the level of protein, sometimes - separate parts of the bladder, succinic acid, but pleocytosis in this fluid may be absent. Course. It is steadily progressing, with an increase in focal symptoms and an increase in intracranial pressure. Often, remissions for 1-2 years can be observed in the course of the disease with multichamber echinococcosis. Diagnostics. It is extremely difficult to recognize echinococcosis. A brain tumor is usually diagnosed, which is verified during surgery or at autopsy.

The presence of echinococcosis of internal organs (most often the liver), some anamnestic data, profession (constant contact with animals), eosinophilia in the blood, Katzoni skin tests and Gedin-Weinberg RSC help to make the correct diagnosis. Comprehensive diagnostic data can be obtained with the help of computer and magnetic resonance imaging. Treatment. With a single echinococcus, its removal is indicated. When removing echinococcal cysts, extreme care must be taken not to damage the thin capsule of the parasite, otherwise dissemination of the process is possible. Multichamber echinococcus is not amenable to surgical treatment, and the prognosis in such cases is unfavorable.

Cysticercosis is a parasitic disease that occurs when eggs of the pig tapeworm (Taenia solium) enter the human gastrointestinal tract. The most common localization of cysticercosis in humans is the brain, eyes, and muscles. The lifespan of the parasite in the brain is from 5 to 30 years. Pathomorphology. A cysticercus is a pea- to walnut-sized cyst filled with clear fluid. On the

inner surface of the bubble is the head of the fin - scolex with hooks and suckers. In most cases, there are hundreds and thousands of parasites in the brain, but there are also single cysticerci. Cysticerci are localized in the soft meninges at the base of the brain, in the superficial parts of the cortex, in the cavity of the ventricles, where they can swim freely. Dying, the parasite calcifies, however, remaining in the brain, supports the chronic inflammatory process. Pathogenesis. Cysticercus has a toxic effect on the central nervous system, causes reactive inflammation of the surrounding brain tissue and membranes. Cysticercosis is accompanied by cerebral edema, hydrocephalus due to increased secretion of cerebrospinal fluid by the choroid plexuses, mechanical obstruction of the liquid circulation, and reactive arachnoiditis. Clinical manifestations. Due to the small size of the bubbles and their low density, in the clinical picture of cysticercosis, symptoms of irritation are mainly observed, and signs of shedding are absent for a long time or expressed very weakly. Thus, patients may have shallow paresis, minor sensitivity disorders, mild aphatic disorders. Symptoms of irritation are manifested by attacks that proceed according to the type of local Jacksonian and general convulsive epileptic seizures. In severe cases, status epilepticus may occur. A characteristic polymorphism of Jacksonian seizures, which indicates the multiplicity of foci in the cerebral cortex. Various changes in the psyche are typical for cysticercosis. They are expressed in a neurotic syndrome, as well as in more severe conditions: disorders, depression, hallucinatory delusional phenomena, Korsakoff's syndrome. Intracranial hypertension and cerebral edema cause paroxysmal, intense headache, dizziness, vomiting, congested optic nerve discs. When a cysticercus is localized in the IV ventricle, Bruns' syndrome may occur, which consists in paroxysmal headache, vomiting, forced head position, breathing disorders; cardiac activity, sometimes loss of consciousness. The basis of the syndrome lies in the obstruction of the outflow of cerebrospinal fluid from the IV ventricle and irritation of the bottom of the IV ventricle by cysticerci. Cysticercosis of the lateral ventricles proceeds according to the type of tumor of frontal or callous localization with periodic loss of consciousness due to blockage of the interventricular (Monroe) opening. Cysticercosis of the base of the brain (usually racemose, in the form of a bunch of grapes) gives a picture of basal meningitis with headache, vomiting, bradycardia, damage to the optic nerves and paralysis of the VI and VII pairs of cranial nerves. Cysticercosis can be the cause of severe damage to the spinal cord. Course. Long-lasting, remitting, with sharply defined periods of deterioration and light intervals for several months and even years. Spontaneous healing is not observed. Examination of the cerebrospinal fluid reveals lymphocytic and eosinophilic cytosis, sometimes an increase in the level of protein (from 0.5 to 2 g / l), in some cases - scolex and fragments of the cysticercus capsule. Lumbar puncture should be done carefully, because with cysticercosis of the IV ventricle, taking cerebrospinal fluid can cause sudden death of the patient. Eosinophilia is often noted in the blood. RSC of blood and especially cerebrospinal fluid with the use of cysticercosis antigen has diagnostic value. Lange's reaction is paralytic. X-rays of the skull sometimes reveal scattered small formations with dense contours - calcification of cysticerci, which can also be in the muscles of the limbs, neck, and chest. Sometimes the cysticerc is found on the fundus. As a manifestation of intracranial hypertension, stagnant discs of the optic nerves are noted on the fundus. Diagnosis and differential diagnosis. It is extremely difficult to diagnose cysticercosis of the brain in the absence of pathognomonic symptoms. The diagnosis is based on the following features of the disease: the multiplicity of symptoms indicating a multifocal brain lesion, the prevalence of irritation phenomena, the presence of signs of increased intracranial pressure, changes in the patient's severe condition with periods of well-being.

X-ray data, eosinophilia in the blood and in the cerebrospinal fluid, positive RSC with cysticercosis antigen help the diagnosis. Cysticercosis should be differentiated from a brain tumor, which is excluded on the basis of variability, multifocal symptoms, and long remissions. In the differential diagnosis with syphilis, meningoencephalitis, epilepsy, the presence of foci of calcification on radiographs of the skull and soft tissues, lymphocytic and eosinophilic cytosis in the cerebrospinal fluid, and specific RSC are important. Computer and magnetic resonance imaging are of decisive importance in the recognition of cysticercosis, which allows to detect both

the cysticerci themselves and accompanying changes. Surgical treatment. Indications for the removal of cysticerci arise in those cases when they are located in the ventricles of the brain and can be the cause of acute occlusion of the cerebrospinal fluid. With the localization of cysticercs in other parts of the brain, indications for surgery arise less often due to the dissemination of the process and the severity of inflammatory changes. With the development of occlusive hydrocephalus, which often complicates cysticercosis of the brain, there may be a need for shunting operations (ventriculoperitoneostomy, ventriculoatriostomy). Prevention. It is necessary to observe the rules of personal hygiene, appropriate processing of food products, vegetables, fruits, sanitary supervision of pig carcasses at slaughterhouses. Forecast. With multiple cysticercosis and cysticercosis of the IV ventricle, the prognosis is always serious. Death can occur during status epilepticus or acute occlusive hydrocephalus. Regarding working capacity, the prognosis is also unfavorable due to increased intracranial pressure, which is accompanied by persistent headaches, frequent epileptic seizures, and changes in the psyche.

Toxoplasmosis is a parasitic disease characterized by a chronic course, damage to the nervous system, muscles, myocardium and eyes, lymphadenopathy, enlargement of the liver and spleen, and the possibility of intrauterine damage to the fetus and newborn.

Etiology of toxoplasmosis. The causative agent is Toxoplasma gondii, which belongs to the most protozoan. In the organism of the intermediate host, the parasite exists in the stage of trophozoites, which reproduce intracellularly; in the chronic and latent course of the disease, they turn into cysts that persist for decades. Proliferative forms are adversely affected by various antiparasitic drugs, drying, heating, disinfectants; neither antibodies nor drugs penetrate through the dense shell of the cyst.

Epidemiology of toxoplasmosis. The disease belongs to zoonoses. The final host is a cat and some other representatives of the feline family, in the epithelium of the small intestine of which the parasite undergoes the sexual development cycle. Cysts that are excreted with feces can persist for up to a year and infect various species of animals and humans. Infection from other animals occurs only when eating raw meat. A sick person does not release the pathogen into the environment and does not pose a danger to others. Intrauterine infection of the fetus from the mother is possible during her infection during pregnancy, when there are proliferative forms of the parasite in the body; with infection on the eve of pregnancy, as well as in the presence of latent or chronic inactive toxoplasmosis in the mother, infection of the fetus does not occur. Toxoplasma infection is widespread (up to 30% of the population), but mostly toxoplasmosis is latent. One case of congenital toxoplasmosis occurs in 1000-3500 newborns (in 0.2-0.5% of infected women).

Pathogenesis of toxoplasmosis. Rooting of the pathogen takes place in the lower parts of the small intestine, from where it enters the regional lymph nodes, blood and is fixed in various organs and tissues. Cysts are formed here, which is sometimes accompanied by the appearance of inflammatory foci and even areas of focal necrosis, where calcifications are then formed. As a result of the vital activity of the parasite, the patient's organism becomes allergic, and as a result, antibodies are produced that protect it from new infection. When the body weakens, the immune system decreases, exacerbations and relapses of the disease are possible. Infection in the early stages of pregnancy can lead to the death of the fetus or the development of severe congenital pathology.

Toxoplasmosis clinic.

The incubation period with laboratory infection lasts about 2 weeks, with natural infection many months may pass before the first clinical signs appear. Acquired and congenital toxoplasmosis are distinguished; according to clinical manifestations - acute, chronic and latent forms. Acute acquired toxoplasmosis develops very rarely (0.2-0.3%) and occurs in the form of encephalitis, typhoid and mixed forms. In the clinical picture, the symptoms of damage to the nervous system (encephalitis, meningoencephalitis, encephalomysitis) come to the fore - high fever, severe headache, convulsions, vomiting, hallucinations, mono- and hemiplegia, paralysis of cranial nerves, meningeal signs. The course of the disease is severe, sometimes with a fatal outcome. Convalescents may have persistent residual effects on the part of the central nervous

system. Maculo-papular rash and hepatosplenomegaly are also observed in the typhoid-like form. In most patients, toxoplasmosis has a chronic course - both from the very beginning (primary-chronic) and after the transferred acute form (secondary-chronic). The disease begins gradually with symptoms of general intoxication - decreased appetite, sleep disorders, weakness, weight loss, irritability, often headaches. Permanent signs are subfebrile that lasts for months, generalized lymphadenopathy, hepatosplenomegaly. Patients often complain of pain in the muscles and joints, in the area of the heart, and a feeling of increased heartbeat. Tachycardia, heart rhythm disorders, expansion of heart boundaries, corresponding ECG changes, abdominal pain, symptoms of mesadenitis, intestinal dyskinesia are noted. Characteristic lesions of the eyes (chorioretinitis, uveitis), nervous system (calcifications in the brain, epileptiform seizures, functional disorders). Secondary chronic toxoplasmosis is distinguished by the presence of gross residual phenomena on the part of the central nervous system. During the latent course, no clinical symptoms can be detected. Congenital toxoplasmosis, as well as acquired, can take the form of an acute or chronic disease. He is characterized by a combination of encephalitis with microphthalmia and congenital cataract. The acute form progresses and can end in the child's death in the first weeks of life. The secondary-chronic form is characterized by general infectious symptoms that periodically worsen, as well as signs of damage to the nervous system and eyes, retardation in mental development; various birth defects, Down syndrome are possible.

Diagnosis of toxoplasmosis.

Diagnosis is based on clinical data, as well as the results of special studies (ECG, fundus examination, radiography of the skull and affected muscles). Unconditional confirmation of the diagnosis of toxoplasmosis is the isolation of the parasite (in the acute form - from cerebrospinal fluid and blood, in stillborns - from tissues using a biological sample and histological examination). Among other methods, serological ones are used - RZK, RNGA, the fluorescence method, as well as the reaction with Seibin-Feldman dye, intradermal allergy test with toxoplasmin. Positive tests only indicate infection, but negative tests, especially a negative allergy test, make it possible to reliably rule out chronic toxoplasmosis. To diagnose congenital toxoplasmosis in a child, the mother must be examined. It is necessary to differentiate toxoplasmosis from chronic tonsillitis, cholecystocholangitis, mesadenitis, appendicitis, chronic adnexitis, arachnoiditis, chorioretinitis, rheumatism, tuberculosis.

Treatment of toxoplasmosis.

In acute toxoplasmosis, the main means of treatment are chemotherapeutic drugs - the combination of chloridine (0.025 g 2-3 times a day), delagil (0.5 g 2 times) or aminoquinol (0.15 g 3 times) with sulfonamide drugs is most often used within 7-10 days. Antibiotics of the tetracycline group are also effective. In case of a severe course of the disease, glucocorticoids are prescribed (in case of meningoencephalitis - mandatory), dehydration therapy. In chronic forms of toxoplasmosis, etiotropic drugs are ineffective. For their treatment, desensitizing and immunostimulating agents, toxoplasmin according to the scheme are used, correction of impaired body functions is carried out. It is necessary to treat concomitant diseases that contribute to the emergence of chronic forms and exacerbations. Physiotherapy and exercise therapy are used. In the case of eye damage, local procedures are recommended. Given the teratogenic effect of chemotherapeutic drugs, their use in the first 3 months of pregnancy is prohibited. If necessary, treatment is limited to a course of vaccine therapy and tonics.

Prevention of toxoplasmosis

Fighting toxoplasmosis of domestic animals. In order to prevent congenital toxoplasmosis, it is important to prevent infection during pregnancy (avoid contact with cats, do not try raw minced meat, etc.). Clinical and serological examination of pregnant women with a complicated obstetric history is carried out.

Prion-related diseases are a group of neurodegenerative diseases of humans and animals with the formation of spongiform encephalopathy, which belong to the group of slow infections and are characterized damage to the central nervous system (CNS), muscular, lymphoid and other systems, always end fatally.

The causative agent of these diseases are prions — an exclusive class of protein-based infectious agents that do not contain nucleic acids in their structure and thus differ from other causative agents of infectious diseases. To date, it has been established that in addition to short-lived proteins, which the body uses as an energy resource and as a building material for new cells, there are also proteins that have completely different, and still largely unexplained, functions. It is with them that the emergence of slow prion infections is associated. Based on the fact that these diseases are infectious in nature, and the infectious agent is transmitted from the source to a susceptible organism, WHO experts use the concept of causative agent in relation to prions.

Currently, prion diseases include:

in animals, scrapie, bovine spongiform encephalopathy (mad cow disease), chronic wasting disease of wild ungulates in captivity, etc.

In humans, prion diseases include kuru, Creutzfeldt-Jakob disease, Gerstmann-Streussler-Sheinker syndrome, and fatal familial insomnia.

There is an assumption that pathogenic prions are related to the causation of some other human diseases: chronic progressive encephalopathy of childhood/Alpers disease, spongiform myositis with prion-associated inclusions. With them, amyloid changes and accumulation of normal prions are found in the tissues, although no pathogenic prions have been found so far.

Актуальність проблеми пріонових хвороб зумовлена тим, що вони надзвичайно небезпечні для життя людей і тварин. Ці недуги виявляються у всіх країнах світу, а збудники скрепі та коров'ячого сказу з Великобританії завезені в ряд країн Європи, Близького Сходу, Азії. Сучасні карантинні заходи для ліквідації епізоотії трудомісткі, завдають величезних економічних збитків сільському господарству, а також мають негативний вплив на соціальну ситуацію. Збудниками пріонових хвороб є патологічні ізоформи пріонів і змінені білкові молекули хазяїна, які не мають нуклеїнової кислоти, але характеризуються патогенними властивостями. Пріони складаються тільки зі змінених (конформаційних) білкових молекул. Відсутність у складі пріонів нуклеїнових кислот визначає незвичність деяких з властивостей. Вони дуже стійкі у довкіллі — з усього живого пріони гинуть останніми: витримують кип'ятіння протягом 30-60 хвилин, висушування, заморожування,ультрафіолетове і гамма-опромінення, хімічну обробку спиртами, формальдегідами, кислотами, не піддаються гідролізу ферментами. Ген, який кодує пріоновий білок, міститься не у складі пріону, а в клітині. Пріоновий білок, потрапляючи в організм, активує цей ген PrNP і зумовлює індукцію синтезу аналогічного білка. Разом з цим пріони при всій своїй структурній і біологічній своєрідності мають ряд властивостей звичайних вірусів (віріонів). Вони проходять крізь бактерійні фільтри, не розмножуються на штучних живильних середовищах, репродукуються до концентрацій 105−1011 на 1 г мозкової тканини, адаптуються до нового хазяїна, змінюють патогенність і вірулентність, відтворюють феномен інтерференції, характеризуються відмінностями, здатністю до персистенції в культурі клітин, отриманих з органів зараженого організму, можуть бути клоновані.

Epidemiology. The source and reservoir of infection In most patients, the pathological prion PrPsc appears spontaneously — by mutation of its own PrPc. In other cases, exogenous infection is possible from animals that are the main source of the pathogen (sheep, goats, cows, deer and other ungulates, minks, cats, and possibly other species). Only in the case of kuru, the source of the causative agent in nature is considered to be a person —□a patient or one who is in the incubation period of the disease

Mechanism and factors of transmission.

Prion diseases are inherited according to the autosomal dominant type (although this is an indirect process - due to the previous genetic autoreplication of the infectious agent). A sick person in everyday conditions does not pose a danger to others. However, the concentration of prions in her organs during the incubation period is significant, and this creates a danger of infection of recipients from infected, but not yet sick donors of blood or internal organs, by an iatrogenic route (intracerebral through transplantation of the cornea, fragments of the dura mater, implantation of

intracranial electrodes, intravenously, intraperitoneally, through the skin, during endoscopy, etc.). The highest concentration of the pathogen is in the brain tissue, but it is found in significant titers in the tissues of other organs, in the blood. A sick person at home or in a hospital does not pose a danger to others, if they do not come into contact with the tissues, especially the brain, of the affected person. Considering the fact that the concentration of prions in the organs during the incubation period is significant, and the period itself is long, a person can become a source of the pathogen during blood transfusions, during transplantation of organs taken from infected, but not yet sick, patients. Animals become infected either due to spontaneous mutations or as a result of eating infected animals.

Mechanisms of pathogen transmission are diverse and include alimentary transmission in nature, hemocontact and aerosol transmission are also hypothetically possible, vertical transmission through the placenta is also possible. A special epidemic danger is this form of the course of slow infections (for example, with scrapie, mad cow disease, etc.), in which latent virus-carrying and typical morphological changes in the body are asymptomatic. Transmission of prions is possible in case of consumption of meat of butchered sick animals or, more often, those that were in the incubation period, when they do not have any symptoms of the disease; and also during the autopsy of dead animals. Given the high heat resistance of prions, ordinary thermal cooking of meat does not neutralize the causative agent of the disease. Infection can also occur with parenteral administration of hormones made from the organs of infected animals (pituitrin) or humans (gonadotropin). Employees of slaughterhouses and meat processing plants are also exposed to the risk of infection during the slaughter of animals in the incubation period and contact with their tissues and organs. The specifics of the epidemic process among consumers of meat from infected animals have not been sufficiently studied, and the very fact of human infection from cows was proven only in the early 90s of the 20th century.

Susceptible contingent and immunity Recipients of transplants of internal organs, those who undergo various medical manipulations, in which devices or mechanisms may be contaminated with tissues of infected donors, recipients of certain medical preparations containing hormones, veterinary and medical surgeons, pathologists, veterinarians are considered especially vulnerable , meat processing industry workers, etc. Immunity is not produced as a result of the disease, prion diseases always end in death.

The general pathogenetic basis of slow infections is the accumulation of the pathogen in various organs and tissues of the infected organism long before the first clinical manifestations and long, sometimes multi-year, persistence in those organs in which pathogistological changes are not detected for a long time. Subsequently, the proliferation of various cells occurs. For example, spongiform encephalopathies are characterized by pronounced gliosis, pathological proliferation and hypertrophy of astrocytes, which causes vacuolization and death of neurons, that is, the development of a spongiform state of brain tissue. The incubation period for all prion diseases is very long — from several months to decades.

Pathomorphological changes.

These changes in prion infections can be divided into a number of characteristic processes, among which degenerative changes in the central nervous system should be mentioned first of all. Thus, being non-antigenic, prions do not cause inflammatory, but degenerative processes. A change in the size and/or shape of the constitutive proteins — normal cellular prion proteins — PrPc leads to their transformation from vitally necessary to deadly dangerous — PrPsc. This phenomenon is proposed to be called "conformational diseases". There are reasons to believe that such conformational proteins can play the role of the main regulators in the body, including such an important process as limiting the duration of life itself. After entering the cell, PrPsc becomes a matrix for the transformation (conversion) of other PrPc molecules into PrPsc, activation of the PrNP gene. It has been proven that in the brain of patients it is associated with lipoproteins of low and very low density. Accumulation of PrPsc in cells of the CNS leads to their irreversible degeneration and death, due to which prion diseases are absolutely fatal.

Clinical manifestations. There are three classic forms of prion spongiform encephalopathies: sporadic (the majority of all cases), familial or hereditary (about 10-15%), iatrogenic (the percentage of such cases has not yet been definitively established).

Accordingly, in humans, the following prion diseases are distinguished by the mechanism of development:

sporadic (sporadic and amyotrophic forms of Creutzfeldt-Jakob disease),

hereditary (hereditary form of Creutzfeldt-Jakob disease, Gerstmann-Streussler-Sheinker syndrome, fatal familial insomnia)

acquired (chicken, iatrogenic form and a new variant of Creutzfeldt-Jakob disease).

In most cases, slow infections occur and develop without a temperature reaction of the body. All subacute transmissible spongiform encephalopathies are manifested by impaired gait and movement coordination. Often these symptoms are the earliest, later they are joined by hemiparesis and paralysis. All forms of prion encephalopathy are manifested by various neurological and mental symptoms. First of all, it is:

disorders of the sensitive sphere (amnesia of various degrees, loss and distortion of sensitivity, loss of functions of the senses);

damage to the motor sphere (ataxia, adynamia, muscle atrophy, paresis and paralysis);

mental disorders (loss of professional skills, depression, drowsiness, aggressiveness, reduced intelligence up to complete dementia).

For the most part, with all prion diseases, patients die from exhaustion or the addition of pneumonia.

You can find more detailed information on this topic in the article Measles (disease).

You can find more detailed information on this topic in the article Creutzfeldt-Jakob disease.

You can find more detailed information on this topic in the article Gerstmann-Streussler-Scheinker syndrome.

You can find more detailed information on this topic in the article fatal family insomnia.

Diagnostics

Changes in the cerebral cortex in a person who died from a variant of Creutzfeldt-Jakob disease. Large ornate plaques are visible. Biopsy smear, staining with hematoxylin-eosin.

Routine paraclinical methods are not very informative. MRI of the central nervous system provides quite serious diagnostic support. It shows hyperintense signals in the cerebral cortex, basal ganglia and thalamus. The described characteristic changes in the form of a "hockey stick" — an increase in signal intensity in the shell and the center of the caudate body resemble this object. The symptom of "honeycombs" is also characteristic - corresponding to the pattern of an increase in the intensity of the signal in the thalamus.

One of the rather sensitive and specific express methods for diagnosing prion diseases is a biopsy of a lymph node and staining the material with Congo red in order to detect amyloidosis. In the presence of amyloidosis, monkeys or mice can be experimentally infected with a suspension prepared from the brain or lymph nodes taken from dead people or animals. From the 4th to the 14th week of the disease, prions can be detected in the spinal cord, brain, and other organs. A prion can be detected in a pharyngeal tonsil biopsy, as well as in a brain biopsy with immunohistochemical typing of proteins. Histopathological examination of the CNS reveals spongiosis with vacuolization of neurons, proliferation of astrocytes and glia without signs of inflammation and demyelination, and amyloid prion protein plaques. In the case of prion-associated myositis with inclusions, necrotic myopathy is histologically revealed with the presence of vacuoles, which in frozen sections contain spiral-like congophilic threads.

To establish an accurate diagnosis of a human prion disease, one of four additional criteria must be identified:

1. the presence of specific amyloid plaques caused by prions (Pr-amyloid plaques);
2. the tissue's ability to be infected with spongiform encephalopathy of animals;
3. the presence of Creutzfeldt-Jakob prion protein isoforms;

4. the presence of a pathogenic mutated PrNP gene.

A protein is detected in the cerebrospinal fluid of patients, which can serve as a diagnostic test for spongiform encephalopathy in humans and animals. The test with streptomycin, which detects the prion in various tissues, is promising.

Treatment

Effective etiotropic and pathogenetic therapy of prion diseases has not been developed. In the early stages, symptomatic agents are used to correct behavior disorders, sleep disorders and myoclonus (amphetamines, barbiturates, antidepressants, benzodiazepines, other neuroleptics); in the late ones — supportive therapy.

It is assumed that drugs that bind and stabilize the structure of the cellular isoform of the prion can reduce the expression of prions and delay the onset of the disease. A search is underway for chemical agents that would destabilize the structure of the pathological isoform of the prion. Chemical effects on endocytosis, exocytosis, intracellular transport and specific destruction of prions may also be effective. However, until now, the prognosis for prion infections is always unfavorable.

Prevention.

There are no specific means of prevention. When working with patients in the process of invasive procedures, as well as when in contact with their biological fluids, it is necessary to follow the rules provided for when working with a pathogen of a particularly dangerous degree (as with HIV infection), using special protective clothing (gloves with a metal layer, a mask, protective glasses, apron). This also applies to persons who are in contact with potentially possible sources of infectious prions and belong to risk groups (veterinary and medical surgeons, pathologists, veterinarians, workers in the meat processing industry, etc.). The corpses of those who died from prion diseases must be cremated.

The three most effective methods of decontamination of prion-contaminated medical instruments are:

1. autoclaving at 134-138 C for 18 minutes (in case of incomplete loading) or at 132 C for 60 minutes. (in the case of using a horizontal autoclave);
2. treatment with sodium hydroxide (preferring a concentration of 1 mol/l and an exposure of 60 min. at 20 C);
3. treatment with sodium hypochlorite (preferably with a solution containing 2.5% active chlorine for 60 minutes at 20 C).

In the future, the quality control of the carried out decontamination is carried out. There is also a restriction on the use of medicinal products made from cow tissue, the production of pituitary hormone preparations of animal and human origin has been suspended, the requirements for their certification have been strengthened, preference is given to genetically engineered drugs. Restrictions on transplantation of the dura mater and cornea have been introduced. Transplantation of tissues, blood transfusions, and prescribing of blood products from persons with undiagnosed dementia are prohibited.

1. What are the symptoms of cysticercosis?

*A) hypertensive syndrome
*B) focal symptoms
C) temperature increase
D) unchanged blood
*D) remitting course of the disease

2. Clinical signs of hypertension syndrome?
3.
*A) headache
*B) vomiting

*B) congestive discs of the optic nerves
D) loss of reflexes
D) sensitive ataxia

4. Treatment of cysticercosis
*A) surgical
B) conservative

4. What is echinococcus?
*A) single chamber
*B) multi-chambered
*B) alveolar

5. What changes occur in peripheral blood with echinococcosis?
*A) eosinophilia
B) basophilia
B) monocytosis
D) increase of SZE
D) low hemoglobin

6. To diagnose echinococcosis, use reactions:
*A) Katsoni
*B) Hedin-Weinberg
C) Pandey
D) Wasserman
D) RIBT and RIF

7. What changes does the cerebrospinal fluid undergo during echinococcosis
*A) positive protein reactions
*B) eosinophilic pleocytosis
*B) succinic acid
D) reduction of sugar content
D) pronounced increase in protein

1. What are the ways of transmission of toxoplasmosis?
*A) alimony
B) lymphogenic
*B) contact
D) perineural
D) airborne

2. Which of the following is not characteristic of acquired toxoplasmosis?
A) pain in muscles and joints
B) spotted-papular rash
*B) intact lymph nodes
*D) hectic temperature
D) damage to the nervous system

3. What are the symptoms of congenital toxoplasmosis?
*A) chorioretinitis
*B) hydrocephalus
C) intracranial hypotension

*D) calcification cells in the brain
D) "empty" Turkish saddle

4. A woman with a history of several miscarriages, which ended 3 previous pregnancies, turned to medical and genetic counseling. Laboratory blood tests were carried out. The serological reaction of complement binding to toxoplasma antigen is positive.
What else needs to be done to establish a diagnosis of "Toxoplasmosis"?

Answer:
1. Intradermal test with toxoplasmin.
2. Carefully check the objective somatic and neurological status of the patient.

Recommended Books.

1. Neurology: study guide / [I.A. Hryhorova, L.I. Sokolova, R.D. Gerasymchuk, A.S. Son, etc.] edited by I.A. Grigorova, L. I. Sokolova - 3rd edition - Kyiv, Medical University "Medicine", 2020 - 640 p.
2. Topical diagnosis of nervous system pathology. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.
3. Methods of examination of a neurological patient: teaching. Guide / edited by L. I. Sokolova, T. I. Ilyash. K., 2015. – 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.
4. 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 literature

1. Bozhenko M.I., Negrych T.I., Bozhenko N.L., Negrych N.O. Headache. Tutorial. - K.: Medknyga Publishing House, 2019. - 48 p.
2. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 1 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2020. - 258 p.
3. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 778 p.
4. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 642 p.

Electronic information resources

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

ISW No. 5

Topic: Congenital defects of the spine and spinal cord. Syringomyelia.

Relevance of the topic: Syringomyelia refers to diseases based on congenital defects in the development of the nervous system. Often congenital or constitutional anomalies create a favorable background for the development of this pathology. With the change of the radiation background in Ukraine, these questions are becoming more and more relevant.

Specific goals:

- know:

a) dysraphic status in syringomyelia;
b) syringomyelia clinic;
c) method of examination of a patient with syringomyelia.

- to be able to

a) diagnose syringomyelia;
b) prescribe paraclinical examinations for syringomyelia;
c) prescribe treatment for syringomyelia;
d) diagnose syringobulbia;

Topic content:

Classification of disorders of the spine and spinal cord. Despite the fact that researchers of the 19th century pointed out the connection between heredity and the frequency of spinal hernias, the real interest of geneticists in this problem appeared in the last decades of the 20th century. Currently, the concept of "spinal dysraphia" unites various disorders of the development of the spinal cord and spine:

spina bifida occulta - Hidden non-union of the spine;

spina bifida cystica uverta - Open splitting of the spine with the formation of a cystic spinal hernia;

rachischisis posterior (totalis et partialis) - Splitting of the spine and soft tissues from spinal cord dehiscence, which occur along the entire length of the spine or only in some part of it.

Hidden non-unions of the spine are usually localized in the lumbosacral region and, as a rule, do not manifest themselves clinically. Often they are an accidental "finding" during an X-ray examination of the spine. The skin in the area of non-union of the vertebral arch is not changed, but pigment spots, subcutaneous fat (lipomas), fistulae (dermal sinuses) can be determined. The anatomical essence of the hidden gap of the spine consists in the incomplete overgrowth of the vertebral arch.

From the time of the first descriptions of hidden non-union of the spine by R. Virchow (1875), Recklinghausen (1886), it was believed that this anomaly of the development of the spine, caused by a violation of ossification, does not require medical assistance. According to the data of A. D. Speransky, published in 1925 in the work "Origin of spina bifida occulta in the sacral section of the human vertebral column", it was claimed that incomplete closure of the sacral arches occurs in 70% of people and is the norm. Only the following anatomical studies and the data of modern diagnostic methods (computed tomography, nuclear magnetic tomography) made it possible to detect concomitant changes in the defect sites of the vertebral arches, which lead to nocturnal urinary incontinence, pain in the lumbosacral region, postural disturbances, less often to weakness of the leg muscles, deformation of the feet, sensitive and trophic disorders. It is these cases of spina bifida occulta that require surgical assistance. Open cystic splits of the spine (True Spinal Hernias) depending on the degree of involvement in the pathological process of nerve structures are divided into the following.

Membranous forms (meningocele) - Splitting of the spine with protrusion into a defect of the dura mater, but without involvement of nervous structures in the process. After exiting the bone defect, the dura mater thins and disappears. The dome of the hernia sac is represented by a thin pial membrane. The skin of the hernial protrusion is thinned, and often absent at the top. The contents of the hernial sac are meninges and cerebrospinal fluid, its shape is usually stalked with

a narrowed leg. A bone defect usually involves two or three vertebrae. Any clinical manifestations in this form of spinal hernia are not noted, and only the threat of rupture of the hernial sac, its increasing size, serves as a basis for surgical plastic surgery of the defect.

Root form (meningoradiculocele) - Splitting of the spine with protrusion of the membranes of the spinal cord and its roots into the defect, which can partially end in the wall of the bag or enter it, creating a loop, but later, spreading into the intervertebral openings, form normal nerves. The bone defect involves 3-5 vertebrae. The neurological defect in this form of spinal hernia depends on the number of roots involved in the pre-pathological process, blindly ending in the wall of the hernia sac. Depending on this, defects can manifest themselves from mild weakness in the limbs and pelvic disorders to gross paresis and urinary incontinence.

Cerebral form (meningomyelocele or meningomyelocele) - Splitting of the spine with involvement of the membranes, spinal cord and its roots in the hernial sac. The pyal membrane lines the hernial sac, the dura mater ends in the area where the spine splits, the spinal cord and roots often end blindly in the hernial sac. The bony defect is usually wide and long, covering from 3 to 6-8 vertebrae. As such, the hernia sac does not have a neck and directly passes from the spinal canal to the hernial protrusion. There is no skin on the top of the protrusion, the hernia is covered with a thin translucent sheet of the pial membrane. The degree of neurological defect is always severe - lack of movement in the limbs, their underdevelopment, deformation, incontinence of urine and feces. It is this cerebral form of spinal hernias that occurs most often, and it often leads to the rupture of the hernial sac with the end of the cerebrospinal fluid - to the cerebrospinal fluid.

Cystic form (myelo cystocele) - a rather rare form of spinal hernias, in which the terminal part of the spinal cord is sharply expanded due to the central canal of the spinal cord. Therefore, the hernia sac is lined from the inside with a cylindrical epithelium, as is the central channel. Nerve roots depart from the outer surface of the hernial protrusion and go to the intervertebral openings. The degree of neurological defect, as with the cerebral form, is severe - lack of movements in the limbs, gross pelvic disorders.

Complicated form (Spina bifida complicata) It is characterized by a combination of one of the above forms of spinal hernia with benign tumors (lipoma, fibromas) that are fixed to the membranes, spinal cord or its roots.

Non-union of the spine and soft tissues with an unformed spinal cord (Rachischisis posterior) is an extreme degree of mutilation, never accompanied by a cystic component and protrusion of the education above the skin. The defect of the skin, soft tissues, posterior half-ring of the spinal canal is gaping, and in its depth a strip of nervous tissue with a large number of small vessels (area medulla-vasculosa) is visible. The skin defect is covered by a fragmented pial membrane with the end of the cerebrospinal fluid. Partial rachischisis in live newborns usually extends to 3-5 vertebrae.

Typical for all types and forms of spinal dysraphia is their posterior location with a defect of the posterior semiring of the spinal canal. Extremely rarely (less than 1% of cases), nonunion is formed on the anterolateral surface of the canal, and anterior spinal hernias occur. With lumbosacral localization, these hernias spread into the pelvis and complicate the process of defecation. At a higher location, they can compress the formations of the chest, neck, and nasopharynx. The location of spinal hernias along the length of the spinal column in 90% of cases is limited to the lumbosacral region. Thoracic and cervical localization of hernias is relatively rare. It is interesting that when studying the material of spontaneous abortions, Japanese scientists found a more frequent violation of the formation of the spine and spinal cord in the thoracic and cervical regions, as well as a high frequency of defects involving the entire spinal column. This, to some extent, suggests that the embryo and fetus with a gross defect in the formation of the neural tube, as a rule, die. In order to understand the essence of the formation of malformations of the spine and spinal cord, it is necessary, at least in general terms, to imagine the process of embryogenesis of these structures. In the first week of pregnancy, the embryo undergoes cell division with the formation of germinal nodules. In the second week - the formation of the axial organs of the embryo. In the third week, the process of formation of the primary neural tube from the outer germ

layer begins, which goes through the stages of primary (3-4 weeks of pregnancy) and secondary (4-7 weeks of pregnancy) neurulation. It is at these stages of embryogenesis that primary neurulation disorders and the formation of spinal dysraphia occur. In the stage of secondary neurulation, defects in the development of the lumbosacral spine may appear. Therefore, the early periods of pregnancy, if it is not related to hereditary factors, are decisive for the formation of defects in the development of the neural tube, and all modern methods of preventing this pathology apply to the periods before the onset of pregnancy and its first weeks. Modern diagnosis of defects in the development of the neural tube Despite the successes in the early diagnosis of defects in the development of the neural tube, due to the introduction into practice of biochemical methods (research of the content of -fetoprotein and acetylcholinesterase in the serum of the mother and amniotic fluid), methods of fetal endoscopy (ultrasound, nuclear magnetic) the main the value in reducing the frequency of this anomaly belongs to preventive measures. Given that the causes of defects in the development of the neural tube are multifactorial and these factors are known, the formation of risk groups of pregnant women with the highest probability of having a child with a defect is justified. Therefore, it is recognized all over the world that when planning a pregnancy, parents need to be examined by a geneticist, and the future mother by a gynecologist, in order to take measures to prevent neural tube defects, classify pregnant women into different risk groups, and monitor the course of pregnancy with varying degrees of vigilance. What factors contribute to the appearance of a defect in the development of the neural tube?

First, a genetic defect inherited from one of the parents.

Secondly, the influence of adverse environmental factors contribute to the appearance of mutations in the gene. It is known that the incidence of neural tube defects ranges from 1:500 to 1:2000 live newborns in different regions of the world and ethnic groups, with an average of 1:1000. However, if there have been cases of children with neural tube defects in the family of parents or close relatives, then the probability of a child with a defect increases to 2-5%. The same applies to the birth of a second child, if the first was born with a defect (the risk is about 5%). Spontaneous abortions (miscarriages), premature births, infant mortality in the family and among relatives are also an alarming moment in this regard. Therefore, the genetic predisposition to the appearance of a child with a neural tube defect is the main indicator of the inclusion of a pregnant woman in the high-risk group. The external factors that contribute to the appearance of a defect in the development of the neural tube include:

- radiation (living in areas contaminated with radionuclides, working with radiation sources);
- toxic substances of chemical origin (petroleum products, fertilizers, pesticides, etc.);
- use of anticonvulsant drugs by a woman before pregnancy and in its first months;
- high body temperature or use of hot baths at the beginning of pregnancy;
- diabetes and obesity;
- unbalanced diet, deficiency of vitamins and especially folic acid.

The detection of one, and even more so, several of these factors, is the basis for including a pregnant woman in the group of high risk of giving birth to a child with a neural tube development defect. The optimal algorithm of prenatal examination to reduce the frequency of defects in the development of the neural tube involves the following. During pregnancy planning - consultations of a geneticist, therapist, obstetrician-gynecologist, and, if necessary, a urologist. Selection of groups of pregnant women with high and low risk of giving birth to a child with a neural tube defect. Prenatal diagnosis and scope of examination of pregnant women differ in different risk groups. In low-risk groups, the following are carried out:

- monthly consultations (examinations) by an obstetrician;
- in the second trimester of pregnancy, blood analysis of the pregnant woman for the content of fetoprotein and acetylcholinesterase (with increased levels - repeated analysis of their content in the amniotic fluid and ultrasound examination of the fetus). If the presence of a neural tube defect is confirmed, the question of termination of pregnancy is raised;

- in the third trimester of pregnancy - ultrasound examination and preparation for childbirth.

In high-risk groups, the following are carried out:

- monthly examination by an obstetrician;
- in the second trimester of pregnancy, repeated monitoring of the content of fetoprotein and acetylcholinesterase in the blood serum and amniotic fluid, repeated ultrasound examination of the fetus in order to identify possible congenital malformations of the fetus, in difficult situations, use magnetic resonance examination.

Confirmation of a defect in the development of the neural tube is usually a reason for termination of pregnancy, but modern methods of prenatal diagnosis are not absolute. They more often diagnose the very fact of the presence of a defect, but it is not always possible to specify the degree of its severity. At the same time, the degree of involvement in the pathological process of nervous structures is considered to be decisive for the prognosis. With meningocele and timely surgical assistance, the child develops fully, and in the future becomes a normal able-bodied person. With meningomyelocele, even surgical care does not ensure a high quality of life, the child will be disabled, often severely. Therefore, the detection of a neural tube development defect in a fetus is always a valid reason for termination of pregnancy.

The situation is much more complicated in families where pregnancy is long-awaited and the prospect of a new pregnancy is unlikely. If it is not possible to clarify the severity of the defect, additional diagnostic methods are used: nuclear magnetic resonance imaging (MRI), but it does not always allow answering the questions. Then the doctors together with the parents, explaining all the circumstances and possible results, decide the fate of the fetus.

Syringomyelia is a chronic progressive disease of the nervous system, characterized by the growth of glia and the formation of cavities in the spinal cord (from the Greek syrinx - tube). If the pathological process is not limited to the spinal cord, but passes to the brain stem, it is called syringobulbia. Etiology and pathogenesis. The main cause of the disease is defects in the embryonic development of the spinal cord with a defect in the overgrowth of the seam in the place where both halves of the medullary tube close. It is often combined with Arnold-Chiari anomaly. Pathomorphology. Pathological changes are localized mainly in the lower cervical and upper thoracic parts of the spinal cord. On the section of the spinal cord, irregularly shaped cavities located in the gray matter around the central canal are revealed.

Clinic. The disease begins at the age of 15 to 40 years. The main symptoms of syringomyelia are dissociated sensitivity disorders (reduction or loss of pain and temperature sensitivity with intact tactile and muscle-joint sensation). Sensitive disorders appear in the form of a "jacket" or "half-jacket". Vegetative disorders in the area of the upper limb and trunk are very pronounced and varied in syringomyelia. Movement disorders are manifested by atrophic paresis of the muscles of the upper limbs. Cerebrospinal fluid in syringomyelia is often unchanged. Course. Slowly progressive. Sometimes there is stabilization of the process for several years.

Syringomyelia should be differentiated from hematomyelia, intramedullary tumor, subacute poliomyelitis, amyotrophic lateral sclerosis. Treatment. Treatment of syringomyelia is surgical when combined with Arnold-Chiari anomaly, as well as symptomatic: proserin, dibazole, antipsychotics and antidepressants for intense pain, massage, exercise therapy.

Test tasks for self-control.

1. Which of the following is not classified as status dysraphic?

a) kyphosis;
b) scoliosis;
c) amaurosis; Answer: in
d) clubfoot;
e) oligodactyly.

2. What is the most rational therapy for syringomyelia?

a) antibiotic therapy;
b) dehydration therapy;
c) X-ray therapy; Answer: in
d) vascular therapy;
e) tissue therapy.

Problems for self-control with answers:

A 35-year-old patient complains of gradually increasing weakness of the muscles of the hands, loss of pain, heat and cold sensation in them.

An objective examination revealed multiple scars after burns on the cyst and forearm on both sides, "jacket"-type analgesia, Claude-Bernard-Horner symptom, symptoms of pyramidal insufficiency in the legs.

What is the patient's diagnosis?

Answer: syringomyelia.

Recommended Books.

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

2. Topical diagnosis of nervous system pathology. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.

3. Methods of examination of a neurological patient: teaching. Guide / edited by L. I. Sokolova, T. I. Ilyash. K., 2015. – 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: training. 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. Tutorial. - K.: Medknyga Publishing House, 2019. - 48 p.

2. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 1 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2020. - 258 p.

3. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 778 p.

4. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson .- "Medicine", 2021. - 642 p.

Electronic information resources

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

- 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

ISW No. 6

Topic: Perinatal lesions of the nervous system.

Etiological factors (intrauterine, birth trauma, brain damage in the early postpartum period). Hypoxic-ischemic encephalopathy (acute period, recovery period). Children's cerebral palsy, clinical forms - spastic, hemiplegic, ataxic, quadriplegic, hyperkinetic. Diagnostics. Treatment (medicinal, non-medicinal).

Prevention. Perinatal encephalopathy (PEP) (peri- + lat. natus - "birth" + Greek. encephalon - "brain" + Greek. patia - "disorder") - a term that would describe a large group of lesions of different causes and unspecified origins of the brain that occur during pregnancy and childbirth. PEP can be manifested in different ways, for example, hyperexcitability syndrome, when the child's irritability is increased, appetite is reduced, the baby often burps during feeding and refuses the breast, sleeps less, falls asleep more difficult, etc. A more rare, but more severe manifestation of perinatal encephalopathy is the syndrome of depression of the central nervous system. Such children have significantly reduced motor activity. The baby looks lethargic, the cry is quiet and weak. He quickly gets tired during feeding, in the most severe cases the sucking reflex is absent. Manifestations of perinatal encephalopathy are often mild, but children who have experienced this condition still require increased attention, and sometimes special treatment.

Causes of the disease

Risk factors for the occurrence of perinatal brain pathology include:

Various chronic diseases of the mother.

Acute infectious diseases or exacerbation of chronic foci of infection in the mother's body during pregnancy.

Eating disorder.

The age of the pregnant woman is too young.

Hereditary diseases and metabolic disorders.

Pathological during pregnancy (early and late toxicoses, threat of abortion, etc.)

Pathological course of childbirth (rapid childbirth, weakness of labor, etc.) and injuries during the provision of assistance during childbirth.

Harmful environmental influences, adverse environmental conditions (ionizing radiation, toxic effects, including when using various medicinal substances, environmental pollution with heavy metal salts and industrial waste, etc.)

Prematurity and immaturity of the fetus with various disorders of its vital activity in the first days of life.

It should be noted that the most common are hypoxic-ischemic (their cause is a lack of oxygen that occurs during the fetal life of the baby) and mixed lesions of the central nervous system, which is explained by the fact that almost any discomfort during pregnancy and childbirth leads to a violation of the supply oxygen of fetal tissues and primarily of the brain. In many cases, it is not possible to establish the causes of PEP.

The 10-point Apgar scale helps to form an objective picture of the child's condition at the time of birth. At the same time, the activity of the child, the color of the skin, the severity of the physiological reflexes of the newborn, the state of the respiratory and cardiovascular systems are taken into account. Each of the indicators is evaluated from 0 to 2 points. The Apgar scale makes it possible to assess the child's adaptation to extrauterine conditions in the first minutes after birth in the delivery room. The sum of points from 1 to 3 indicates a severe condition, from 4 to 6 - a condition of moderate severity, from 7 to 10 - a satisfactory condition. Low scores are considered risk factors for the child's life and the development of neurological disorders and dictate the need for emergency intensive care.

Unfortunately, high scores on the Apgar scale do not completely exclude the risk of neurological disorders, a number of symptoms appear already after the 7th day of life, and it is very important to detect possible manifestations of PEP as early as possible. The plasticity of the child's brain is extremely high, timely medical measures help in most cases to avoid the

development of neurological deficits, to prevent disorders in the emotional and volitional sphere and cognitive activity.

The course of PEP and possible forecasts

During PEP, three periods are distinguished: acute (1st month of life), recovery (from 1 month to 1 year in full-term infants, up to 2 years in premature infants) and the result of the disease. Different syndromes are distinguished in each period of PEP. Most often, a combination of several syndromes is observed. Such a classification is expedient, as it allows distinguishing syndromes depending on the age of the child. Appropriate treatment tactics have been developed for each syndrome. The expressiveness of each syndrome and their combination make it possible to determine the severity of the condition, correctly prescribe therapy, and make forecasts. It should be noted that even minimal manifestations of perinatal encephalopathy require appropriate treatment to prevent adverse consequences. All patients with severe and moderate brain damage require inpatient treatment. Children with mild disorders are discharged from the maternity hospital under the ambulatory observation of a neurologist.

The syndrome of increased neuro-reflex excitability is manifested by increased spontaneous motor activity, restless superficial sleep, lengthening of the period of active wakefulness, difficulty falling asleep, frequent unmotivated crying, revitalization of unconditioned innate reflexes, variable muscle tone, tremors (twitching) of the limbs, chin. In premature infants, this syndrome in most cases reflects a decrease in the threshold of convulsive readiness, that is, it indicates that the baby can easily develop convulsions, for example, with an increase in temperature or the action of other stimuli. With a favorable course, the severity of symptoms gradually decreases and disappears within 4-6 months to 1 year. With an unfavorable course of the disease and the absence of timely therapy, an epileptic syndrome may develop.

Convulsive (epileptic) syndrome can appear at any age. In childhood, it is characterized by a variety of forms. Imitation of unconditional motor reflexes is often observed in the form of paroxysms of bending and tilting of the head with tension of the arms and legs, turning the head to the side and extending the arms and legs of the same name; episodes of convulsions, paroxysmal twitching of the limbs, imitations of sucking movements, etc. Sometimes it is difficult even for a specialist to determine the nature of emerging convulsive states without additional research methods.

Hypertensive hydrocephalus syndrome is characterized by an excessive amount of fluid in the spaces of the brain containing cerebrospinal fluid (cerebrospinal fluid), which leads to increased intracranial pressure. Doctors often call this disorder to parents just like that - they say that the baby has increased intracranial pressure. The mechanism of occurrence of this syndrome can be different: excessive production of cerebrospinal fluid, impaired absorption of excess cerebrospinal fluid into the bloodstream, or their combination. The main symptoms of hypertensive-hydrocephalic syndrome, which doctors focus on and which parents can control, are the rate of growth of the child's head circumference and the size and condition of the cerebellum. In most premature newborns, the normal head circumference at birth is 34-35 cm. On average, in the first six months, the monthly increase in head circumference is 1.5 cm (up to 2.5 cm in the first month), reaching about 44 cm by 6 months. In the second half of the year, growth rates are decreasing; up to a year, the circumference of the head is 47-48 cm. Restless sleep, frequent profuse vomiting, monotonous crying in combination with an explosive, intensified pulsation of the large fontanelle and throwing the head back are the most typical manifestations of this syndrome.

However, large head sizes are often found in absolutely healthy babies and are determined by constitutional and family characteristics. The large size of the fontanel and its "delay" in closing are often observed in rickets. The small size of the fontanel at birth increases the risk of intracranial hypertension in various adverse situations (overheating, increased body temperature, etc.). Conducting a neurosonographic examination of the brain allows you to correctly diagnose such patients and decide on the tactics of therapy. In the vast majority of cases, by the end of the first half of the child's life, the normalization of the growth of the head circumference is noted. Some

of the sick children have hydrocephalic syndrome without signs of increased intracranial pressure until 8-12 months of age. In severe cases, the development of hydrocephalus is noted.

Comatose syndrome is a manifestation of the severe condition of a newborn, which is assessed by 1-4 points on the Apgar scale. Affected children show pronounced lethargy, a decrease in motor activity up to its complete absence, all vital functions are suppressed: breathing, heart activity. Seizures may occur. The severe condition persists for 10-15 days, while sucking and swallowing reflexes are absent.

The syndrome of vegetative-visceral dysfunctions, as a rule, appears after the first month of life against the background of increased nervous excitability and hypertensive-hydrocephalic syndrome. Frequent vomiting, delayed weight gain, disturbances in heart and respiratory rhythm, thermoregulation, change in skin color and temperature, "marbling" of the skin, disturbances in the functions of the gastrointestinal tract are noted. Often, this syndrome can be combined with enteritis, enterocolitis (inflammation of the small and large intestines, manifesting as a stool disorder, a violation of body weight), caused by pathogenic microorganisms, with rickets, aggravating their course.

The syndrome of movement disorders is detected from the first weeks of life. From birth, a violation of muscle tone can be observed, both in the direction of its decrease and increase, its asymmetry can be detected, a decrease or an excessive increase in spontaneous motor activity is noted. Often, the syndrome of motor disorders is combined with a delay in psychomotor and language development, because impaired muscle tone and the presence of pathological motor activity (hyperkinesis) prevent purposeful movements, the formation of normal motor functions, and language acquisition.

With delayed psychomotor development, the child later begins to hold his head, sit, crawl, and walk. Predominant impairment of mental development can be suspected in the case of a weak monotonous cry, impaired articulation, poor facial expressions, late appearance of a smile, delayed visual and auditory reactions.

The concept of nuclear jaundice (synonym: bilirubin encephalopathy) means severe damage to the central nervous system (CNS) in newborns due to an excessive increase in the level of bilirubin in the blood (hyperbilirubinemia). Reasons: unconjugated bilirubin dissolves very well in fats, but almost does not dissolve in water. Therefore, it binds to albumin for transport to liver cells. With a significant increase in the level of bilirubin, when the transport capacity of albumin is exhausted, free bilirubin can overcome the blood-brain barrier and enter the brain to the basal ganglia. There, bilirubin inhibits the processes of oxidative phosphorylation, which leads to cell death. The so-called basal ganglia (pale globe, shell and caudate nucleus) are very sensitive to the action of bilirubin and are damaged the most, which is why the name of the pathology "nuclear jaundice" has been established. Severe encephalopathy leads to death. Risk factors: on the one hand, risk factors include all conditions that are accompanied by increased formation of bilirubin and increase the risk of developing nuclear jaundice. Among them: increased destruction of blood cells (hemolysis), especially with Rhesus conflict and other blood group incompatibilities. On the other hand, there may be damage also from the side of the blood-brain barrier, when bilirubin can penetrate into the brain even at lower concentrations. For example, hypoxia increases due to acidification of the blood (acidosis), a decrease in blood sugar (hypoglycemia) or hypothermia (hypothermia). Also, a decrease in the concentration of albumin (hypoalbuminemia) leads to the fact that less bilirubin binds in the blood and this leads to a relative increase in free bilirubin, which more easily penetrates into the central nervous system. Also, medications that bind to albumin and thus displace bilirubin lead to an increase in free bilirubin. These include ceftriaxone, sulfonamides, furosemide, digoxin, and diazepam.

Clinical picture: Symptoms of acute bilirubinencephalopathy can be divided into three stages. In the initial phase, drowsiness, muscle hypotonia, and low motor activity appear. In the intermediate phase, the baby's sharp cry is often observed, progressive suppression of consciousness (stupor), irritability and increased muscle tone with overextension of the neck (retrocollis) and spine (opisthotonus). In the progressive phase, the stupor can turn into a coma,

the muscle tone increases even more and eventually convulsions may appear. A fatal outcome is possible.

Chronic bilirubin encephalopathy. If the patient experiences an acute phase, late complications may develop - central deafness, extra-pyramidal movement disorders in the form of athetoid cerebral palsy and retardation in neuropsychological development.

Treatment: To reduce the risk of developing irreversible changes in nuclear jaundice, healthy full-term infants aged 72 hours with a level of unconjugated bilirubin above 340 μmol/l receive phototherapy with blue light with a wavelength of 425-475 nm. If the bilirubin level is above 430 μmol/l, a replacement blood transfusion is performed. In the presence of the risk factors listed above, treatment should begin earlier.

Cerebral palsy (CP) is a collective group of stable, non-progressive motor syndromes (paresis, paralysis, hyperkinesis, ataxia, others), combined with mental and speech disorders, less often epileptic seizures, fluid-dynamic disorders or without them, which are the result of organic damage to the central nervous system. systems in the prenatal, intranatal and early neonatal period. Cerebral palsy is usually diagnosed at the end of the first to second year of a child's life, provided that there is a defect in the motor system. The prevalence of cerebral palsy ranges from 1.5 to 3.0 - 5.0% (in Ukraine - 2.6 - 2.7%). More often, such a diagnosis is registered in children who were born prematurely.

Among the known etiological factors, the role of antenatal damage (intrauterine infections, dys neuroontogenesis, hypoxia and ischemia), natal (childbirth trauma, asphyxia) and postnatal (hemolytic disease of the newborn, others) has been confirmed. The predominant factor is hypoxic damage to brain structures, which leads to morphological changes verified in cerebral palsy: intracranial non-traumatic hemorrhages, multicystic encephalomalacia, periventricular leukomalacia, parasagittal cerebral necrosis, pathology of the basal ganglia, structures of the posterior cranial fossa, brain infarctions, porencephaly, and others.

Classification of cerebral palsy (ICK-10)

The International Classification provides the main clinical variants of cerebral palsy, which are associated with damage to three motor systems: pyramidal, extrapyramidal, cerebellar.

G 80.0 – spastic cerebral palsy (double hemiplegia)

G 80.1 – spastic diplegia (Little's syndrome)

G 80.2 – spastic hemiplegia (infantile hemiplegia)

G 80.3 – dyskinetic cerebral palsy (hyperkinetic, dystonic, athetoid)

G 80.4 – ataxic cerebral palsy (atonic-astatic)

G 80.8 is another form of infantile cerebral palsy.

Mixed forms of infantile cerebral palsy.

G 80.9 – infantile cerebral palsy neutralized.

Stages of cerebral palsy

I. Early stage (from three weeks of life to 3-4 months). At the end of the acute period of the transferred pathology, there are no signs of compensation in the clinical picture: there are autonomic disorders, nystagmus, convulsions, intracranial hypertension, a syndrome of movement disorders (muscular hypotonia, dystonia, pyramidal insufficiency, delayed fading of physiological reflexes, etc.).

II. The initial residual stage (from 5 to 6 months to 3 to 4 years): stable organic motor disorders (spastic paresis, paralysis, hyperkinesis, ataxia, others) and pathological motor stereotype are formed. The child's lag in statokinetic development, delayed reduction of postotonic reflexes is objectively diagnosed.

III. Chronic residual stage (after 3-4 years). It is characterized by the final design of a pathological motor stereotype, the formation of contractures and deformation, and the subsequent clinical definition of other syndromes: epileptic, hydrocephalic, microcephalic, cognitive, behavioral and speech disorders, visual impairment, and others.

Signs that are threatening in relation to the possible formation of cerebral palsy.

1. Severity of the condition in the early neonatal period: APGAR score no higher than 3-4 points, multiple organ failure syndrome, diffuse muscle hypotonia, sharp suppression of unconditioned reflexes, presence of convulsions, cerebrospinal fluid hypertension, gross neurological symptoms, both cerebral and focal (mainly trunk), bulbar, pseudobulbar syndromes.

2. In the period of infancy, the presence of focal neurological symptoms and syndromes (including asymmetric manifestation of physiological reflexes), as well as seizures and other paroxysmal conditions, fluid-dynamic disorders. Clinical definition of delay in physiological postnatal ontogenesis (myelination of CNS structures):

Extended periods of reduction of unconditioned reflexes: Kussmaul's search reflex, proboscis reflex, Babkin's hand-mouth reflex, grasping reflex, Moreau's reflex, reflex of support and automatic gait, Galant's reflex, Perez's reflex, crawling reflex; and congenital tonic reflexes: tonic labyrinth reflex, tonic reflex from the head to the trunk, tonic reflex from the pelvic girdle to the trunk, symmetric cervical tonic reflex, asymmetric cervical tonic Magnus-Klein reflex. The extinction of primitive reflex automatisms of the spinal-trunk level ends by 2

- 4 months of life (in premature infants, individual components of the Moro reflex can persist for up to 6 months);

Prolonged periods of the appearance of postural reflexes (progressive reflexes): righting reactions (labyrinth righting reflex, cervical righting reaction, trunk righting reflex acting on the head, trunk righting reflex acting on the trunk), protective reactions (protective extensor reaction of the arms, reflex Landau), balance reactions and a delay in the rate of formation of postural reflex mechanisms, necessary for the performance of any motor acts.

3. Changes in muscle tone: long-term muscle hypotonia after birth, delayed normalization of muscle tone after 3-4 months of life, presence of symptoms of "punctured ball", "ballerina" and others.

4. Formation of pathological postures and attitudes that make it impossible to perform purposeful action.

5. Delay in the dynamics of the child's statokinetic development until the 1st year (regardless of the term of prematurity, from 7 to 8 months of age, full-term and premature children develop equally).

6. Delay in mental and pre-speech development.

7. Organic changes in the structures of the central nervous system (dilation of the ventricles of the brain, periventricular leukomalacia, multicystic encephalomalacia, expansion of the subarachnoid spaces, porencephaly, focal and diffuse atrophy, some congenital malformations, and others) obtained during studies using direct neuroimaging methods (neurosonography, computer computer tomography, magnetic resonance imaging of the brain).

Forms of infantile cerebral palsy

Spastic diplegia (Little's syndrome) is mainly diagnosed in children who were born prematurely (consequences of intraventricular hemorrhages, other factors). In this form, tetraparesis is actually observed, but muscle spasticity in the legs prevails. This form refers to the most favorable in relation to the possibilities of social adaptation. There are delays in mental and speech development, the presence of elements of pseudobulbar syndrome, dysarthria, strabismus, and others.

Double hemiplegia is the most severe form of cerebral palsy, which is more often a consequence of chronic pre-natal hypoxia with diffuse damage to the cerebral hemispheres. Spastic tetraplegia (tetraparesis), pseudobulbar syndrome, cognitive disorders, speech disorders, elements of extrapyramidal insufficiency are clinically diagnosed. Most children have epileptic seizures.

Spastic hemiplegia is a variant of cerebral palsy, which is formed when one of the hemispheres of the brain is predominantly affected (as a result of the action of a focal factor: hematoma, ischemia, porencephaly, other or congenital defects). It is clinically characterized by the development of spastic hemiparesis (Wernicke-Mann type), delayed mental and speech development. In this form, focal epileptic seizures are common.

The dyskinetic (hyperkinetic) variant of cerebral palsy is one of the possible consequences of the transferred hemolytic disease of newborns, which was accompanied by the development of "nuclear" jaundice. With this form, the structures of the extrapyramidal system and auditory analyzer are mostly damaged. The clinical picture is characterized by the presence of hyperkinesis: athetosis, choreoathetosis, torsion dystonia (in children in the first months of life - dystonic attacks), dysarthria, oculomotor disorders, hearing loss. In most children, preservation of intellectual functions is noted, which is prognostically favorable in relation to social adaptation and learning.

Atonic-astatic variant of cerebral palsy. It is observed when the cerebellum and cerebellar pathways are predominantly damaged as a result of birth trauma, a hypoxic-ischemic factor, or a congenital malformation. Clinically, it is characterized by a classic symptom complex: muscle hypotonia, ataxia and various symptoms of cerebellar asynergy (dysmetria, intention tremor, dysarthria, etc.). The possibility of this occurrence is being considered

variant of cerebral palsy with damage to the cerebral cortex (mainly the frontal lobe). In the latter case, in addition to the presence of muscle hypotonia and astasia - abasia, there are cognitive disorders - various degrees of oligophrenia, which ultimately makes it impossible to make a diagnosis of cerebral palsy.

Mixed forms. Despite the possibility of diffuse damage to all three motor systems of the brain: pyramidal, extrapyramidal and cerebellar, the above-mentioned clinical symptom complexes reliably allow diagnosing a specific form of cerebral palsy in the vast majority of cases. The last provision is important in relation to drawing up the patient's rehabilitation card.

The differential diagnosis is based on the concept of cerebral palsy, which is a collective group of stable non-progressive motor syndromes and implies the expediency of exclusion:

Progressive diseases of the central nervous system: neuromuscular diseases, metabolic diseases, mitochondrial and lysosomal diseases, hereditary degenerative diseases, hamartoses and others; Consequences of organic damage to the central nervous system (inflammatory diseases, traumatic brain injury, cerebrovascular diseases, others) that have been transferred in the term after the early neonatal period;

Oligophrenias, where the degree of impairment of cognitive functions significantly outweighs disorders in the motor sphere.

At the same time, children with cerebral palsy may develop residual-organic epileptic syndrome, hydrocephalus syndrome, mental, behavioral and speech disorders. It is important to remember that cerebral palsy is characterized by pseudo-processuality - changes in the clinical picture accompanying the child's growth and further maturation of the structures of the nervous system.

An in-depth study of the problem of cerebral palsy involves the possibility of separating from this collective group of syndromes other nosological forms of diseases.

Medical and social rehabilitation of children with cerebral palsy

In the context of the state policy in the field of health care, the development of medical and organizational technologies that will contribute to the improvement of care for children with limited health opportunities is of great importance. First of all, this concerns the establishment in Ukraine of the basic provisions of social pediatrics, which conceptually ensures the design of the child's living space in society, taking into account the state of his health

According to the decision of the Scientific Medical Council of the Ministry of Health of Ukraine, in medical and preventive institutions (centers for medical and social rehabilitation of children with organic damage to the nervous system), a model of complex rehabilitation of children with organic damage to the nervous system (including cerebral palsy) "Tandem-partnership" "child-family" has been implemented -specialist", which consists in a harmonious combination of medical and socio-pedagogical aspects of rehabilitation. According to this model, mandatory training of parents in rehabilitation techniques used for the treatment of their child is provided.

The launched model is used in the preparation of an annual complex individual integrated rehabilitation program, where four main functional systems that are subject to correction are defined:

Functional system of movements (motility) – rehabilitation from the restoration of the reflex elementary motor act through the initial motivation of motor development with volitional efforts to the establishment at the next stage of the practical need and awareness of the motivation of movements, purposeful actions.

The functional system of the sensory sphere - from elementary sensation, perception of the surrounding world within the framework of preserved analyzers through the development of the feeling of one's body and movements to sensory integration and social perception.

The functional system of the cognitive sphere - from the primary knowledge of the objective world to conscious play, the development of the prerequisites of intelligence, the formation of cognitive activity and the motivated need for adaptation in the environment. In the language system, rehabilitation is carried out in the direction of the formation of language functions, structure and its reproduction.

The functional system of the child's emotional-communicative sphere and behavior is rehabilitated starting with elementary emotional and communicative reactions connected with the instinctive needs of the child through the development of the emotional-communicative integration of "mother-child" to higher emotions, feelings, the formation of motivations and ways of communication of the child with the surrounding people, the possibility of regulating directed volitional behavior.

The most common methods of medical rehabilitation of children with cerebral palsy include the author's complex integral models, drug treatment, kinesiotherapy, physiotherapy, acupuncture therapy, neuro-orthopedic and neurosurgical correction, sanatorium-resort rehabilitation and others.

I. The author's complex integral models of rehabilitation. The method of V.I. Kozyavkina - the system of intensive neurophysiological rehabilitation (SINFR) is based on biomechanical correction of the spine and large joints using reflexotherapy, mobilizing gymnastics, a special massage system, rhythmic gymnastics, apitherapy, mechanotherapy and other treatment blocks. The system is structurally and functionally divided into two subsystems: intensive correction and stabilization and potentiation. The method of K.O. Semenova - dynamic proprioceptive correction (DPK), which is based on the formation (restoration) or imposition of a new motor stereotype by influencing the functional system of antigravity using the medical suit of cosmonauts.

The method of K. and B. Bobat is a neurodevelopmental therapy aimed at suppressing the activity of pathological tonic reflexes, postural reactions and motor stereotypes with the subsequent restoration of statics and motor skills (crawling, standing, walking) regardless of the age of the patient. The method of V. Voyta is a system treatment according to the method of reflex-locomotion (innate reactions of reflex crawling and reflex turning over), which involves the possibility in young children, mainly from the risk group, to "transform" pathological reactions into a physiological motor stereotype and, thereby, prevent the formation of cerebral palsy. Methods of functional biomanagement, which are based on the principle of biological feedback.

II. Drug treatment. Active use of drug treatment is advisable in the acute and recovery periods of diseases of the nervous system under the condition of unstable compensation of impaired functions. In the chronic-residual period of cerebral palsy, medication is indicated in the presence of epileptic, hypertensive-hydrocephalic syndromes, hyperkinesis, muscle tone disorders, cognitive and behavioral disorders. At the same time, indications for the use of this or that medicinal agent should be based exclusively on the principles of evidence-based medicine.

III. Physiotherapy includes methods of treatment that use active and passive movements: physical therapy (Physical therapy), occupational therapy (Ergotherapy) and physical therapy itself - the use of gymnastic exercises to increase the strength, endurance and mobility of patients. In their practical work, kinesiotherapy specialists use methods of manual therapy and

biomechanical correction, relaxation techniques (including post-isometric muscle relaxation), physical therapy, massage, mobilization, manipulation, and others.

IV. Physical factors (physiotherapy) are used in a complex of measures, which include: mechanotherapy, thermotherapy, hydrotherapy, light therapy, magnetotherapy, electrotherapy and combined combined methods of therapeutic physical factors. At the same time, in modern conditions, the expediency of wide use of such "old" electrotherapy methods as galvanization, electrophoresis, diadynamotherapy, amplipulsetherapy, interference therapy, diathermy, inductothermy and others in children with organic damage to the nervous system is being reviewed.

V. Acupuncture therapy is used in a complex of rehabilitation measures using various modifications (classical reflexology, micro-acupuncture, scalp therapy and others).

VI. Orthopedic and neurosurgical correction for cerebral palsy is carried out exclusively according to indications and in medical institutions specified by the Ministry of Health of Ukraine.

VII. Sanatorium-resort rehabilitation of children with cerebral palsy is carried out in sanatoriums of a neurological profile ("Hadzhibey", "Iskra", "Batkivshchyna", others) using climate treatment, balneotherapy, therapeutic mud and other natural factors. Socio-pedagogical rehabilitation is an equal component of the "Tandem" model. Medical psychologists, together with speech therapists and teachers, diagnose the level of development of mental and language functions of the child, provide psychotherapeutic and pedagogical assistance, including the harmonization of the socio-psychological climate in the family. This aspect of complex rehabilitation includes: psychological correction, psychopathological correction, defectological correction, socio-psychological correction, social therapy, Montessori therapy, conductive pedagogy according to Pete, speech therapy correction, sensory integration. Important importance is given to determining the effectiveness of rehabilitation measures. This involves the definition of the following rehabilitation criteria: coefficient of the quality of the implementation of the rehabilitation program (course treatment); coefficient of the quality of the implementation of the rehabilitation process (during the year); rehabilitation potential - a set of characteristics of the state and development of the functional systems of the child in interaction with environmental factors that determine the possibility of social adaptation ;rehabilitation forecast - the possibility of realizing the rehabilitation potential in all its properties.

Throughout their lives, children with cerebral palsy are provided with assistance in institutions of various departmental subordination in compliance with the continuity and standards of medical, pedagogical, psychological and social components of a complex individual integrated rehabilitation program.

Recommended Books.

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

2. Topical diagnosis of nervous system pathology. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.

3. Methods of examination of a neurological patient: teaching. Guide / edited by L. I. Sokolova, T. I. Ilyash. K., 2015. – 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: training. 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. Tutorial. - K.: Medknyga Publishing House, 2019. - 48 p.

2. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 1 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson. - "Medicine", 2020. - 258 p.

3. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 2 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson. - "Medicine", 2021. - 778 p.

4. Medicine according to Davidson: principles and practice: 23rd edition: in 3 volumes. Volume 3 / edited by By Stuart G. Ralston, Ian D. Penman, Mark W.J. Straken, Richard P. Hobson. - "Medicine", 2021. - 642 p.

Electronic information resources

- Medical Books On-line Library (Neurology) – free download
http://medbookshelf.info/category/neurology/
- Clinical guidelines in neurology. (Order of the Ministry of Health of Ukraine No. 487 dated 17.08.2007) https://zakon.rada.gov.ua/rada/show/v0487282-07#Text

ISW No. 7

Topic: Medicines used in neurology. The procedure for providing palliative care to incurable patients .

Relevance of the topic: Indications and contraindications for drugs used in neurology should be known by all doctors. Also, the palliative care procedure for untreated patients is very important in daily clinical practice.

Personal goals:

Know:

Classification of drugs used in neurology.

Indications and contraindications for drugs used in neurology.

Palliative care procedure for untreated patients.

Be able:

- Prescribe adequate treatment.
- Provide palliative care for untreated patients.

Classification of drugs:

I. Preparations of the autonomic nervous system

- Cholinergic (parasympathetic) drugs
- Drugs that block cholinergic receptors
- Adrenergic drugs (catecholamines, non-catecholamines)
- Drugs that block adrenergic receptors

II. Neurological and neuromuscular drugs

- Muscle relaxants of skeletal muscles
- Drugs that block neuromuscular transmission
- Drugs against parkinsonism
- Antiepileptic drugs
- Anti-migraine drugs

III. Medicines for pain

- Non-opioid analgesics, antipyretics and NSAIDs
- Opioid agonists and antagonists
- Anesthetic drugs

IV. Cardiovascular drugs

- Inotropes
- Antiarrhythmic drugs
- Drugs against angina pectoris
- Against hypertension

V. Hematological preparations

- Hematinics
- Anticoagulants
- Thrombolytic drugs

VI. Drugs for the respiratory system

- Beta2-adrenergic agonists
- Anticholinergic drugs
- Corticosteroids

VII. Preparations for the gastrointestinal system

- Anti-ulcer drugs
- Anti-infective drugs

VIII. Anti-inflammatory, anti-allergic and immunosuppressive drugs

- Antihistamines
- Corticosteroids

IX. Psychotropic drugs

- Sedative and hypnotic drugs
- Antianxiety drugs
- Antidepressants and mood stabilizers

X. Drugs of the endocrine system

- Anticidal drugs and glucagons
- Estrogens
- Preparations for the thyroid gland

XI. Preparations for fluid and electrolyte balance

- Preparations for replacement of electrolytes

XII. Antitumor drugs

The World Health Organization (WHO), in a 1990 report, defined palliative care as "the active general care of patients whose disease is unresponsive to treatment." This definition emphasizes the terminal nature of the disease. However, the term can also be used more generally to refer to anything that relieves symptoms, even if there is hope for recovery by other means; thus, according to a more recent statement by the WHO, palliative care is "an approach that improves the quality of life of patients and their families who face the challenges of life-threatening illnesses." In some cases, palliative care may be used to alleviate the side effects of treatment, such as to relieve nausea associated with chemotherapy.

The term is not generally used to refer to chronic diseases such as diabetes, which, although not curable, have treatments that are (ideally) effective enough not to be considered a progressive or life-threatening disease in the same sense as cancer or progressive neurological diseases. disease. However, the term is sometimes used to refer to certain diseases, such as chronic progressive lung disease and end-stage renal disease or chronic heart failure.

Although the concept of palliative care is not new, in the past most physicians focused on aggressively trying to cure patients, so attention to patient comfort was seen as "abandoning" them. Recently, the concept of quality of life has gained popularity, although many argue that it is still far from the goal. A relatively new development is the concept of a medical team that is entirely focused on palliative care; this is often called hospice or palliative care.

History of hospice

Hospices were originally places of rest for travelers in the 4th century AD. In the 19th century, the religious order established hospices for the dying in Ireland and London. Modern hospice is a relatively new concept that emerged and gained momentum in Great Britain after the founding of St Christopher's Hospice in 1967. Dr. Saiseli Saunders is considered the founder of

the hospice movement. Since its inception, the hospice movement has grown significantly. There were just under 1,700 hospice services in the UK in 2005. These included 220 adult inpatient units with 3,156 beds, 33 pediatric inpatient units with 255 beds, 358 home care services, 104 home hospice services, 263 day care services and 293 hospital teams. Together these services helped more than 250,000 patients in 2003/4. Funding varies from 100% NHS funding to almost 100% charity funding, but services are always free to patients. The first hospice in the United States was founded in 1974. Relatively generous Medicare reimbursement for hospice care has greatly increased the use of hospice in the United States. There are currently approximately 3,300 hospice services in the United States.

Practice of palliative care

Most often, palliative care is provided at home for people at the end of life. This can also be done in independent inpatient units (hospices) and in regular hospital departments.

In most countries, hospice care is provided by an interdisciplinary team consisting of doctors, nurses, chaplains, social workers, physical therapists, rehabilitation specialists, as well as volunteers and, most importantly, family members. The main goal of the team is to ensure the patient's comfort as much as possible. Additional team members often include caregivers, volunteers from the local community (some untrained, but some also medically trained) and cleaners. In the UK, palliative care services offer inpatient care, day care, outpatient care and work in close partnership with mainstream services. Hospices often have a full range of services and specialists. In the US, patients are usually admitted to a palliative care program if there is a realistic expectation of death within 6 months. However, this does not mean that if a patient continues to live after six months in hospice, they are automatically discharged from the service. Such restrictions do not exist in other countries such as the UK. Caregivers, both family and volunteer, are important to the palliative care system.

Materials for self-control of training quality:

A 26-year-old graduate student with severe throbbing pain on the left side of the head, who has nausea, vomiting and photophobia, applies to the admissions department. She tried ibuprofen with no relief. On further questioning, she reports that she has had similar headaches three to four times a month for the past year. Her mother also had similar problems. Her examination is normal. Which of the following medications is currently appropriate for the treatment of this headache?

and. Ergotamine tartrate

b. Nitroglycerin

in. Verapamil

g. Amitriptyline hydrochloride

d. Phenobarbital

A 70-year-old woman with a regressive stage of kidney disease is prone to restless legs syndrome with the appearance of uremia. This can be controlled with which of the following drugs?

and. Haloperidol

b. Clonazepam

in. Caffeine

Mr. Nifedipine

d. Rifampicin

A 55-year-old right-handed man was hospitalized in the medical department with pneumonia. The patient usually consumes 4 to 8 beers per day. Given the possible seizures, cognitive impairment, and autonomic instability that may occur during withdrawal, which of the following is the most appropriate intervention?

and. Consult the "detox center" to begin planning the patient's discharge

b. Provide intravenous additional doses of alcohol to smooth the withdrawal from alcohol

in. Provide intramuscular or oral chlordiazepoxide several times daily at a dose determined by the level of seizures

d. Start phenytoin as a single daily dose

d. Delay treatment of pneumonia until the risk of neurological problems decreases

A 23-year-old woman with HIV infection presents with vision loss. After testing, a diagnosis of retinitis caused by cytomegalovirus (CMV) is made. Which of the following is the most appropriate treatment for this patient?

and. Cytarabine

b. Vidarabin

in. Ribavirin

Mr. Interferon

d. Ganciclovir

A 42-year-old man presents with horizontal nystagmus in primary gaze and during both left and right gaze, gait ataxia. Which of the following is the most likely cause of the patient's evoked nystagmus?

and. Hysteria

b. Narcotic poisoning

in. Eye fatigue

Mr. Myopia

d. Hypermetropia

A 62-year-old man is being treated for tuberculous meningitis with isoniazid and rifampicin. To avoid additional signs of neuropathy, which of the following should be used with these antibiotics?

and. Ceftriaxone

b. Thiamine

in. Erythromycin

d. Vitamin B12

d Pyridoxine

Recommended Books:

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., 2015. – 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: training. 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.

Electronic information resources

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

ISW No. 8

Topic 8: Damage to the nervous system in the presence of HIV infection.

Relevance of the topic: In connection with different periods of occurrence of damage to the nervous system, different pathogenesis and pathomorphological changes, as well as localization of the pathological process and different clinical manifestations, it is important to study the main clinical syndromes of AIDS. In order to develop measures to prevent the further spread of this deadly infection.

Specific goals:

know:

1. Etiology, pathogenesis, main clinical manifestations of neuro-AIDS.
2. Damage to the nervous system associated with infections that develop against the background of immunodeficiency caused by toxoplasmosis, herpes simplex virus, cytomegalovirus infection, papavirus, fungi.
3. Damage to the nervous system in AIDS.
4. Ways of transmission of viral infection.
5. Clinical manifestations of subacute encephalitis, cryptococcal meningitis.
6. Local lesions of the brain: causes, symptoms.
7. Neurological manifestations of HIV infection before the development of AIDS.
8. Ways to prevent AIDS.

be able:

1. To study early forms of HIV infection and differentiate them from other diseases.
2. To study patients with encephalitis and meningitis infected with HIV infection.
3. To study the neurological manifestations of HIV before the development of AIDS.

Questions for the seminar session to be discussed:

1. What is the causative agent of AIDS?
2. Causes of neurological disorders in AIDS.
3. Clinical manifestations of subacute encephalitis.
4. Clinical manifestations of cryptococcal meningitis.
5. Causes of local brain lesions.
6. Symptoms of local brain lesions.
7. Name the symptoms of progressive multifocal leukodystrophy.
8. What are the manifestations of retinitis in AIDS patients?

9. Symptoms of vacuolar myelopathy.
10. Clinic and treatment of peripheral neuropathy.

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

1. The most common causes of reduced visual acuity in AIDS patients are:
A. Meningitis
B. Encephalitis
C. Retinitis
D. Myelopathy
E. Neuropathy Answer: S.

2. A symptom of vacuolar myelopathy is:
A. Hemianaesthesia
B. paresthesias
C. weakness in the legs
D. ataxia
E. hemiparesis Answer: V,S.

3. Which of the following is not characteristic of tuberculous meningitis?
A) gradual onset of the disease
*B) sudden onset of the disease
*B) high, hectic temperature
D) subfebrile temperature
D) lesions of the CNS

4. What is characteristic of the changes in the cerebrospinal fluid with tuberculosis of the NA?
A) neutrophilic pleocytosis
*B) lymphocytic pleocytosis
*B) decrease in the amount of glucose
D) increasing the amount of glucose
D) significant increase in protein content

5. Which of the drugs should not be prescribed for tuberculosis?

A) streptomycin
B) rifampicin
B) isoniazid
D) ethambutol
*D) benzohexonium

6. Name the clinical signs that are not related to cryptococcal meningitis:
A. Weight loss
V. Fever
C. Nausea, vomiting
D. Meningeal symptoms
E. Paralysis Answer: E.

7. Specify the manifestations of progressive multifocal leukodystrophy:
A. Hyperhidrosis
B. Paresthesias

S. Ataxia
D. Hemiparesis
E. Reduction of visual acuity Answer: C, D, E.

8. A patient with AIDS, some time after the onset of the disease, began to complain of paresthesias in the legs, weakness in them, difficulty in walking. Objectively: Lower spastic paraparesis, conduction-type anesthesia from the L4-L5 level, impaired deep sensitivity in the toes. Ataxia when performing checkpoints and when walking.

What is the patient's probable diagnosis?
Answer: Vacuolar myelopathy.

Neurological complications affect a significant proportion of HIV-infected individuals and are an important cause of death and morbidity. With progressive deterioration of immune function, the brain and nervous system become susceptible to a number of opportunistic infections and tumors. The most common are cryptococcosis, tuberculosis, cytomegalovirus (CMV) disease, toxoplasmosis, progressive multifocal leukoencephalopathy (PML), and primary lymphoma of the central nervous system. HIV can directly affect the nervous system and cause HIV-associated dementia, distal sensory peripheral neuropathy, myopathy, and vacuolar myelopathy. Side effects of antiretroviral drugs and drugs can affect the nervous system. In addition, long-term HAART can lead to metabolic disturbances that may increase the risk of cerebrovascular disease. Clinical manifestations may be subtle or atypical; dual infections often coexist, and a number of differential diagnoses may need to be considered. Laboratory and radiological studies can give only indirect information about the main etiological agent. HIV itself can cause nonspecific abnormalities in the cerebrospinal fluid including lymphocytic pleocytosis and elevated protein levels even in asymptomatic patients. Access to lesions of the central nervous system for direct histopathological confirmation is often not possible. Based on the presumptive diagnosis, appropriate laboratory and radiological studies or empiric therapy may be prescribed.

Clinical signs of meningitis in an HIV-infected patient may include fever, headache, nausea and vomiting, neck pain, photophobia, drowsiness, or cranial nerve symptoms. On physical examination, neck stiffness, a positive Kernig sign, papilledema, cranial nerve palsy, or impaired level of consciousness may be present. The clinical picture of meningitis against the background of HIV infection may be atypical and consist only of non-specific symptoms, such as worsening of the general condition. The most common causative agents of meningitis in HIV-infected patients are *Cryptococcus neoformans* and *Mycobacterium tuberculosis* (MTB). Bacterial meningitis can also occur in connection with HIV infection. In addition, acute HIV infection can cause aseptic meningitis associated with seroconversion. Sometimes neurosyphilis can be accompanied by meningitis. The patient should be evaluated for previous or concurrent tuberculosis or systemic fungal infection. Skin lesions and lymphadenopathy are looked for so that scraping or aspiration cytology, respectively, can be performed to identify the etiological agent. Blood serum should be checked for cryptococcal antigen and blood should be taken for the culture of fungi and mycobacteria. A chest X-ray should be taken to detect any associated lung lesions. In the absence of focal neurological symptoms or papilledema, a lumbar puncture should be performed as soon as possible. SMP must be sent for microscopy, biochemistry, Gram smear and bacterial culture, acid-fast staining and mycobacterial culture, negative staining and fungal culture, as well as cryptococcal antigen. Polymerase chain reaction (PCR) for MTB should be performed if the test is available. CSF should also be sent for cytology in the rare case of lymphomatous infiltration causing meningitis, and for venereal disease reference laboratory (VDRL) neurosyphilis and/or fluorescent treponemal antibody (FTA) testing. Patients with cryptococcal meningitis are usually treated with a combination of intravenous amphotericin B and oral flucytosine for at least 2 weeks. After this period and when the clinical condition stabilizes, the

patient can be switched to high-dose oral fluconazole until the CMP is sterilized. For patients with severe infection and high levels of cryptococci in the CSF, amphotericin B therapy may need to be extended to 4-6 weeks. The patient should then receive secondary prophylaxis with fluconazole 200 mg daily until the CD4 count rises above 200/μl after HAART. Unlike pulmonary tuberculosis, tuberculous meningitis (TBM) usually occurs in more advanced immunodeficiency with a CD4 count below 50/μL. The diagnosis of TBM is less straightforward unless the patient is known to have tuberculosis, which usually affects the lungs. CSF testing for acid-fast bacilli (AFB) is usually inefficient, and mycobacterial culture may take several weeks to give a positive result. PCR for MTB may be useful if positive, but a negative PCR does not completely exclude the diagnosis. Empiric antituberculosis therapy may be warranted in the clinical setting of meningitis when a bacterial cause is considered unlikely and CSF testing for yeast and cryptococcal antigen is negative. The duration of treatment may be extended for patients who cannot receive isoniazid or rifampicin due to side effects. Corticosteroid therapy has been shown to reduce mortality but not disability from TBM. Initiation of HAART should generally be delayed until the patient's condition is stabilized with anti-TB drugs, and the clinician should recognize the possibility of paradoxical worsening of meningitis due to VSD shortly after initiation of HAART.

An HIV-infected patient with massive brain lesions may present with headache, focal motor weakness or sensory impairment, visual or speech impairment, seizures, and impaired level of consciousness. Physical examination may reveal hemiparesis, hemisensory impairment, dysphasia, or a visual field defect.

Common opportunistic diseases that can present with massive brain lesions include cryptococcoma, tuberculosis, toxoplasmosis, lymphoma, and PML.

The patient should be evaluated for a history of tuberculosis or concurrent tuberculosis, systemic fungal infection, and non-Hodgkin's lymphoma (NHL). A history of injection drug use may indicate the possibility of a bacterial brain abscess. A physical examination should be performed to detect any skin lesions, lymphadenopathy, hepatosplenomegaly, or heart murmurs. The blood should be cultured and checked for antibodies to toxoplasma and fungal antigen. A chest X-ray should be performed to detect any lung involvement. The goal is to detect any signs of systemic disease that may provide clues to the etiology of the cerebral lesion.

Contrast-enhanced CT should be performed to determine the number and location of focal brain lesions, as well as features such as contrast ring enhancement, surrounding edema, mass effect, and midline shift. If the lesion cannot be clearly identified on computed tomography, as is the case with lesions located in the posterior cranial fossa, or with little or no contrast enhancement, magnetic resonance imaging (MRI) should be performed for better delineation.

The treatment strategy depends on the radiological characteristics of brain lesions on CT and MRI. Focal lesions of the brain with enhancement of the contrast ring, mass effect, and surrounding edema may be the result of purulent brain abscess, tuberculosis, cerebral toxoplasmosis, or lymphoma. If the patient has a history of intravenous drug use or clinical toxicity with peripheral leukocytosis, high-dose antibiotics (ceftriaxone, metronidazole with or without cloxacillin) should be started as soon as possible after blood culture is obtained. The lesions are most likely to be tuberculous or tuberculous abscesses if the patient has active tuberculosis and negative serology for Toxoplasma, especially if there is enhancement of the meninges on a contrast-enhanced CT scan. The anti-tuberculosis regimen should be optimized and corticosteroid therapy may be added, especially if there is a significant mass effect from the brain lesion or if recently developed neurological symptoms may suggest VSD after initiation of HAART.

In other clinical settings, the most common cause of contrast lesions of the brain is cerebral toxoplasmosis, especially if they are multiple and located in the basal ganglia or gray-white matter junction. Toxoplasmosis usually occurs in HIV-infected patients with CD4 levels below 50/μL. IgG antibodies against toxoplasma were found in most patients. Even if the Toxoplasma serology is negative, the patient should still receive empiric antitoxoplasmosis therapy with pyrimethamine (200 mg as a single dose, then 50-75 mg daily) and clindamycin (600 mg every 6 hours). Most patients with cerebral toxoplasmosis respond to therapy within 2 weeks. Corticosteroids should be

avoided if serology is negative for Toxoplasma unless the patient has a significant mass effect causing severe clinical symptoms, so as not to give the false impression of a favorable response to therapy.

PCNSL is another important differential diagnosis of contrast-enhancing brain tissue lesions. It may have similar radiographic manifestations to cerebral toxoplasmosis, and the diagnosis may depend on stereotaxic biopsy of the lesion after failure to respond to a two-week empiric course of anti-toxoplasmosis therapy. In addition, thallium-201 single-photon emission computed tomography (SPECT) and PCR for EBV DNA in cerebrospinal fluid can help distinguish between neoplastic and infectious causes of cerebral lesions. The management approach is detailed in Chapter 33.

Lesions affecting predominantly white matter without mass effect or contrast enhancement are usually the result of PML. Diagnosis is based on concordance of clinical and MRI findings. The frontal and parietal-occipital lobes are most often affected by large single or multiple scalloped lesions on the gray-white junction. PCR examination of cerebrospinal fluid for JC virus (JCV) has a sensitivity of 75% and a specificity of about 100%. A brain biopsy may be required in patients with negative JCV PCR on at least two examinations. There is no specific treatment for PML, and the mainstay is improving immune function with HAART.

Some HIV-infected patients may develop a more diffuse disease process that affects brain function. The onset can be subacute or more insidious, and symptoms can range from cognitive impairment, personality or mood changes, impaired motor function to confusion or clouding of consciousness. Such clinical manifestations are usually due to HIV encephalopathy, leading to a syndrome known as HAD. Differential diagnosis includes CMV encephalitis and neurosyphilis. Acute HIV infection can also be accompanied by encephalitis associated with seroconversion.

The patient should be evaluated for any reversible causes of cognitive impairment or confusion. It is necessary to conduct a funduscopic examination to detect signs of CMV retinitis. Blood should be checked for electrolytes, vitamin B12, folic acid, thyroid hormone levels, cryptococcal antigen, and VDRL. The possibility of depression should be considered and the patient should be referred to a neuropsychologist to assess baseline cognitive function and monitor progress after treatment. The cerebrospinal fluid should be tested for infectious agents and an electroencephalogram should be performed to detect any seizures. CT and MRI should also be ordered to detect any lesions in the brain.

A patient with HIV encephalopathy usually has a triad of cognitive, motor, and behavioral disorders. It can occur at any time during the course of HIV infection, but is more common in advanced immunodeficiency. There is a hidden onset of intellectual decline with forgetfulness, decreased concentration, slowing down of the thinking process, and impaired productivity. At its mildest, patients may have only mild cognitive-motor disorder (MCMD), which may go unrecognized by healthcare professionals. Motor disorders can be manifested by clumsiness, weakness of limbs and unsteadiness of gait. The patient may change in personality, become apathetic and withdraw from usual hobbies and social activities. This is accompanied by a progressive deterioration of memory, speech and other cognitive functions, which eventually leads to severe psychomotor retardation, akinetic mutism, overt dementia and organic psychosis.

Physical examination is usually normal but may reveal hyperreflexia and frontal release signs such as glabellar and muzzle reflexes. Neuropsychological tests show signs suggestive of subcortical dementia, with impairments mainly in psychomotor speed, verbal and non-verbal learning. The AIDS clinical trial group used time lapse, finger tapping (dominant hand), scoreboard, and number symbol as screening tools for HAD. A nonspecific mild lymphocytic pleocytosis and an increase in the level of protein in CMP are observed. Brain CT usually shows diffuse cerebral atrophy with ventricular dilatation, and MRI may show T2 hyperintense signals mainly in the periventricular white matter and centrum semioval without mass effect or contrast enhancement. The diagnosis is usually established by excluding other infectious causes of encephalitis.

CMV infection and brain disease may be more common than clinically recognized. It can take the form of diffuse micronodular encephalitis or ventriculoencephalitis. This usually occurs against the background of progressive HIV infection with a CD4 lymphocyte count below 50/μL. The onset of ventriculoencephalitis is more acute with rapidly progressive lethargy and confusion, often associated with nystagmus and cranial nerve palsy. CMV encephalitis can have an indolent course with features mimicking a more aggressive form of AD with a greater tendency to delirium, confusion, and focal neurologic deficits. Patients usually progress rapidly, leading to death within weeks. This can occur in the background of CMV disease in other parts of the body, and the patient may already be receiving ganciclovir therapy, which increases the likelihood that the virus will become resistant to the drug. Electrolyte abnormalities such as hyponatremia and hyperkalemia may indicate adrenal insufficiency due to CMV adrenalitis.

CT and MRI may show findings similar to HIV encephalitis with cerebral atrophy with ventricular dilatation and diffuse T2-hyperintense white matter, but there may be evidence of ventricular or meningeal enhancement. CMP may reveal polymorphonuclear pleocytosis, as well as elevated protein levels and decreased glucose levels. PCR for CMV DNA is usually positive and is predictive of CMV encephalitis in the appropriate clinical context. Quantitative analysis of CMV DNA in CSF may have prognostic value and may be used to monitor response to treatment.

Patients with CMV encephalitis may respond poorly to ganciclovir or foscarnet, especially if they are already receiving this treatment for systemic CMV disease. Combined therapy with ganciclovir (5 mg/kg every 12 hours) and foscarnet (60 mg/kg every 8 hours) may be prescribed depending on the severity of the disease and poor prognosis. Maintenance therapy with ganciclovir (5 mg/kg per day) and foscarnet (90 mg/kg per day) is necessary for patients with a clinical response.

HIV-infected patients may develop complications due to damage to peripheral nerves, spinal nerve roots, spinal cord, or muscles. Symptoms are determined by the structure of the anatomical lesion, and each of them is associated with a different underlying etiology. *Acute inflammatory polyradiculoneuropathy* (Guillen-Barre syndrome) can occur in the early stages of HIV infection, including acute HIV seroconversion disease. *Mononeuritis multiplex* , affecting cranial or peripheral nerves, can also occur early in the disease and be caused directly by HIV or CMV co-infection. More often, patients develop *peripheral neuropathy* , which is mainly sensory and affects the lower extremities. It is usually associated with HIV-associated DSPN, but may also be caused by nucleoside reverse transcriptase inhibitors such as stavudine, didanosine, zalcitabine, or other potentially neurotoxic drugs such as isoniazid, especially when multiple such agents are used in combination.

Less commonly, a patient may present with *progressive HIV polyradiculopathy* late in the course of the disease, and the main differential diagnosis is polyradiculitis associated with CMV, herpes simplex virus (HSV), or varicella zoster virus (VZV). Meningeal inflammation due to tuberculosis, neurosyphilis, cryptococcosis, and lymphoma can also result in damage to multiple spinal nerve roots. Damage to the spinal cord can be a consequence of myelitis associated with acute HIV or CMV seroconversion disease. More insidious spinal cord disease with predominant involvement of the posterior column may be caused by tabes dorsalis, subacute combined brain degeneration, or HIV-associated vacuolar myelopathy. Proximal myopathy with patchy red fibers on muscle biopsy associated with zidovudine-induced mitochondrial pathology. Treatment of neuromuscular syndrome is mainly symptomatic. It is necessary to identify and remove the harmful drug.

HIV-infected patients may also develop a stroke-like syndrome consisting of the sudden onset of focal neurologic dysfunction, such as weakness, loss of sensation, and impaired vision or speech. Because patients live longer on HAART and many develop hypertension and metabolic complications, including diabetes, hypercholesterolemia, and hypertriglyceridemia, they are at risk for cerebrovascular disease. This can take the form of ischemic stroke and intracranial hemorrhage. Clinical history should be reviewed for injecting or recreational drug use, hypertension, diabetes, hyperlipidemia, and smoking. Blood should be checked for platelet count, kidney function tests,

fasting glucose, cholesterol, triglycerides and VDRL. Blood culture, electrocardiogram, and echocardiogram should be performed if a cardiac source of embolism is suspected. Younger patients should also be tested for antibodies to cardiolipin and lupus anticoagulant. To differentiate an ischemic stroke from a hemorrhagic one, it is necessary to conduct a CT scan of the brain.

Management should follow the same principles as for normal stroke patients. Be admitted to an organized stroke program with stabilization of vital functions, prevention of complications such as aspiration or deep vein thrombosis, and early rehabilitation. To prevent stroke recurrence, the underlying causes should be treated appropriately, and patients with ischemic stroke should be given aspirin as secondary prophylaxis.

Recommended Books:

1. Neurology: National textbook / I.A. Hryhorova, L.I. Sokolova, R.D. Gerasimchuk and others; under the editorship I.A. Hryhorova, L.I. Sokolova. - K.: VSV "Medicine", 2014. - 640 p. + 32 p. color incl.

2. Shevaga V.M. Neurology: a textbook / V.M. Shevaga, A.V. Payenok, B.V. Expensive – 2nd ed., revision. and additional - K.: Medicine, 2009. - 656 p.

3. Vinychuk S.M. Neurology: a textbook for students and interns of neurologists / S.M. Vinychuk - K.: Zdorovya, 2008. - 756 p.

Additional

1. Popp John A., Deshaye Eric M. Guide to neurology; lane from English V. Yu. Khalatova; edited by acad. N. N. Yakhno. – M.: GEOTAR-Media, 2012. – 688 p.