**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 LECTURES ON THE ACADEMIC SUBJECT

Faculty, Course: Stomatological, 3th year
Academic Discipline: **Neurology**

**Approved by:**
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Head of the Department  Anatoliy SON

**Developers:**
Son Anatoliy Serhiiovych, PhD, DSci, Professor, Head of the Department
Dobrovolskyi Vasyl Vyacheslavovych, PhD, Associate Professor
Babych Eliza Vitaliivna, Assistant Professor
Hnatiuk Iryna Mykhailivna, Assistant Professor
Sarazhyna Kateryna Sviatoslavivna, Assistant Professor
Serbin Ihor Volodymyrovych, Assistant Professor
Yarovaya Ksenia Oleksandrivna, Assistant Professor

## LECTURES

*Lecture No. 1*

**Topic: Vascular diseases of the brain and spinal cord**

**Actuality of theme.**

Vascular diseases of the brain make up from 30 to 50% of diseases of the cardiovascular system. In their structure, the leading place belongs to acute disorders of cerebral blood circulation, which affect a large number of the population in all countries of the world. For every 100 million inhabitants, there are about 500 thousand strokes and cerebrovascular crises per year. According to the WHO, mortality from strokes is 12-15% of total mortality, i.e. it ranks 2nd-3rd after heart diseases and malignant tumors. A year after a stroke, 38-40% of patients die, within 5 years - 69%. The vast majority of patients remain permanently disabled and only 18-20% return to work. Therefore, the issues of prevention and treatment of vascular diseases of the nervous system are not only of medical, but also of great social importance. And the sudden occurrence of an acute violation of cerebral blood circulation and the need to provide emergency medical care require doctors of any specialty, and especially family doctors, to know this pathology.

Entire lectures

**Educational :**

- To acquaint students with the etiology and pathogenesis of HPMK (1st degree of abstraction)
- During the lecture, provide students with knowledge of the classification of HPMK, the main clinical symptoms of strokes (2nd degree of abstraction)
- On the basis of the lecture material, provide students with the opportunity to master the skills of diagnosis and treatment of hemorrhagic and ischemic strokes and transient disorders of cerebral circulation (3rd degree of abstraction)

**Educational :**

- Aimed at the development of a professionally significant personality substructure ;
- Education of modern professional thinking in students ;
- Ensuring students learn the leading importance of domestic clinical, scientific and pedagogical schools, including Odesa, which made a significant contribution to the study of cerebro-vascular pathology;
- Acquisition of deontology and medical ethics skills by students.

**Plan and organizational structure of the lecture**

| No | The main stages of the lecture and their content | Goals in levels of abstraction | Type of lecture, methods and means of activation of acquirers, equipment | Time allocation |
|---|---|---|---|---|
| AND<br>1.<br>2. | Preparatory stage<br>Setting an educational goal<br>Providing positive motivation | <br>AND<br>AND | According to the publication "Methodical recommendations for planning, preparation and analysis of a lecture" | 5%<br>(5 min) |
| II | The main stage | | | 85% |

| | | | | |
|---|---|---|---|---|
| 3. | Presentation of lecture material according to the plan:<br>– Actuality of theme<br>– Definition<br>– Classification<br>– Etiology and main links of pathogenesis<br>– Clinical picture<br>– Diagnostics<br>– Leading syndromes and differential diagnosis<br>– Evaluation of the severity of the course<br>– Treatment<br>– Prevention | <br><br>II<br>II<br>II<br>II<br><br>II<br>II<br>II<br><br>II<br>II<br>II | Slide presentation of lecture material<br><br><br><br>Extracts from the medical histories of patients.<br>Excerpts from clinical protocols of the Ministry of Health of Ukraine on providing medical care to patients. | (75 min) |
| III<br>4.<br><br>5.<br>6. | The final stage<br>Summary of the lecture, general conclusions<br>Answers to possible questions<br>Tasks for independent preparation | <br>III<br><br>III<br>III | List of references, questions, assignments | 10%<br>(10 min) |

**Content of the lecture material**

According to the current International Classification of Diseases of the Tenth Revision, vascular diseases of the brain are classified not in Class VI "Diseases of the Nervous System", but in Class IX "Diseases of the Circulatory System", and are considered in headings 1.60-1.69, which are included in the block "Injuries of Vascular Diseases" the brain does not exist in MKH-10, they are a syndrome of cardiovascular diseases. This means that acute disorders of cerebral blood circulation (CBI) can occur in the event of dysfunction of various links of the complex circulatory system: the heart, which performs the role of a pump that ensures the rhythmic flow of blood into the vessels; endothelium of blood vessels and vascular content, which is understood as the amount, composition and properties of blood. Therefore, a cerebral stroke is not a local process affecting only the vessels of the brain, but a systemic vascular pathology.

**Classification of vascular diseases of the brain**

1) **Diseases and pathological conditions leading to HPMK**
2) **II . The main clinical forms of HPMK.**

A. Initial phenomena of insufficiency of blood supply to the brain
B. HPMK

1. Transient violations of the MK
   a) transient ischemic attacks
   b) cerebral hypertensive crises
2. Acute hypertensive encephalopathy
3. Membranous hemorrhages
   a) subarachnoid
   b) Epi - and subdural
4. Hemorrhage in the brain
   a) parenchymatous
   b) parenchymatous-subarachnoid
   c) ventricular
5. Brain infarction (not embolic)

a) in the case of pathology of the main arteries of the head
b) in case of pathology of intracerebral vessels
c) other etiology
6. Brain infarction (embolic)
a) cardiogenic
b) other etiology

B. Impairment of cerebral blood circulation is slowly progressive
1. Dyscirculatory encephalopathy
D. The nature of cerebral blood circulation disorder is not defined
D. Consequences of a previously suffered cerebral stroke

**Localization of the lesion**
1. Hemispheres of the brain
2. Brain stem
3. Ventricles of the brain
4. Multiple foci
5. The localization is not determined

**Character and localization of vascular changes**
A. Nature of vascular pathology
B. Localization of pathology

**Characteristics of clinical syndromes**

**State of working capacity**

Brain strokes

Small strokes are diagnosed in cases where signs of organic damage to the central nervous system persist for more than a day, but disappear within three weeks.

Hemorrhagic strokes can be arterial or venous, occur by rupture of blood vessels and by diapedesis. By localization, parenchymal, ventricular, subarachnoid, epidural and subdural hemorrhages are distinguished. Combined and combined hemorrhagic strokes (parenchymal-subarachnoid, parenchymal-ventricular, multiple, etc.) are common.

In this section, parenchymal and subarachnoid hemorrhages are considered.

Parenchymal hemorrhage

Cerebral hemorrhage is characterized by a history of hypertension, hemorrhagic diatheses, post-traumatic and congenital aneurysms, septic endocarditis, alcoholism; rapid development of clinical symptoms against the background of acute psychoemotional stress or physical overload, nowadays; significant increase in blood pressure; rare tense pulse; significant hyperthermia (especially when blood breaks into the ventricles of the brain), pronounced violation of vegetative and vital functions (cardiac activity and breathing); very strong headache; vomiting (sometimes "coffee grounds"); meningeal symptoms, floating movements of the eyeballs; hormetonia; convulsive attacks; hyperglycemia; changes in the blood characteristic of the "alarm reaction" of Selye's stress syndrome (aneosyphilia, lymphopenia, an increase in rod- and segmented-nuclear neutrophils, general hyperleukocytosis); compensatory significantly pronounced hypercoagulation (if the cause of hemorrhage is not hemophilia and other coagulopathy); facial hyperemia; congestive discs of the optic nerves against the background of hypertensive angioretinopathy; complication of venous outflow from the cranial cavity; dislocation of the middle structures according to ECHO-EG by 4-8 mm or more, blood in the cerebrospinal fluid; unconscious comatose state; cerebral edema clinic. Complications such as bedsores, pulmonary edema, thromboembolism of the pulmonary artery, etc. occur early. A

fairly frequent complication of hemorrhage into the brain substance is the breakthrough of blood into the ventricles, which are accompanied by a sharp deterioration of the patient's condition, hyperthermia, (40-41 $^0$ C) breathing, deepening of other vegetative disorders, the development of hormonal syndrome, which is manifested by a constant change in the tone of the limbs, when they are hypertensive states with a sharp dissolution pass into a hypertensive state. Paroxysmal increase in muscle tone, especially if it prevails in the extensors, is similar to defibrillation rigidity, which is observed not only in the case of blood breakthrough into the ventricles meningeal symptoms of leukocytosis in peripheral blood, smooth or pendulum-like movements of the eyeballs also appear.

The condition of patients with brain hemorrhage is very serious, most of them die. Mortality reaches 75-95%. The prognosis of hemorrhages in the ventricles of the brain is even more difficult.

With a favorable course of the disease, patients gradually come out of a comatose state, which turns into a soporose state. Consciousness is gradually restored, corneal and tendon reflexes appear. The patient begins to swallow. General brain symptoms gradually regress, and focal symptoms come to the fore. The restoration of movements begins gradually, first in the proximal parts of the limbs, in the leg, and then in the hand. Hemiplegia turns into deep hemiparesis. Muscle tone of paralyzed limbs is restored. In parallel with the restoration of movements, there is a restoration of sensitivity.

Diagnostics

It is very important for a practicing doctor to find signs of focal neurological pathology in a patient who is in an unconscious state as a result of a stroke. Often the task comes down to clarifying the existence of hemiplegia. Signs of the latter will be the absence of spontaneous movements in the arm and leg on the side of the paralysis, while there are such (often of the type of automated gesturing) on the other side. An incompletely closed eye, slow lowering of the raised eyelid, smoothed nasolabial fold, displacement of the corner of the mouth to the healthy side, symptom of "sail" on the side of paresis of facial muscles, external rotation of the paralyzed foot, symptom of dropping hip, atony of paralyzed muscles helps to determine the side of the paralysis. "muscles - because of which the hand raised up on the side of the paralysis falls faster ("falls like a whip"), the absence or significantly low expression of Kering's symptom on the paralyzed side, weak expression of the mimic pain reaction when the hypothesized and motionless half of the body is irritated, unilateral pathological reflexes.

The most informative research methods are computer and magnetic resonance imaging. Echoencephalography, ophthalmoscopy, and electrocardiography are highly informative and more accessible (especially in non-transportable patients). In all cases, the following are mandatory: bilateral tonometry, when an injury is indicated - craniography, general analyzes of urine and blood, biochemical blood tests (glucose, residual nitrogen, urea nitrogen, creatinine, coagulogram, hematocrit, blood serum osmolality, indicators of acid-base status, mineral substances (potassium, calcium, sodium), lipids, protein fractions, samples reflecting the functional state of the liver).

Sometimes there are indications for serological testing of blood and cerebrospinal fluid. Often, the question of differential diagnosis is clarified or determined after a lumbar puncture.

According to special indications, primarily to raise the question of the need for neurosurgical intervention, contrast methods of research (angiography, etc.) are also used.

Treatment of patients with acute cerebrovascular accident (ACC) should be based on a staged approach: pre-hospital stage, intensive care unit, neurological department, rehabilitation department, rehabilitation in sanatorium conditions and further dispensary observation with a continuous program of pharmacotherapy in conditions of secondary prevention and continued rehabilitation. This staged treatment significantly reduces the mortality and disability of patients with HPMC.

The pre-hospital stage includes the provision of medical care until the moment of urgent hospitalization of patients with HPMK in stroke, neurological or neurosurgical departments.

Prehospital care should be provided to patients in the first minutes, hours after the onset of a stroke. This care is mainly provided by specialized neurological teams, line ambulance teams, neurologists, therapists, general practitioners (family doctors).

According to methodological recommendations, modern principles of diagnosis and treatment of patients with acute disorders of cerebral circulation" - Kyiv, 2005.-63 p. The following standard is generally accepted for the organization of providing medical care to patients with HPMK at the pre-hospital stage.

2. Assessment of the condition of the respiratory tract, breathing, blood circulation
3. Restoring the patency of the respiratory tract, elimination of the sinking of the tongue. If necessary, tracheal intubation.
4. Inhalation of humidification of oxygen
5. Artificial lung ventilation (as indicated)
6. Puncture of the vein, installation of a catheter for intravenous infusions.
7. Determination of the level of glucose in the blood by an express method.
8. After providing primary medical care, the patient is urgently hospitalized in a neurological or stroke (if available) department.

This standard should take up to 30 minutes to complete.

Blood pressure at this stage should not be lowered below 200/120 mm Hg, and if it is higher, it should be lowered by no more than 15%-20%. For this purpose, labetolol 5-20 mg bolus is used, or IV drip constantly 2-5 mg per minute; propronolol 1-5 mg, bolus IV, metoprolol 5-10 mg bolus IV, esmolol 200-300 mg. In the absence of adrenoblockers, magnesium sulfate 25% - 10.0 - 20.0 ml IV, captopril 6-12.5 mg per os , klofelin 0.15-0.075 mg per os .

In case of arterial hypertension, vasopressor drugs should be administered - dopamine 5-20 mcg/kg/min IV drip, or (i) adrenaline 1-5 mcg/kg/min IV drip on the background of infusion therapy.

In the presence of a convulsive syndrome, intravenous administration of benzodiazepines (sibazone, midazolam) is indicated.

To prevent cerebral edema, the compression of the neck veins should be reduced, the head position should be raised by $20^{0}$-$30^{0}$.

After the measures taken, the patient is urgently hospitalized.

At the hospital stage, the patient should undergo computer tomography of the brain, ECG, if necessary - echocardiogram, blood analysis - hemoglobin, hematocrit, erythrocytes, leukocytes, platelets, blood glucose, plasma electrolytes, blood coagulation parameters, biochemical tests - urea, transaminases, creatinine Duplex scanning of the main vessels of the head (for 1-3 days) in ischemic stroke. If necessary, make an X-ray of the chest organs.

Correction of respiratory disorders is carried out (tracheal intubation, inhalation of an oxygen-air mixture, early activation of the patient, respiratory gymnastics, APC). Measures to stabilize the function of the cardiovascular system and blood pressure continue, for which the same drugs as at the pre-hospital stage continue to be administered. Normalize the water-electrolyte balance. In the presence of hydrocephalus, 300-350 ml of fluid is injected; in case of diarrhea, vomiting, hyperhidrosis and high temperature - the amount of liquid is increased.

Monitor the blood glucose level: if it rises above 10 mmol/l, insulin should be administered, if it falls below 2.8 mmol/l, a 10% glucose solution should be administered.

Body temperature is constantly monitored. When it increases, paracetamol 500-1000 mg, analgin 50% - 2.0-4.0 ml IV or IV, diphenhydramine 1% - 1.0-2.0 ml are administered. If these measures do not help and the temperature is maintained, then antibiotics are prescribed. Control of dysphagia is carried out (probing in the presence of bulbar phenomena, fight against meteorism, give laxatives, hypertonic enemas)

Prevention of complications is carried out - brain hydration (mannitol 25-50 g every 3-6 hours, 10% sodium chloride solution 50-100 ml, furosemide); pulmonary complications

(antibiotics, correction of blood gas composition); urinary tract infections (catheterization as indicated, general urinalysis and bacteriological examination of urine, if necessary, antibiotics are prescribed), bedsores, deep vein thrombosis (compression stockings, fraxiparin, fragmin 7-10 days, warfarin per os ). They monitor the improvement of the function of the gastrointestinal tract (metoclopramide).

Then they proceed to differentiated therapy of ischemic and hemorrhagic stroke.

Effective assistance for ischemic stroke includes a set of the following measures:

A) basic therapy, prevention and treatment of complications

B) improvement of brain perfusion

C) prescription of nootropic drugs

D) use of surgical treatment methods

Stabilization of brain perfusion is achieved by prescribing Cavinton 5-20 mg per 500.0 ml and an isotonic solution of sodium chloride intravenously for 3-5 days, Sermion 4-8 mg per 80-100 ml of an isotonic solution of sodium chloride intravenously for the first 3- 4 days, and then 5 mg (1 tablet) 3-4 times a day under blood pressure control.

Antiplatelet agents:

Acetylsalicylic acid 100-300 mg (per day)

Clopidogrel (Plavix) 75-300 mg (per day)

Trental 2%-5 ml (1 ampoule) per 250.0 ml of isotonic sodium chloride solution IV drip for the first 3-5 days

Anticoagulants

Fraxipalin (nodraparin) 0.3-0.6 ml once a day subcutaneously in the fold of the anterior-lateral abdominal wall.

Warfarin, phenylin (anticoagulants of indirect action) are prescribed a day before the withdrawal of fraxiparin. Drugs that intensify venous outflow from the cranial cavity.

Troxevasin 5.0 ml - 10% intravenous solution, or 1-2 g per day for the first 5-7 days of the disease, and then 2 capsules. 2-3 g per day for 2-3 months.

Escuzan 15-20 drops 2-3 times a day for 2-3 months.

Phlebodia 600 mg 1 pill per day continued

Hemodilution involves the goal of correcting blood viscosity under the control of hematocrit, which should normally be 30-35%. It is best to combine it with means that normalize or correct the functional activity of the myocardium and blood pressure.

For hemodilation, reopoliglukin or rheomecrodex is used in a dose of 150.0 ml to 400.0 ml IV drip 2 times a day, depending on blood pressure.

Contraindications to hemodilution are severe heart and kidney failure, sudden heart enlargement, unstable angina, hemorrhagic transformation of brain infarction.

Nootropic drugs

Cerebrolysin 10.0-60.0 ml IV drip on 250.0 ml isotonic solution of sodium chloride No. 20-25. Contraindicated in epileptic attacks.

Gliatilin 1 g intravenously or intravenously 3-4 times a day for severe course of the disease and 1 t 1-2 times a day for relatively mild course of the disease for the first 5-7 days, and then 1200 mg per day orally.

Semax 1% or 0.1% 2 drops in each nostril 3-4 times a day.

Glycine 1-2 g per day under the tongue

Entsefabol 2 pills or 10.0 ml of suspension 3 times a day Contraindicated in epileptic attacks.

Antihypoxants:

Tocopherol 5%-2.0 ml or 10%-1.0 ml intravenously 1-2 times a day or in capsules -

2 caps. x 3 years/ per day.

Mildronate 10% - 5.0-10.0 ml IV 1-2 times per day.

Emoxipin 1% - 15.0 ml per 20.0 ml isotonic solution of sodium chloride 1 r per day No. 10, then 1% - 5.0 ml IV No. 14.

Mexidol 100 mg per 1 kg of body weight IV.

Differential ateralia of hemorrhage in the brain involves a complex:

A) prescription of basic therapy, prevention and treatment of complications

B) stabilization of brain perfusion

C) appointment of differentiated drug therapy

D) use according to indicators of surgical methods of treatment

Differentiated drug therapy includes:

1. stimulation of hemostasis
2. decrease in the permeability of the arterial walls
3. inhibition of formation of fibrinolysis
4. the purpose of nootropics.
5. these directions of therapy are implemented by prescribing ethamsylate, epsilon-aminocaproic acid, antagonists of proteolytic enzymes (hordox, contrical).

**Ethamsylate (dicinone** ). The initial dose is 1-2 ampoules (a 2 ml ampoule contains 250 mg of stamzilat) intravenously or intravenously. Then 1 ampoule intravenously or intravenously after 4-6 hours. The total duration of treatment is 5-6 days.

It is possible to prescribe orally 2 pills (250 mg of etamsylate) after 6 hours. Duration of reception is individual. It is determined by the speed of sanitation of the cerebrospinal fluid.

Epsilon aminocaproic acid. 100-150 ml of a 5% solution is administered 1-2 times a day intravenously in 60 minutes. Within 5-7 days. Then - appoint per os 3 g after 6-8 hours. The total duration of treatment is up to 3 weeks.

**Counter-part** . On the first day, 20,000-30,000 IU per 300-500 ml of an isotonic sodium chloride solution are prescribed intravenously as a drip over a period of 90-120 minutes. In the next 5-10 days, intravenous drip of 10,000 units of 2 g per day in 200-300 ml of isotonic sodium chloride solution.

During treatment with kontrikal, amylase content in blood and urine, as well as trypsin in blood, which should not exceed normal values, are checked.

**Hordox.** It is prescribed on the first day intravenously at a dose of 500,000 units, and then 100,000-300,000 units every 2-3 hours.

In order to prevent blood clot formation in peripheral vessels, it makes sense to combine the drugs with the appointment of heparin 2500-5000 units subcutaneously in the front wall of the abdomen 2-3 times a day.

Constant laboratory control of blood coagulation indicators is carried out.

To prevent the development of angiospasm in case of subarachnoid hemorrhage, Nimotop (an active blocker of L- type calcium channels and transmembrane calcium influx) is recommended for 2 pills (one pill contains 30 mg of Nimotop) every 6 hours for 21 days; or nemotan (nimodipine) 2 pills every 4 hours for 14-21 days.

Early rehabilitation of patients with HPMK should begin as early as possible with specially trained medical personnel. From the first day, it is necessary to carry out passive rehabilitation to reduce the risk of developing countertactures, joint pain, bedsores, pneumonia, deep vein thrombosis and pulmonary artery thromboembolism. Passive rehabilitation includes massage of large muscle groups and passive movements in all joints of the limbs. Procedures are carried out daily after washing and treating the patient's skin with moisturizing and, if necessary, antiseptic creams.

**Violation of spinal blood circulation**

Etiological factors :

A) **Pathology of the aorta.**

1. Atherosclerosis of the aorta, which is characterized by the development of symptoms of insufficient blood supply to the lower extremities (Lehrish syndrome, intermittent claudication, ischemic neuritis of the sciatic nerve).
2. Coarctation of the aorta: a symptom of arterial cerebral hypertension, segmental diapedesis hemorrhages ($C_I - C_{iv}$), sometimes Brown-Sécart syndrome, which is associated with ischemic phenomena in the spinal cord below the level of stenosis.

B) ***Anomaly of spinal vessels (aneurysms, varicose veins)***

Symptoms: sharp radicular pain, focal spinal symptoms that vary depending on the level of damage. The course with remissions and exacerbations is progressive.

B) **Osteochondrosis of the intervertebral discs.**

1. Acute ischemic disorders of blood circulation most often occur in the lower parts of the spinal cord, less often in the cervical region.

Provocative factors are light trauma, physical overexertion, sharp movements, alcohol consumption, cooling.

Occurs like a stroke over the course of one hour to one day,
sometimes at night while sleeping.
The clinical course depends on the level of damage.
Movement disorders are combined with sensitive (segmental in the ear area
coding, below - conductive type). Pathogenetic connections with compression and irritation of the anterior spinal or radiculomedullary artery (most often Adamkevich's artery), degeneratively - a changed disc or osteophyte.

Ischemia can proceed according to the type of transient blood circulation disorders or according to the type of heart attacks. In the acute period (day 3-5), the temperature and SRH may increase with normal leukocytosis.

The amount of protein in the cerebrospinal fluid increases, where erythrocytes and neutrophils can be found, which is associated with microhemorrhages in the ischemia zone. With mild degrees of damage, the regression of symptoms begins after a week or a little later (flabby paralysis becomes spastic, the level and degree of sensitive disorders decreases, the function of sphincters is restored). In the case of widespread heart attacks, the end is usually fatal: in the immediate period - as a result of joining heart disorders and respiratory disorders, in the distant period - from pneumonia, urogenital infection, intoxication due to bedsores and sepsis.

2. Slowly progressive spinal ischemia ( discicular ischemic myelopathy, or cervical discogenic myelopathy )

Etiology and pathogenesis: compression factor - blood vessels are more likely to be compressed than brain tissue; secondary adhesion membrane process at the level of the disc and beyond; changes in the vessels of the spinal cord as a result of proliferation of the intima and adventitia with subsequent secondary disruption of perimedullary and collateral blood circulation. It most often occurs in men at the age of 40-60. Provocative factors are injuries, hard physical work, sudden movements, intercurrent infections and surgical interventions. It is often localized in the cervical, less often in the lumbar region of the spinal cord. The course is often long, stable on the prosthesis for 5-10 years, sometimes progressive.

Main syndromes **:**

- amyotrophic (pronounced muscle atrophy of the proximal parts of the limbs, sensitive disorders);

- spastic-atrophic (sweetness and atrophy of one of the limbs with a transition to the opposite side with a gradual increase in atrophy in the hands and spasticity in the legs, sometimes with bulbar syndrome, sensory disorders are weak, segmental type, often - radicular pain, in the late stage - sphincter disorders) ;
- spastic (begins with numbness in the hands and feet, pain in the joints followed by the development of spastic tetraparesis, sensitive disorders increase in the caudal direction, in the later stages – pelvic disorders)

All forms of myelopathies are characterized by the predominance of motor disorders over sensitive ones.

There are no parallels between the intensity of the ischemic process and the degree of dystrophic changes in the spine.

These conditions should be differentiated from other sciatic diseases of the spinal cord, tumors, amyotrophic lateral sclerosis, syringomyelitis, myelitis, multiple sclerosis, hereditary diseases.

**Hemorrhagic disorders of spinal blood circulation** are divided into the following types :

1. Hematomyelia (Brown-Sécar syndrome, Minor's syringomyelia syndrome, anterior horn syndrome)
2. Hematorachis, most often occurs at the dissection of an arterio-venous aneurysm, injuries of the spine. A severe painful radicular syndrome with irradiation in all directions is observed. Occurs suddenly. Often there is a sharp scapular pain along the spine, headache, nausea, vomiting, light stupor, lethargy, lethargy. Appears : Kernig's symptom, often in combination with Laseg's painful symptom ; the rigidity of the occipital muscles recedes into the background. Focal spinal symptoms can appear at any time of compression of the spinal cord of varying severity.
3. Epidural hematoma most often occurs during the dissection of a vascular-spinal malformation. At the same time, there is a sharp local pain in the spine, accompanied by symptoms of spinal cord compression.

***Treatment***

It is carried out taking into account etiological factors and pathogenetic mechanisms of the disease.

In the acute period of ischemic spinal strokes, drugs are prescribed that eliminate swelling of the spinal cord (mannitol, furosemide, ethacrynic acid, glycerin), normalize heart activity and blood pressure, improve microcirculation and metabolism of brain tissue (reopoliglucin, cavinton, pentoxifylline, dipyridamole, nicotine preparations acids, nootropil, cerebrolysin), preventing thromboembolism (heparin).

In the recovery period, physical methods of treatment are used (diadynamic currents, electrophoresis of potassium iodide, dibazol, applications of paraffin, ozokerite), massage and physical therapy.

Surgical intervention is indicated for compression-vascular spinal disorders and unsuccessful conservative treatment.

Epidural hematomas require surgical treatment.

Treatment measures for patients with aortic lesions are determined jointly with surgeons.

**The cause of chronic disorders of cerebral blood supply is cerebral atherosclerosis, hypertension, diabetes, cervical osteochondrosis, arterial hypotension, rheumatism, etc.**

Risk factors are hypodynamia, irrational nutrition, chronic psycho-emotional stress, craniocerebral injury, change of weather, acute cardiac disorders, etc.

Initial manifestations of insufficiency of blood supply to the brain (NCM)

These are such disorders in which the patient complains more - headache, dizziness, noise in the head, memory impairment, reduced mental capacity. The duration of complaints is not less than 3 months. They intensify during mental work, staying in a stuffy room. There are no focal changes in the central nervous system. During special tests, a violation of the GNI is revealed. Depending on the semiotics, there are three variants of the clinical course of PPNKM: preclinical, clinical and paroxysmal.

Preclinical (asymptomatic) course - unstable blood pressure, signs of vegetative-vascular dystonia: acrocyanosis, acrohyperhidrosis, persistent red dermatographism, paleness or redness of the skin, tremors of the fingers, eyelids, invigoration of tendon reflexes.

Patients do not present subjective complaints.

Signs of insufficient blood supply to the brain are revealed only during functional EEG tests - changes in the bioelectric activity of the brain, hypersynchronous and desynchronous types of EEG.

On REG - an increase or decrease in the tone of the vascular wall, lability of pulse waves, interhemispheric asymmetries, difficulty in venous outflow.

On the ECG, sinus arrhythmia, violation of the depolarization phase, displacement of the ST segment and T wave are often present.

Changes in biochemical indicators and an increase in the content of cholesterol, triglycerides, and lipoproteins. Clinical (permanent) variant (variant course of PPNMK).

Subjective signs of insufficient blood supply to the brain appear - headache, dizziness, noise in the head, memory disorders, reduced mental capacity, etc. These complaints intensify during mental stress, especially in conditions of hypoxia or overfatigue, poor sleep. After rest, the patient's condition improves or completely normalizes. Objectively, subcortical reflexes, abdominal asymmetry, revival of tendon and periosteal reflexes can be observed. In patients, signs of general vascular disease are found: coronary cardiosclerosis, hypertrophy of the left ventricle of the heart, changes in the vessels of the fundus (angiopathy), symptoms of atherosclerotic damage to other areas. The diagnosis is also confirmed by changes in brain biopotentials, REH and biochemical indicators.

The paroxysmal course of PPNKM occurs mainly in patients with vegetative-vascular dystonia, arterial hypertension, and much less often - in the case of atherosclerosis of vessels.

Vegetative-vascular paroxysms of the cephalic, vestibular, syncopal, sympatho-adrenal, vago-insular and mixed type dominate.

The diagnosis of PPNKM is made on the basis of complaints, the clinical picture of the disease, as well as based on the data of electrophysiological and laboratory research methods. It is important to identify symptoms of general vascular disease: atherosclerosis, atrial hypertension, vegetative-vascular dystonia.

**Slowly progressive disorders of blood supply to the brain - dyscirculatory encephalopathy** .

At the same time, there are all signs of NMK, micro-signs of damage to the central nervous system (anisoreflexia, anisocoria, reflexes of oral automatism, convergence paresis, etc.). These are the initial manifestations of dyscirculatory encephalopathy.

There are three stages of encephalopathy according to the degree of severity of VND pathology and signs of focal damage. There may be dyscirculatory encephalopathy with a predominant lesion of the blood supply in the VBB or in the carotid system. The following phenomenological options are distinguished: with hypothalamic crises, syncopal states, transient disorders of cerebral blood supply, permanent or acute psychotic disorders with a predominance of intellectual-mnestic or emotional disorders.

Compensated, subcompensated, decompensated dyscirculatory encephalopathy of atherosclerotic, hypertensive, spondylogenic origin is very often the background against which strokes develop.

**Syndrome of damage to various vascular basins** .

Lesions of the middle cerebral artery. Complete occlusion of it leads to the spread of softening of the brain parenchyma, which is fed by this artery and its branch. A coma occurs, hemiplegia, hamianesthesia, hemianopsia, visual paresis, and aphasia develop in the peripheral cell.

**The syndrome of damage to the anterior cerebral artery** is manifested by the development of paresis of the leg opposite to the focus (or hemiparesis with a predominance of damage to the leg), apraxia, the occurrence of subcortical reflexes and changes in the psyche, characteristic of damage to the frontal lobe.

**Syndrome of damage to the posterior cerebral artery** . With cells in the basin of the posterior cerebral artery, the main diagnostic value is visual disorders - visual agnosia, hemianopsia with preservation of macular vision, square hemianopsia. A thalamic syndrome with hyperpathy, paroxysmal pain, sensitivity disorders, etc. may develop.

**The syndrome of damage to the basilar artery** is variable due to the peculiarities of its anatomical structure and the level of blockage of the vessel. Cells of softening capture the bridge. The initial symptoms of arterial thrombosis are malaise, nausea, dizziness, pain in the back of the head, tinnitus and headache. There may be oculomotor disorders, double vision, visual paresis, swallowing disorders. These symptoms are joined by limb paresis. With the acute development of the syndrome, general brain symptoms, deep coma, tetraplegia, hormetonia are expressed.

**The syndrome of damage to the vertebral artery** is characterized by short-term loss of consciousness, dizziness, vomiting, autonomic disorders, nystagmus. It is often combined with impaired blood circulation in the posterior lower cerebellar artery. At the same time, Wallenbern-Zakharchenko syndrome develops.

**Syndrome of damage to the internal carotid artery** . This type of pathology is characterized by remitting symptoms of ischemia in the branches of the carotid artery, the development of hemiplegia and aphasia (with a focus in the left hemisphere). Ophthalmoplegic syndrome is often present on the side of the blocked artery - reduced vision, up to blindness, on the opposite side - hemiplegia. Bernard-Horner syndrome may also occur on the affected side. Asymmetry of the pulsation of the carotid arteries on the neck (increased pulsation of vessels proximal to the blockage) and increased pulsation and increased pressure in the superficial carotid artery on the side of the cell are observed. Treatment of patients with PPNKM is of particular importance, since neurological disorders at this stage of the disease are reversible, and therefore timely therapy is simultaneously a prevention of acute disorders of cerebral circulation. A system of step-by-step treatment has been developed: polyclinic - hospital - resort - polyclinic, which includes dispensary supervision.

In the preclinical stage, if signs of vegetative-vascular lability are detected, a complex of preventive measures is used aimed at eliminating risk factors, stabilizing blood pressure in the presence of arterial hypertension, and compensating the initial manifestations of atherosclerosis. Measures aimed at improving the working and living conditions of patients are of great importance. It is important to observe the diet: you should avoid food rich in animal fats, cholesterol, salt, you should consume dairy food, fish, and boiled meat. Patients need to rest twice a year, sanatorium-resort treatment is recommended for them.

In the stage of clinical manifestations of the disease, outpatient or inpatient treatment is carried out taking into account the vascular pathology against which PPNKM developed. In the presence of neurosis-like complaints in the case of autonomic dystonia syndrome , sedatives

(bromine, valerian), intermittent courses of treatment with tranquilizers in small doses, as well as belataminal, diphenhydramine, and calcium preparations are recommended. In case of arterial hypertension, Halidor, Cinnarizine, Anaprilin, Obzidan, etc. are prescribed. If blood pressure is low, caffeine, tinctures of ginseng, Chinese lemongrass, pantocrin, levzei are recommended. Various methods of reflexology and electrosleep are used.

Lipotropic agents (methionine, cetamifen) play an important role in the treatment of vascular atherosclerosis. In case of hemorheological changes, aspirin, curantyl, trental, sermion, plavix, agapurine (orally) are prescribed.

Drug treatment is also aimed at improving neuronal metabolism. For this purpose, nootropics (piracetam, nootropil, encephabol, cerebrolysin, solcoseril, actovegin) are recommended.

A significant place is occupied by physiotherapeutic measures, physical therapy and sanatorium-resort treatment, improvement of working and living conditions.

Treatment courses must be carried out twice a year. In the case of arterial hypertension, maintenance doses of hypotensive agents are used to normalize blood pressure.

In case of a paroxysmal course, the PPNKM will prescribe L-adrenoblockers (piroxane), β-adrenoblockers (anaprilin, obzidan), antidepressants (amitriptyline, melipromine), antlepsin (orally).

From physiotherapeutic methods, the triad according to Graschenkov is used: intranasal electrophoresis of diphenhydramine, electrophoresis of areas of cervical sympathetic nodes and epigastric plexus.

Prevention.

Primary - a system of measures aimed at preventing the development of vascular disease of the brain: elimination of risk factors, improvement of working and living conditions, healthy lifestyle.

Secondary - involves early identification and registration of patients with PPNKM, their timely treatment in order to prevent the progression of cerebrovascular insufficiency. It is necessary to use the possibilities of day hospitals, as well as non-drug methods of therapy. A large role in the prevention of PPNKM is played by medical examination of patients, planned neurological examinations.

**Materials for student activation during lectures**

1. What are transient disorders of cerebral circulation ? Pathogenesis ?
2. The patient developed motor aphasia and right-sided spastic hemiparesis. Where is the lesion ?
3. The patient had a hemorrhage in the left leg of the brain. What symptoms are observed in this case ?
4. The patient suddenly felt an unbearable headache, after a few minutes a large epileptic attack occurred, after which the patient lost consciousness. Meningeal symptoms were detected. What happened to the patient?

**General material and methodological support of the lecture:**

1. Educational premises.
   - lecture room

2. Equipment.
   - desks
   - chairs
   - blackboard, chalk

3. Equipment
   - Stand " Basic neurological tools "
   - Electrified model " Conducting pathways of the brain and spinal cord "
   - Electrified model " Localization of functions in the cerebral cortex "
   - Epidiascope, slides
   - Neurological tools for patient examination

4. Illustrative materials
   - Application of electronic presentation
   - Educational tables " Blood supply of the brain and spinal cord "
   - Thematic patients

**Literature.**

**Basic**

1. Neurology: study guide / [I.A. Hryhorova, L.I. Sokolova, R.D. Gerasymchuk, A.S. Son, etc. ] edited by I.A. Grigorova, L. I. Sokolova - 3rd edition - Kyiv, Medical University "Medicine", 2020 - 640 p.
2. Topical diagnosis of pathology of the nervous system. Diagnostic search algorithms. Shkrobot S.I., Saliy Z.V., Budarna O.Yu. Ukrmedknyga, 2018. – 156 p.
3. Methods of examination of a neurological patient: teaching. manual / edited by L. I. Sokolova, T. I. Ilyash. - 2nd edition. - Kyiv: Medicine, 2020. - 143 p.
4. Emergency medicine. Emergency medical care: textbook / I.S. Zozulya, V.I. Bobrova, H.G. Roschyn and others / edited by I.S. Cuckoos - 3rd edition, trans. and additional - Kyiv. - VSV "Medicine", 2017. - 960 p.
5. Negrych T.I., Bozhenko N.L., Matvienko Yu.Sh. Ischemic stroke: secondary inpatient care: 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. Strecken, Richard P. Hobson .- "Medicine", 2021 . - 642 p.
5. Medical Books On-line Library ( Neurology ) – free download
http :// medbookshelf . info / category / neurology /

**Electronic information resources**

Clinical guidelines in neurology. (Order of the Ministry of Health of Ukraine No. 487 dated August 17, 2007) https ://zakon.council.gov.ua / rada / show / v 0487282-07# Text

*Lecture 2.*

**Topic: Main neurostomatological diseases and syndromes. Neuropathy of the trigeminal nerve and its individual branches. Iatrogenic neuropathies of the trigeminal nerve.**

**Relevance of the topic** : Acquaintance with the problem of neuropathy of the trigeminal nerve and its individual branches is relevant due to the fact that it can be observed at any age with odontogenic diseases. The ability to correctly establish a diagnosis and prescribe adequate therapy is essential for reducing the suffering of patients.

**Specific goals:**
**know:**
1. The main symptoms of neuropathy of the trigeminal nerve .
2. Differential diagnosis of headache
3. Treatment of neuropathy of the trigeminal nerve

*be able:*
1. To diagnose neuropathy of the trigeminal nerve .
2. To diagnose damage to individual branches of the trigeminal nerve
3. Prescribe headache treatment

**Plan and organizational structure of the lecture**

| No | The main stages of the lecture and their content | Goals in levels of abstraction | Type of lecture, methods and means of activation of acquirers, equipment | Time allocation |
|---|---|---|---|---|
| 1.<br>2. | Preparatory stage<br>Setting an educational goal<br>Providing positive motivation | <br>AND<br>AND | According to the publication "Methodical recommendations for planning, preparation and analysis of a lecture" | 5%<br>(5 min) |
| 3. | The main stage<br>Presentation of lecture material according to the plan:<br>– Actuality of theme<br>– Definition<br>– Classification<br>– Etiology and main links of pathogenesis<br>– Clinical picture<br>– Diagnostics<br>– Leading syndromes and differential diagnosis<br>– Evaluation of the severity of the course<br>– Treatment<br>– Prevention | <br><br>II<br>II<br>II<br>II<br>II<br>II<br>II<br>II<br>II<br>II | Slide presentation of lecture material<br>Extracts from the medical histories of patients.<br>Excerpts from clinical protocols of the Ministry of Health of Ukraine on providing medical care to patients. | 85%<br>(75 min) |
| 4.<br>5.<br>6. | The final stage<br>Summary of the lecture, general conclusions<br>Answers to possible questions<br>Tasks for independent preparation | <br>III<br>III<br>III | List of references, questions, assignments | 10%<br>(10 min) |

**Content of the lecture material**

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

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

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

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

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

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

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

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

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

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

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

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

Branches depart from the mandibular nerve:

1 Buccal nerve innervating the mucous membrane of the cheek.

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

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

the small stony nerve (branches of the tympanic nerve from the glossopharyngeal nerve) fit to it. These fibers are interrupted at the node and go to the parotid gland via the auriculotemporal nerve.

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

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

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

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

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

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

Facial pain caused by damage to the trigeminal nerve system

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

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

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

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

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

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

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

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

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

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

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

Trigger factors - conditions or actions under which pain paroxysms occur. Most often, such factors are washing, eating, brushing teeth, opening the mouth, sometimes talking. Most patients, due to the fact that irritation of trigger areas occurs during eating, use the contralateral side during chewing. Therefore, over time, signs of myofascial pain syndrome may appear, which is superimposed on the neuralgic syndrome. This can make differential diagnosis difficult.

In some patients, as a result of irritation of the precentral gyrus, spasms of facial facial muscles (more often - the muscle that raises the corner of the mouth) occur.

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

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

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

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

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

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

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

Due to the fact that swallowing is a strong trigger factor, suppositories with carbamazepine ( 0,1 г) are offered, which are quickly absorbed and therefore the analgesic effect occurs faster. Drugs that act on the opiate mechanism of antinociception are used: sodium oxybutyrate, phenibut ½ tablet at night; antidepressants (use a tricyclic antidepressant - amitriptyline 25 mg 3 times a day during the entire period of exacerbation); tranquilizers: relanium (diazepam, sibazone, seduxen). The use of these drugs is especially indicated in the presence of a phobic syndrome of waiting for an attack. The optimal dose is 1 tablet 2 times a day, or 2 ml of a 2% solution intravenously in an isotonic sodium chloride solution.

Apply action to trigger areas: lubricate the skin of the face with 5% anesthetic or 5% lidocaine ointment, alcohol tincture of water pepper. The nerve is decompressed in the bone

canals (infraorbital, mandibular). For this purpose, dehydration is used (furosemide - 1 tablet in the morning once every three days; verospiron - 1 tablet every other day; eufilin - 5-10 ml of 2.4% solution intravenously slowly);

Neurotrophic agents (thiamine - 1 ml intramuscularly every other day No. 10; Neurovitan - 1 tablet 2 times a day for 1 month; Nootropil - 5 ml intravenously daily No. 10); To improve blood circulation (trental - 1 tablet 3 times a day, curantyl - 1 tablet 2 times a day, nicotinic acid - 1 ml intramuscularly daily No. 10).

In case of irritation of vegetative formations of the face, use beloid, belataminal - 1 tablet 2 times a day; nicotinic acid - 1 tablet 3 times a day; spasmolitin - 0,1 г3 times a day.

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

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

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

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

Odontogenic neuralgia of the trigeminal nerve

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

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

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

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

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

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

Post-herpetic lesion of the branches of the trigeminal nerve

Recently, an increase in the incidence of herpetic lesions of the nervous system has been noted. Herpes zoster virus resides in the sensitive ganglia of the nervous system, in particular, in

the Gasser's ganglion in an inactive state before its activation, which may be due to unfavorable factors - a decrease in immunity, malignant neoplasms, the use of immunosuppressants, the presence of a competing infection. Acute herpetic infection is more often observed in the elderly, regardless of gender, in 45% of cases the first branch of the trigeminal nerve is affected.

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

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

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

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

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

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

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

In the neuritic (acute) stage of the disease, antiviral agents are prescribed: zovirax (5-10 mg per 1 кгbody weight intravenously in 100 ml of isotonic sodium chloride solution, or in 0,4 гtablets 2 times a day for 5-7 days, or in the form of an ointment for external use ), valtrex (1000 mg 3 times a day daily for 7 days), rimantadine ( 0,5 г4 times a day for 5-7 days), bonafton and floral in tablets or ointment for external use, deoxyribonuclease (0.2% solution for eye drops or in the form of inhalation for 10-12 minutes 2-3 times a day), interferon (for instillation in the eyes and nose 2 drops 3-4 times a day). To stimulate the formation of endogenous interferon, curantyl ( 0,025 г1 time per day), dibazol ( 0,05 г2 times per day), midokalm ( 0,05 г2 times per day) is used. For pain relief, dimexide with novocaine in a ratio of 1:3 is used for compresses, analgesics (2 ml of 50% analgin solution intramuscularly up to 2 times a day, baralgin). In case of pronounced pain syndrome, the introduction of lytic mixtures (2 ml of 50% analgin solution, 1 ml of 1% diphenhydramine solution, 2 ml of 0.5% novocaine solution, 1 ml of 2.5% aminazine solution) is indicated.

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

During the development of post-herpetic neuralgia, it is inappropriate to use anticonvulsants, because they are practically ineffective; quartzization, as it leads to swelling and

burning of tissues, which will only worsen the condition of patients; dyes that do not have antiviral activity (methylene blue, etc.).

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

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

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

Neuralgia of individual branches of the trigeminal nerve

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

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

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

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

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

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

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

If there is a pronounced pain syndrome, a complex powder is prescribed (dibazole - 0,005 г, thiamine - 0,005 г, nicotinic acid - 0,03 г, ascorbic acid - 0,3 г, glutamic acid - 0,3 г, spasmolitin - 0,1 г). Take 1 powder 2 times a day or 2 powders together at night, antihistamines, tranquilizers, antipsychotics.

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

Neuralgia of the ototemporal nerve, which belongs to the III branch of the trigeminal nerve, contains sensitive and secretory fibers for the ear ganglion, connected by anastomoses

with the facial and other nerves (Frey's syndrome). This disease was first described by L. Frey in 1923 under the name "auriculotemporal nerve syndrome".

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

The main manifestations of neuralgia of the ototemporal nerve are paroxysmal pain syndrome with signs of autonomic dysfunction during the consumption of solid, spicy, sour, spicy, sweet food, and sometimes even when mentioning it, as well as in the case of emotional and physical factors (smoking, hypothermia or overheating of the body, stressful conditions, functional disorders of the nervous system).

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

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

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

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

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

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

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

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

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