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DEPARTMENT OF PSYCHIATRY WITH NEUROLOGY, NEUROSURGERY AND MEDICAL REHABILITATION

METHODOLOGICAL DEVELOPMENT ON PRIVATE NEUROLOGY AND NEUROSURGERY.

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Reviewers:
Astakhova Z.T. - Head of the Department of Internal Medicine No. 4, Doctor of Medical Sciences, Professor

Totrov I.N. - Head of the Department of Internal Medicine No. 1, Doctor of Medical Sciences, Associate Professor

Violation of cerebral circulation (chronic, strokes, transient-transient ischemic attacks).

The purpose of the lesson : to study disorders of cerebral circulation, their classification, etiology, pathogenesis, clinic, differential diagnosis, treatment based on modern data.

The student should know:

1. Which includes the name of the cerebrovascular accident;
2. Classification of vascular diseases of the nervous system;
3. The clinical picture of ischemic strokes;
4. The clinical picture of hemorrhagic strokes;
5. Understanding of transient ischemic attacks;
6. Encephalopathy;
7. Modern methods of diagnosing cerebral circulation disorders;
8. Modern methods of treatment of vascular diseases of the nervous system.

The student should be able to:

1. Examine the patient and identify signs of cerebrovascular accident;
2. To be able to interpret the results of additional studies in the differential diagnosis of vascular diseases;
3. To prescribe undifferentiated or differentiated therapy for patients with vascular diseases.

Chronic cerebrovascular accident

Etiology and pathogenesis . Chronic insufficiency of cerebral circulation (CHF), or chronic cerebrovascular insufficiency (CHF), is the most common cerebrovascular pathology . It usually occurs against the background of vegetative-vascular dystonia, as well as general cardiovascular diseases. More often it is atherosclerosis, hypertensive disease and their combinations, diabetes mellitus, vasculitis is possible with systemic diseases of the connective tissue (lupus erythematosus, syphilis and other diseases accompanied by vascular lesions, blood diseases leading to an increase in its viscosity (erythremia, macroglobulinemia , cryoglobulinemia .. etc.) Marked and some other forms of pathology can lead to changes in general and local hemodynamics and on this background to the batch, and further chronic gradually about progressive manifestations of cerebrovascular disease are noted

step HSMN:. initial manifestations of brain ovogo circulation . and encephalopathy in this initial manifestations insufficiency of cerebral blood supply ychno occur when blood flow to the brain is less than 45-30 ml / min at constant admission of blood to the brain in the range 35 to 100 g / min evolves encephalopathy critical recognized regional blood flow. in the brain to the limit ah 19 ml / 100 g / min is the functional threshold of blood supply to the brain, at which the functions of the corresponding parts of the brain are already impaired. When regional arterial blood flow in the brain is reduced to 8-10 ml / 100 mg / min (infarction threshold of the blood supply to the brain), nerve cell death occurs. Cessation of blood flow in the brain for 5-8 min leads to time - vitiyu irreversible changes therein.

In the pathogenesis of CHSMN are important: 1) morphological changes in the extra- and intracranial parts of the main vessels of the head, as well as cerebral vessels and their dysfunctions; 2) reduction COMPENSATION - the beaten features of collateral circulation; 3) failure of self recharge - lyatsii cerebral circulation; 4) disorders of the central hemodynamic - Miki; 5) changes in the rheological properties of blood.

Risk factors. Among the risk factors HSMN can be marked - us smoking, alcohol abuse, physical inactivity, obesity. The facts - the risk of the frames should be attributed to genetic predisposition Patolo - cal conditions and diseases that can lead to chronic and acute cerebrovascular events. Among such diseases is particularly great importance hypertension, secondary, in particular - NOSTA renal, hypertension and atherosclerosis.

One of the leading risk factors for cerebrovascular patho - ogy is vegetative-vascular dystonia. For it is also possible hereditary predisposition, which displays in the phenotype provoke the severity and duration emotiogenic stress will repay - consequence, the presence of endocrine imbalance.

A very important risk factor for HNMK is pozhi - Loy age. This entails that during aging in connection with developing gam - Misia morphological and functional vascular changes, Nara - sheniem reduced metabolic capacity of the organism to ADAPT - tion. Environmental factors in the conditions of limited adaptive capacity of the aging of the body easier to cause "damage" its ADAP - , Gravitational-adaptive mechanisms and often lead to the development of - bolevany cardiovascular system than in young and middle-aged people. The major structural changes occurring at older - NII in large arterial trunks include sclerotic change the inner shell layer muscle atrophy, reduction of collagen fibers. These changes lead to a decrease in elasticity, extensibility and the appearance of rigidity of the vascular wall. Vozras - tnoj subject to restructuring and capillary network. It decreases with age - Xia number of functioning capillaries per unit volume of the tissue. According to electron microscopy with a thickening ba knitting membrane capillaries collagenization fibrils, reduction dia - meter long pinocytosis reduction activity. These changes lead to a decrease in the intensity of transcapillary exchange contribute to disruption of tissue oxygen supply and gi - poxvirus.

With aging, weakened reflex reactions in response to irritate - of baroreceptors carotid sinus and the aortic arch, disturbed function - onirovanie pressosensitive

mechanism of regulation of blood PRESSURE - Nia that promotes reflexogenic hypertension in individual STAR - Sheha age. In experimental studies have shown that when a hundred - rhenium changing regulatory influence of the brain on the cardiovascular system.

Initial manifestations of cerebrovascular insufficiency

Initial symptoms of cerebrovascular insufficiency (NPNMK) represent early stage HNMK - consequence vegetative - tively dystonia, early manifestations of atherosclerosis, arterial - hydrochloric hypertension and some other forms of vascular pathology. Mark - of NPNMK not customary, but possible. Manifest NPNMK Nepo - stand the feeling of heaviness in the head, sometimes short-term head - whirling feeling of instability when walking. The character - us fatigue, loss of memory, thinking pace rasstroyst - wa sleep. Occasional headache and noise in the head are possible. NPNMK often occur after the emotional and physical Perrin - conjugation, the use of alcohol, under the influence of unfavorable meteorological - rologicheskih factors.

In the neurological status, signs of vegetative- vascular and emotional lability can be detected , convergence failure , moderately pronounced symptoms of oral automatism are possible . Neuropsychological examination of the patient normally does not identify - that slow thinking, especially in the process of solving the intel - tual tasks, without qualitative changes in higher mental functions. The increasing manifestations of NPNMK lead to the development of the next stage of CHNMK - discirculatory encephalopathy.

Encephalopathy

Encephalopathy is a diffuse, multifocal lesion of the brain, in which, due to various reasons (except for infectious and inflammatory processes in the brain), there are not only subjective complaints of headache, fatigue, memory loss, etc., but also objective signs of multifocal or diffuse cerebral organic Patolo - gies that can be identified in the neurological and neyropsiholo - cal examination of the patient.

Encephalopathy (DE) due to chronic untill - sufficient cerebral circulation, causing diffuse measurable - neniya in brain tissue and scattered neurological focal mikrosimptomatiku usually in combination with emotional lability and intellectually - mnesticheskimi disorders the severity of which is variable, but tends to a gradual buildup. DE usually develops against the background of general vascular pathology. In this regard , the following forms of DE are distinguished: ***atherosclerotic, hypertensive, venous, and*** often DE is of a mixed nature.

In the words of JA distinguished 3 stages of development of its clinical car - slime. In the stage, the ability to work in most cases is preserved, in the // *s t a g e* it is reduced to one degree or another, and in the /// *stage the* patient is usually disabled. In the process of DE diagnosis must be considered somatic status, data REG, Doppler ultrasound, ophthalmoscopy, with - standing coagulation - antisvertyvayuschey blood system and, in some cases, and the results of CT - or MRI studies.

Diagnostics of the chronic vaso-cerebral insufficiency

When diagnosing CHFN, carefully collected information about complaints, anamnesis, in particular information about the course of the disease, as well as the results of a targeted somatic, primarily cardiological examination of the patient and, of course, a complete neurological examination, contribute to the diagnosis . Since CHMN usually manifests itself against the background of a general disease of the cardiovascular system, it is necessary to identify this disease. In the process of examining a patient with suspected CHFI, data that are important for diagnosis can be obtained by X-ray examination of the heart, the aortic arch and its branches, ECG, REG, USG, ophthalmoscopy, CT and MRI, PEG, in some cases, especially when / deciding on surgical correction of revealed cerebrovascular insufficiency,
sha angiography of the GREAT vessels of the BELOVA and cerebral vessels. When examining, one should strive to identify the nature of the main task and the features of the pathophysiological process that led to ., Its stage and form. All this is necessary to carry out the most
effective treatment.

Treatment and prevention of chronic Hard Of eniya cerebral circulation

Chronic cerebrovascular insufficiency usually occurs against the background of vegetative-vascular dystonia and general cardiovascular diseases , primarily atherosclerosis, primary or secondary arterial hypertension, as well as connective tissue diseases, diabetes mellitus ,syphilis, blood diseases , etc. In this regard, the diagnosis is facilitated by the identification of these somatic
diseases. Treatment should be combined and at the same time; is aimed both at the disease leading to cerebrovascular insufficiency and at compensating for the existing deficit of cerebral hemodynamics and at improving the general processes in the cerebral tissue. Treatment can be conservative and surgical .

C o n s e r v a t i v e t r e a t m e n t . When HSMN important is work and rest, removal of emotional stress, proper nutrition, physical activity without significant congestion, therapeutic exercise, smoking cessation, treatment of the underlying diseases of the cardiovascular system. It is advisable to use sedatives , angioprotectors (prodectin, or anginin, nimodipine, etc.), antiplatelet agents (small doses of aspirin, stugeron, trental, courantil, etc.), nootropic drugs (nootropil, encephabol, cerebrolysin, semax, meclofenoxate, etc.) .). In hyperlipidemia, diet and hypolipidemic agents (probucol, enduracin, lovastatin, etc.) are indicated . According to the testimony - the pits should be hypotensive, anti-sclerotic treatment, treatment of diabetes, etc. In the treatment of antihypertensive drugs should monitor the status of blood pressure and prevent excessive it CNI - zheniya, and in severe hypertensive DE desirable that blood pressure was stable and maintained for a few higher-level warning is - rezhdayuschem cerebral ischemia in terms of self-regulation of cerebral disorders - of circulation. Of the vasoactive agents for DE , ergot preparations can be shown - dihydroergotoxin (redergin), nicergoline (sermion), dihydroergocristin. When expressed vegetovascular disto - SRI with a tendency to raise blood pressure medications

shown vinca - ka Vinton (vinpocetine), Oxybral (vincamine) and Ginkgo Biloba drugs (tanakan etc.).

With the trend towards progression of cerebrovascular pathology le - chenie should be carried out almost continuously, while through every 2-3 months is recommended to change the vasoactive drugs and nootropics.

When indicated used tranquilizers, antidepressants, vitamin complexes, drugs aimed at the treatment of akinetic-rigid syndrome, tremor, and other patogenetiches - Kie and symptomatic agents. Patients with dyscirculatory encephalopathy - patiey (DE) require dynamic medical supervision. When the DOE may be advantageous to care for the employment of the patient, and if necessary - and timely direction on MSEK for his solution - Niya issue of disability.

Surgical t r e a t m e n t . Surgical treatment is carried out mainly in the case of occlusion of the extracranial sections of the main vessels of the head, subclavian arteries. Operations on these vessels, as well as on the aorta, are usually performed by specialists in vascular surgery.

Acute disorders of cerebral circulation

Acute cerebrovascular accident (CVA) can be of the type of transient ischemic attack (TIA) and the Institute - Counsel.

Transient disorders of cerebral circulation

Hypertensive crises and transient ischemic attacks (TIA) are usually referred to as PNMK . They differ from the clinical features of stroke - cal picture, the difference is in fact conditional.

The causes, as well as the pathogenesis of PNMC and stroke, sometimes *do not* fundamentally differ; the difference between PNMK and minor ischemic stroke is more quantitative than qualitative.

The term "transient ischemic attacks" (TIA) include various discirkulatornaya brain rasstroyst - va, characterized by paroxysmal clinical manifestations, co - torye may be characterized by cerebral and focal neurologic - kimi symptoms expressed in varying degrees, but is limited - GOVERNMENTAL time 24 hours In. this can not be taken into account preserves - to sculpt a longer term pathological, but functionally unimportant focal neurological symptoms, such as a reflector - asymmetry of or extensor response in causing plantar reflex (Babinski's sign).

The introduction of imaging techniques (CT , MRI) into clinical practice has made it possible, not so rarely, to reveal small infarction and sometimes hemorrhagic foci in the brain tissue during PNMC . Whether - terature recent years indicates that these findings may be the Detect - wife about 25% of TIA [Awad V. et al, 1986;. Dennis O. et al., 1990; Hankel C., Warlow R. et al ., 1994]. This circumstance even led to the proposal to name such cases of PNMC as "cerebral strokes with transient symptoms", which did not receive support, since it only introduced confusion into the existing classifications. However, the TIA has its own characteristics and they should not include those comprising - Nia as systemic and non-systemic dizziness, fainting, seizures, migraine, multiple versions of epileptic seizures.

Thus, PNMC is an independent clinical form of acute cerebrovascular disease. The presence of TIA normally rastse - Niva as a risk factor for more severe forms of acute with - sudisto brain pathology - stroke.

TIA - the most common form of acute vascular Ministry of Health - the traction pathology. Published statistics on their frequency of - nizheny. They may not be reliable, since due OF SHORT - NOSTA emerging clinical manifestations, patients often do not form - are having to medical advice. Transient ischemic attacks described by various names: disrupting dynamic - of the cerebral circulation, Pre-stroke conditions, pretromboz, . Microinsult etc., but none of these names do not reflect the nature of the clinical phenomenon. The diversity of its clinical manifestations are due to - on a variety of pathogenetic mechanisms and unequal length - telnostyu, distribution and localization dyscirculatory YaV - tions. The peculiarities of the pathogenesis of this group on cerebrovascular - Rushen is usually customary to distinguish transient ischemic attacks and gi - peripheral arterial crises.

Etiology. The reasons for PNMK are manifold. Among them , lesions of the great vessels of the head and cerebral vessels are especially common . These include, first of all, atherosclerosis and hypertension , as well as their combination. Less frequent are the various forms of vascularization LTL (infectious, allergic infectious, and syphilitic al.) Or systemic connective tissue disease, leading to shock vessels (polyarteritis nodosa, thromboangiitis obliterans Bu nivartera-Buerger, systemic lupus erythematosus, etc.). In addition, the cause of PNMC can be blood diseases (various forms of anemia, polycythemia, hemophilia), myocardial infarction and cardiac disorders such as atrial fibrillation, paroxysmal tachycardia , Morgagni-Adams-Stokes syndrome, valvular heart disease, and atherosclerotic lesion of the aortic arch, my disease is mine, Takayasu's disease, coarctation of the aorta, etc.

The presence of PNMK should always be considered as a very significant risk factor for the development of stroke, and in each case of PNMK it is necessary to conduct a thorough examination of the patient, aimed at identifying its cause. The discovery of the process of examination of patients, re - bore the TIA, paroxysmal cardiac disorders, in particular violations of his work rhythm, fraught with a certain! difficulties, since the ECG obtained between such paroxysms is often without deviations from the norm. In these cases, it is desirable to carry out outpatient cardiac monitoring in the usual conditions for the patient in order to record the state of cardiac activity for some considerable time. Sometimes, to determine the functional state of the heart, it becomes necessary to conduct an ultrasound or radioisotope study.

Pathogenesis. Pathogenetic basis TIA may be segmental - tare or diffuse vascular spasm or stenosis of main head vessels and cerebral vessels, as well as disorders of the general hemodynamic - Miki, impaired oxygenation of the blood, changing its physico-chemical biochemical properties rejection atheromatous plaques microembolisms with polycythemia, myeloproliferative diseases and leukemias, breakdown of self cerebral blood flow, manifestations syndrome steal redistribution of blood while taking a hot bath, acute blood loss disorders neuroreflex mechanisms, particularly in the case of failure of self-regulation of

cerebral blood flow due to the sharp rise or pade - Niemi BP, slowing of blood flow due to violation of its physico-chemic - FIR properties (viscosity increase, increase agglyutsinatsii and aggregation of blood cells).

TIA can be provoked microthromboembolism, obuslov - lennymi decay sclerotic plaques located in the wall of cereals - GOVERNMENTAL vessels, in particular the main vessels of the head, as well as microthromboembolism arising in connection with rheumatic or septiches - Kim endocarditis.

It should be borne in mind that PNMK often occurs against the background of DE, may be a consequence of an acute increase in local ischemia in case of insufficiency of collateral circulation, a pronounced fall or rise in blood pressure, as well as in cases of small foci of hemorrhage accompanied by collateral edema. TIA in vertebrobasilar system may occur in the presence of a pathology or craniovertebral with cervical osteochondrosis in conjunction with vertebral artery atherosclerosis and occurring at the same reflex spasm of arteries or one of them.

C l i n i c a l m a n i f e s t a t i o n s . The clinical picture of PNMC is multivariate and is mainly determined by the localization, volume and characteristics of the pathogenesis of the pathological focus in the brain.

Transient ischemic attacks may occur in the form of paroxysmal state in which the lead is about - schemozgovaya neurological symptoms. This is how PNMK usually manifests itself as a hypertensive crisis.

Hypertensive cerebral crisis - a variant TIA occurring on the background of arterial hypertension, generally at a sharp rise of blood - first pressure, which can be triggered by physical or emotio - tional overvoltage alcohol intake, overheating, exacerbation - em renal disease, etc. When hypertensive crisis patient who searches. Vaeth severity in the head, which passes in the fast growing, first zaty - mammary and then diffuse headache conjugated with progressing - conductive tissue edema located in the cranial cavity, in particular with swelling of the brain. In this case, usually a pain in the heart, nausea, vomiting, often repeated, noise in the head, dizziness, weakness, emotional instability, accompanied by a pronounced VAZ - Thorne reactions, feeling short of breath, palpitations. Nered - to visual disturbances occur: feeling of indistinct outlines of the surrounding objects, their fragility, the mist before his eyes. During the culmination of the crisis, thought disorder, short-term loss of consciousness are possible . Sometimes identified paresthesia, pyramidal signs, elements - cops aphasia, dysarthria, koordinatornye disorders, meningeal signs. Clinical manifestations of essential hypertension are characteristic: high blood pressure, hypertensive retinopathy, myocardial hypertrophy, emphasis of the II tone on the aorta, etc.

For transient ischemic disorders of cerebral circulation, or ***transient ischemic attacks*** *(TIA),* focal neurological disorders persist for no more than 24 hours; cerebral pro - effects are possible, but they are moderate in severity and often occur in connection with the swelling of the tissues surrounding the area of ischemia.

When transient ischemic attacks phenomena distsirkulyatsii WHO - is possible in certain vascular region. By localizing the dis - circulation in the internal carotid artery

on the side protivopo - false pathological processes in the brain, are common paresthesia, disturbances of sensitivity may occur manifestations of central paresis of one of the extremities, palsy of brahiofatsialnomu type or hemiparesis, Inog - da opto-pyramidal alternating hemiplegia (with lesions vnut - renney carotid artery at the level of a discharge from her eye on the side branch lesion appears blurred vision, blindness, sometimes, on the opposite side - hemiparesis), possible local seizures. When left-sided localization of the pathological process may proyav - lyatsya aphasic disorders.

Transient disturbances in the vertebrobasilar system character - form a primarily dizziness, nystagmus, coordinates violation - tion movements and vegetative-vascular disorders. Often there noise in my head, "veil" in front of his eyes, photopsias, defects in visual fields of type quadrant or complete hemianopsia, occipital headache, nausea, vomiting, and sometimes diplopia due to dysfunction of the cranial nerves, enabling the movement of the eyeballs, violations sensitivity - telnosti on the face, often around the mouth. Possible elements bulbar syn - Dre. General weakness, weakness and rapid exhaustion are often noted. In the presence of cervical osteochondrosis, paroxysmal conditions of the TIA type in the vertebrobasilar system can be triggered by sharp turns or throwing the head back.

Acute hypertensive encephalopathy, which usually occurs in the malignant form of hypertension, is also commonly referred to as PNMC. A rise in blood pressure to very high numbers accompanied by a pronounced increase in venous pressure and usually diffuse cerebral edema, and at the same time, a pronounced general headache is characteristic, aggravated to a sharp one with coughing and sneezing, nausea, repeated vomiting, noise in the head, dizziness, bradycardia, meningeal phenomena. Paresis, coordination disorders, changes in consciousness, seizures are possible. In the fundus, there are pronounced manifestations of hypertensive retinopathy, signs of congestive optic discs are possible.

The frequency of PNMK can vary widely. PNMK is often preceded by cerebral circulation disorders of the type of stroke.

Treatment. With repeated PNMK, it is necessary, first of all, to clarify their causes and to treat the underlying disease. Also shown are therapeutic measures aimed at improving cerebral hemodynamics.

In cases of TIA, it is necessary to conduct courses of treatment with antiplatelet agents, angioprotectors, if indicated, anticoagulants are prescribed, reopolyglucin (400 ml), sulfacamphocaine 2 ml 2-3 times a day, 10-20 ml of a 2.4% solution of aminophylline are injected intravenously. All drugs also have an antiplatelet effect. It should be borne in mind that in the presence of signs of cardiac pathology, it is undesirable to administer large volumes of aminophylline fluid. Courses of treatment with drugs of nootropic action are advisable. If the stenosis of the internal carotid artery is more than 70%, the question of endarterectomy should be discussed.

In hypertensive crises, systematic treatment with antihypertensive drugs in adequate doses is necessary.

Ischemic stroke

Ischemic stroke arising from disorder gemodi - Namiki system in certain major vessel head or brain - Vågå vessel is HSMN threatening complication or consequence of developing acute thromboembolic vascular trunk and head vnutriche - Repnev vessels. In this case, an ischemic focus (cerebral infarction) is formed in the brain , characterized by the death of a portion of the brain tissue.

Ischemic stroke is developed in connection with the same pathological conditions that can lead to transient ischemic ata - cam (TIA) and mentioned previously. As with TIA, strokes occur more frequently in patients with atherosclerosis, hypertension or background By combining - Niya clinical manifestations of these diseases; cause of ischemic stroke may be heart disease, anemia, polycythemia, and Drew - Gia pathological conditions which predispose the general and local reduction of cerebral blood flow. When this syndrome symptoms may steal, which may manifest in two ways: 1) directly on - kradyvanie is the direct blood flow deficit due to stenosis of arteries and resulting reduction therein perfusion pressure due to failure of self-control; 2) secondary robbing, because - as is known as the ***Robin Hood syndrome*** - redistribution of blood, leading - present in the formation of the ischemic lesion.

Etiology and pathogenesis

Ischemic stroke may be preceded by transient cerebrovascular accident. It develops more often in a dream or shortly after waking up, after a hot bath, drinking alcohol. Provoke him significant differences in blood pressure, usually its decline, acute heart Nedo - sufficiency, atrial fibrillation. A gradual increase in neurological symptoms is characteristic for several hours, less often for a day (ischemic stroke "in progress"). But in '/ 5 cases, it develops sharply. This is typical, in particular, when it is caused by cerebrovascular embolism.

In the pathogenesis of ischemic stroke, the role of disturbances in the nervous regulation of vascular tone, stenosing processes (atherosclerosis, temporal arteritis, infectious granulomatous arteritis, Takayasu's and my-my's diseases, changes in the physicochemical composition of blood, primarily its coagulability and rheological properties) is important. Various forms of vasculitis and vascular malformations can also be the cause of ischemic stroke.

Focal symptoms in ischemic stroke often prevail over the cerebral, while their character is determined by the location and races - prostranennostyu cerebral infarction, the location of which corresponds to the area of blood supply to the vessel in which the violation occurred bloodstream. Ischemic strokes can be non-thrombotic (non-thrombotic softening), thrombotic and resulting from cerebral vascular embolism .

Non-thrombotic stroke occurs as a consequence of cerebrovascular insufficiency, when a critical decrease in cerebral blood flow occurs due to a violation of general hemodynamics or disruption of cerebral circulation self-regulation in the presence of stenosis or pathogenic tortuosity of a large extra- or intracranial cerebral vessel. It can be caused by prolonged angiospasm or slowing of blood flow due to increased blood viscosity, anemia, massive blood loss. In p; In the genesis of small-focal cerebral infarctions in patients with essential hypertension, it seems that the disruption of self-regulation

of the lumen of cerebral vessels, which increases during the period of pronounced changes in blood pressure , is important .

There is an opinion that an ischemic stroke, regarded as non-thrombotic , can also develop as a result of thrombus formation, in which the thrombus is recanalized by the time of examination.

The outcome of non-thrombotic strokes depends on the timing and degree of compensation for disturbed blood flow due to regression of manifestations of angiospasm, improvement of collateral circulation, increased blood pressure, and that on the possibility of reducing the permeability of the vascular walls and the severity of cerebral edema.

Thrombotic stroke develops as a result of thrombosis of the main arteries of the head and cerebral vessels, which is based on pathological changes in their walls, often caused by the formation of atherosclerotic plaques (endothelial damage, intimal proliferation, ulceration) leading to stenosis, i.e. narrowing of the lumen of the vessel, an increase in blood viscosity, a change in the protein coefficient in it due to an increase in the albumin content, as well as an increase in the coagulative activity of the blood, impaired central hemodynamics, in particular, a decrease in blood pressure, a slowdown in arterial blood flow in the basin of vessels found to be in a state of stenosis ... The thrombus, gradually increasing, can completely close the lumen of the vessel, causing its occlusion.

In the process of thrombus formation, ischemia and hypoxia increase in the basin of the affected vessel, which leads to the development of an infarction focus in it, characteristic of ischemic stroke.

Embolic (thromboembolic) stroke occurs when an embolus **enters the** lumen of the cerebral vessel, which is usually a particle of a disintegrating parietal thrombus in the heart cavity or arising from congenital and acquired valvular heart defects, in particular in the case of mitral valve stenosis, its prolapse or aortic defect, with rheumatic, bacterial endocarditis, with myocardial infarction, acute postinfarction heart aneurysms, cardiosclerosis and myocarditis, occurring with atrial fibrillation and the formation of parietal thrombi, with heart valve replacement, with atrial soma. Thromboembolic stroke is also possible with thrombophlebitis of the veins of the extremities, abdominal cavity, small pelvis in combination with congenital atrial or interventricular septum obstruction . The cause of cerebral embolism can also be a disintegrating sclerotic plaque in the ascending part or in the aortic arch, as well as in the great vessels of the head (arterio-arterial embolism).

Less embolism of cerebral vessels can arise in the case bronholegoch GOVERNMENTAL diseases, in particular when bronchiectasis, empyema, cavity, lung abscesses, malignant tumors, infectious common - tional diseases. The cause of cerebral thromboembolism can also be fatty or gas, usually air, embolism. Fat embolism of cerebral vessels is a possible consequence of a fracture of tubular bones or traumatic damage to the subcutaneous fat. Airy - naya embolism occurs when lung surgery, when applied pneumo - thorax, with caisson work, with traumatic injury of cereals - GOVERNMENTAL veins.

Iatrogenic embolism of cerebral vessels can arise during shunt - tion coronary artery angioplasty, carotid operations arte - riyah.

Cerebral infarction , usually occurs in ischemic stroke, nuclear explosion - wish to set up the white (it is called, and gray). He is represented by a pale, dryab - loi subjected to necrosis of the brain tissue, is a consequence of expression - conjugated and persistent violations of metabolism in neuronal and glial structures arising in ischemic stroke. In the center of the ischemic lesion (core infarct focus center) level of blood flow is below the threshold of myocardial infarction, whereas at its periphery, in March - nal area, blood flow corresponds to the functional or ischemic whom threshold.

Numerous variants of ischemic stroke are characterized by the development of cerebral infarctions of different localization and size. At the same lo - localization of foci and its dimensions are determined by the pool of the affected vessel. Furthermore, some embodiments are distinguished infark - comrade brain based on the individual, their characteristic features.

Ischemic stroke, leading to the formation of infarct in one hemisphere of the cerebrum, sometimes accompanied by a change of blood flow rate and inhibition of physiological activity in symmetrical structures opposite hemisphere that can determine formed - vanie therein a second (mirror) of the ischemic lesion or ***specular infarct brain*** .

Lacunar infarcts of the brain is called a heart attack, caused by - ny acute cerebrovascular ischemia by type - the first stroke to the formation of small infarction focus by transformed - yuschegosya over time into a small (0.5-2 cm) cyst - gap. In this case, histological examination often reveals a special type of vascular lesions - *lipogialinoz* small penetrating arteries, usually devel - vayuschiysya in patients with hypertension. Lacunar cerebral infarctions more often occur against the background of arterial hypertension or diabetes mellitus. They were described in 1901 by the French neuropathologist R. Mari (1843 - 1940). It should be borne in mind that, if the lacunar infarction of the brain affects the brain structures that have a clear functional significance (on - an example, in the rear thigh of the internal capsule), it may lead to expression - adjoint and bad poddayuschiysya- restoration of neurological deficit. However, lacunar cerebral infarction can occur in the form of a small stroke, PNMK, and sometimes it is asymptomatic.

In all types of cerebral infarction, histological examination first makes it possible to reveal the changes in the brain substance caused by ischemia, the nerve cells dying due to ischemia and necrobiotic changes in the neuroglia; vessels are more resistant to ischemia, although signs of dystrophy are revealed in them, in particular, stratification and vacuolization of the basement membrane, endothelial hyperplasia, and the disappearance of mitochondria. In the central part of the heart attack, the disintegration of the nervous tissue occurs. The organization of this necrotic focus ends with the formation of a gliomesodermal scar or (in the case of large sizes, infarction; focus) ends with the formation of a cystic cavity, which can sometimes be multi-chambered.

Ischemic strokes in the carotid basins are much more common than in the vertebrobasilar (4: 1). In the carotid basin , strokes caused by embolism are more common. Sometimes there is multiple embolism in the vessels of one or more basins. With thrombosis and embolism of the cerebral artery, the blood supply to the zone of its vascularization is disturbed. In this case, desolation of the capillaries

occurs, the invagination of the vessel walls into their lumen is possible, the unevenness of the lumen and the tortuosity of the vessels appear due to a change in their tone of cerebral edema.

In the pathogenesis of thromboembolism of cerebral vessels, their spasm due to mechanical stimulation of the receptors of the vascular wall by embolism (vascular spasm) is important . If, after the termination of the reflex spasm, the embolism moves further, then the blood flow in the proximal part of the artery is restored and becomes impaired only in its distal branches. In cases fragmentation embolus and moving it in a distal vessel or small branches occur infrequently small focal krovoi zliyaniya in previously ischemic tissue, causing formation *hemo rragicheskih (red) cerebral infarctions* and infarctions mixed type. Mr. emorragicheskie infarction lesions are usually small, they are red, localized, usually in the gray matter of the brain, the cortex or in Podkoren postglacial sites and more are due to thromboembolism. Red infarction can also occur in connection with the saturation of the ischemic focus with blood in cases of rapid activation of collateral circulation , which can be facilitated by an increase in blood pressure and large sizes of infarction zones . A similar transformation of a white cerebral infarction into red can be triggered by a surgical operation, during which blood flow is restored in a previously occluded great vessel. In an infected cerebral vessel embolus, the clinical picture of cerebral infarction is complicated by the development of an inflammatory process in the ischemic zone , manifested by the appearance of a metastatic encephalitis focus, meningoencephalitis, and brain abscess.

One of the causes of acute cerebrovascular accident may be the dissection of the walls of the common or internal carotid artery. It is usually accompanied by local pain and soreness of the artery, while pain can radiate to the area of the face on the side of the lesion, often on the same side there is a positive Horner symptom. In this case, angiograms can reveal a "rope symptom" (uneven narrowing of the lumen of the internal carotid artery extending to the intracranial region), tape-like occlusion - an uneven narrowing of the lumen of an artery in a small area, sometimes with detachment of the intima in this area, as well as an exit contrast material outside the lumen of the arte - Ree.

Research in recent years has led to a new stage in the understanding of the processes of damage to the nervous tissue during cerebral ischemia; thus they under repeated absence of direct identity between the concepts acute Fokal hydrochloric cerebral ischemia procedure and implies possibility hydrochloric reversibility of metabolic changes in the brain, and the concept of cerebral infarction, characterized by persistent morphological the defects - vol.

With *atherosclerosis* , extracranial vessels are more often affected. In this case, there is a certain pattern in the location of atherosclerotic plaques. Most often they are localized at the bifurcation of "common carotid artery and near the mouth of the vertebral arteries Nara. Sheniya cerebral circulation may be caused and occlusion b brachiocephalic trunk (innominate artery), subclavian arteries, aortic stenosis extracranial vessels involved in kro. - vosnabzhenii brain, reducing its blood supply can be an hour - the partially, and sometimes completely, offset by anastomosis of vessels located distal portions of stenosis in this

regard. CVA stenosis of major vessels often have the character of TIA or developed by a small stroke type clinical. picture violated - key's function of cerebral circulation in the basin affected magicians - . stralnogo vessel there may be patchy sometimes *in* connection with the cross-flow of blood through the collaterals of preserving the functions of the vessel into the zone of ischemia occur clinical manifestations nedostatochnos - minute blood flow and unaffected vessel basin *(the phenomenon obkra blowing).* The possibility of reduced collateral circulation in patients with concomitant nym defeat the main vessels of the head and intracranial vessels , which often manifests itself in old age.

Atherosclerotic changes in intracranial blood vessels, especially in the cerebral vessels located distal to the arterial circle of pain - Shogo brain, usually lead to more severe consequences and are often the cause of ischemic stroke, manifesting the development of cerebral infarction and severe persistent focal neurologic pathology.

Differences pathogenesis of ischemic stroke pathogenesis TIA on the type of transient ischemic attacks (TIAs) are not only quantitative - governmental consists in maintaining a longer focal nev - rologicheskih symptoms. Ischemic stroke represents a qualitatively special condition characterized by complex hemodynamic and metabolic changes in the brain tissue in the process insufficient - the accuracy of its blood supply and leading to the formation of irreversible morphological changes in the substance of the brain.

Studies in recent years have an idea about a hundred - diynosti hemodynamic and metabolic changes occurring in brain tissue at different stages of failure of its blood supply and leading to the development of changes in the neuronal pool, microglial activation, as well as a violation of the trophic maintenance of the brain.

The development of acute cerebral ischemia triggers pathochemical k'as - Cadenet reaction and causes changes in neuronal pool, astrocytosis, microglial activation and combined with them violation trophic ensure brain. All these changes lead to the formation of a brain stroke by two main mechanisms: necrotic death of apoptosis, or genetically programmed cell death.

Modern pathogenetic concepts allow us to present a diagram of the sequential stages of the "ischemic cascade" on the basis of causal relationships: 1st stage - decrease in cerebral ; 2nd stage - glutamate "excitotoxicity"; 3rd stage - intracellular calcium accumulation; 4th stage - activation of intracellular ; 5th stage - increased synthesis of NO and development of oxidative *c;* stage - expression of early response genes; 7th stage - p ischemia (reaction of local inflammation, microvascular and BBB damage); 8th stage - apoptosis.

The severity of ischemic stroke is determined, first of all, by the severity of the decrease in blood flow, the duration of pre-reperfusion ischemia. The area of the brain with the most pronounced decrease in blood flow (less than 10 ml / 100 g / min) - the "nuclear" zone of stroke - becomes irreversibly damaged within 6-8 minutes from the onset of the first clinical symptoms of cerebral ischemia. In this case, the nuclear zone is surrounded by ischemic, but still living tissue with a decrease in cerebral blood flow to 20-40 ml / 100 g / ml of "ischemic penumbra", or penumbra, in which energy metabolism is still preserved and only functional, but not structural changes.

The duration of the existence of the penumbra is individual for an individual with ischemic stroke and determines the temporal boundaries ("therapeutic window"), within which therapeutic measures can be most effectively carried out, aimed mainly at maintaining vitality and restoring functional ischemic penumbra, or penumbra.

The development of most of the cerebral infarction ends within hours from the moment the first signs of a stroke appear. The completion of the formation of the infarction focus lasts for 48–72 hours and longer from the influence of persistent cerebral edema and other long-term consequences of ischemia.

Thus, the therapy of ischemic stroke should be as early as possible, preferably in the first 3 hours of the disease, while being especially intense and pathogenetically directed during the first 3-5 days, constituting the most acute period of stroke.

With extensive cerebral infarctions, COLLET edema of the brain tissue is usually expressed . In this regard, a displacement of structures is possible, while an infarction focus in the large hemisphere can cause dislocation of the brain stem, the development of petechial hemorrhages and edema in it. Edema of the brain with symptoms of dislocation of its trunk often leads to n; respiration, which in such cases is the cause of death of patients with both ischemic and hemorrhagic stroke, with 1 the severity is usually even more significant. The immediate cause of death in patients with stroke may also be the primary respiratory and general hemodynamic effects caused by the development of a hemorrhagic focus directly in the stem structures.

Hemorrhagic stroke

For hemorrhagic stroke are nontraumatic krovoiz - Impact of the cranial cavity. They are 5 times less common than ischemic strokes ; in most cases occurs in people aged 50-60 years. Intracranial hemorrhages can be divided into intracerebral and intrathecal hemorrhages , of which subarachnoidal hemorrhages are predominant . Depending on the source of hemorrhage hemorrhagic strokes can be divided into arterial and ve - noznye.

Etiology and pathogenesis

Hemorrhagic stroke is more common in patients with essential hypertension II or III stage, secondary hypertension, in particular - NOSTA with renal disease which is the cause of hypertension in about 5% of cases, and when dyscirculatory encephalopathy caused by a combination of hypertension and atherosclerosis of cerebral vessels ... Hemorrhagic stroke can be with - yarn with hypertensive crises arising in certain income statement - holyah violating endocrine balance (pheochromocytoma, basophilic pituitary adenoma). Cause hemorrhagic stroke can be systemic connective tissue diseases, including red ox - erythematosus, periarteritis nodosa, and coagulopathy (hemorrhagic diathesis, DIC), blood diseases (haemophilia, leukemia, aplastic anemia, thrombocytopenia, hyperfibrinolysis et al.) , vasculitis, eq - lampsiya, vitamin deficiency, congenital angioma, septic conditions, Ur - mia. Approximately 20% of the cause of hemorrhagic stroke is - are arterial aneurysms, arteriovenous malformations, bundle - Nia arterial wall. Hemorrhage into the cranial cavity can be provoked

thrombolytic therapy, in particular taking Fibro nolitic heskih drugs, anticoagulants and abuse alko - golem, cocaine, amphetamines group of drugs (amphetamine, the first tin et al.).

Hemorrhagic stroke develops as a result of vessel rupture (per rexin) or increased permeability of the vascular wall (reg *diapedesin).* Arising from the increased permeability of the vessel wall Single-AF - nye (petechial) hemorrhage, merging may form a vast - nye hemorrhagic foci. The incident area originated hemorrhage - dissolved disorders of metabolic processes in nervous tissue (hypoxia times - vitie necrotic process) impregnating the blood brain tissue under - vergsheysya destruction.

At a hemorrhagic stroke is difficult to determine from which the vessel was bleeding, so their locations are differentiated by localization of the foci in the brain (hemorrhage in lobnovisoch Noi area, bleeding in the brain bridge, etc.), rather than by name sosudis - of the pool in which there was a violation of cerebral hemodynamics, as is customary in ischemic strokes. Hemorrhages often occur in the deep parts of the cerebral hemispheres, which allowed J. Charcot (1867)
to associate the majority of hemispheric hemorrhages with rupture of one of the deep branches of the middle cerebral artery, which he named arteria gemorragica **;** most likely, this meant one of the lateral bands of the coulostriate arteries.

The hemorrhage is usually due to high blood pressure, aneurysms rupture less cerebral vascular amyloid angiopathy, by - Vyshen vascular permeability, reduction svertyvaemos - ti blood. Hemorrhagic stroke with arterial hypertension often occurs due to rupture of small penetrating arteries. In this case, the subcortical nodes, thalamus, bridge, cerebellum are affected (in order of decreasing frequency). Foci of hemorrhage arising from hemorrhoids - cal diathesis, anticoagulant therapy are possible in other Oblas - tyah brain. Amyloid angiopathy is a cause of hemorrhage - Cesky stroke in the elderly, with foci of hemorrhage usually cover a particular proportion of the brain, they can be repeated, sometimes multiple. In hemorrhagic stroke, the destruction of brain tissue, edema of cerebral structures, impaired venous and cerebrospinal fluid outflow occurs , and intracranial hypertension develops.

Rupture of the diseased vessel wall occurs more frequently when a sharp rise in blood pressure and leads to the formation of n - mat. This usually occurs before the gap changes of vessels - stand wall. These changes, in principle characteristic of arterial gi - pertenzii at which arise subendothelial infiltration at - Vyshen permeability of the endothelium of blood plasma and its shaped elements perivascular extravasation, the formation of microaneurysms, bundle vascular wall and finally its rupture. Development krovoiz - EFFECTS brain promote breakdown of self cerebral hemodynamics enhancement of lipid peroxidation, increase fibrinolitiches Coy activity of the blood. Increased intracranial pressure when blood - outpouring into the brain may lead to a decrease of the perfusion pressure in cerebral vessels and lead to almost total brain hypoxia of the brain.

Cerebral hemorrhage often develops from subcortical vascular otho - dyaschih from larger vascular trunks at an angle close to right - mu. Hematoma and Related her surrounding tissue edema can obuslo - vit displacement (dislocation)

transtentorialnoe brain structures and their impaction or occlusion liquor flows and impaction tonsils MH - cerebellum into the foramen magnum.

Hemorrhage into the brain to differentiate intracerebral (parenhi - matous), intraventricular and intrathecal (subarachnoid often). Parenchymal-intraventricular or parenchymatous-sub-enveloped hemorrhages are also possible .

Parenchymal hemorrhage in the cerebral hemispheres and cerebellum in most cases are due to rupture of the vessel (hemorrhage - of reg rexin) and manifest themselves in the form of hematomas. Hemorrhage reg rexin are also the cause of intraventricular and intrathecal, parenchymal , intraventricular or podobolochechnyh-parenchymal hemorrhage. Rupture of the vessel and hematoma formation often proish - dit with hypertension but may be due razryva- aneurysm or arteriovenous fistula. "Spontaneous" hematoma in those Menno-occipital or forehead - Noah region sometimes fuss - mess as a result of rupture of microaneurysms or micro angioma.

Hemorrhagic propyl - tyvanie (reg hemorrhage *diapedesin*) can be provoked intoksika - tion, beriberi, bolez - blood nyami, the presence in the brain of the patient hearth Ishe - mission etc. In this form of bleeding more likely it is time - zhayutsya thalamus or brain Axle; if the reason for tacos - the second hemorrhage is a bleeding diathesis, then it can manifest in the material of the half-large - dence.

Certain special - Nosta have a ***hemorrhage in the meninges.*** Most often they are in *the subarachnoid* space and the WHO - Nick due to aneurysm rupture of arterial vascular base of the brain. There may be bleeding in the *subdural* and *epidural nye* space is formed - vaniem Correspondingly hematomas - vuyuschey localization, but in most cases these are hematoma traumatic origin.

Intracerebral hemorrhage is typically localized in the brain of a large (80%) and differentiated into medial and lateral depending on their location in a large hemisphere relative to the internal capsule are possible and massive bruising propagating ni Party or on both sides of the internal capsule. Hemorrhages in the brain stem and cerebellum account for approximately 10% of all cases of intracerebral hemorrhage.

Intraventricular hemorrhage may result from vascular wreath product - Nij. However, most often blood gets into the ventricular system of the brain at a copper - cial intracerebral hematoma extending to paraventriku polar structure, which leads to blood entering into the ventricles of the brain. Such hemorrhages are usually called ***parenchymal-intraventricular.*** When blood enters the ventricular system are possible stenosis or occlusion of liquor pathways and the development of occlusive gidrotse - falii, signs decerebration. Breakthrough Blood of hematoma formed - sheysya in the parenchyma of the cerebral hemispheres, into the ventricular system of the brain is a severe complication. It occurs in the first hours of a stroke, sometimes after 1-5 days and is detected in the majority of deaths from krovoiz - Impact of the brain.

With intracerebral hematomas of supratentorial localization, in most cases there is a correlation between their size and the degree of impairment of consciousness. Hematoma whose diameter is 3-4 cm, usually accompanied by impairment of consciousness, cerebral and expressed - hydrochloric focal symptoms. Hemorrhage in the internal capsule there is a correlation between the

level of consciousness in the most acute ne - IRS intracerebral hematoma and prognosis. When coma during the acute intracerebral hemorrhage achieving mortality - is 92%, and in cases where the patient at this time is in a state og - lusheniya or sopor, 42% of the patients die.

Clinical manifestations and diagnosis

Hemorrhagic stroke is usually develops acutely, suddenly during vigorous activity, physical activity, emotional reactions, ve - duschih to the rise in blood pressure, and is characterized by a combination of brain and hearth - O symptoms. Features of clinical picture in many ways defined - are the character, localization rate of development and size of hemorrhage - Cesky hearth, the severity of collateral edema, hemorrhage in relation hearth liquor system, its effect on the brain stem. Characterized - thorns sudden loss of consciousness, expressed cerebral, while pas - renhimatoznyh hemorrhagic stroke - and focal neurological signs. Along with intense headache, vomiting often occurs; possible epileptic paroxysm, psychomotor agitation. Plain - us pronounced vegetative disorders.

When supratentorial hemorrhage, loss of consciousness sometimes WHO - arises not immediately, and may occur within a few minutes, at least by a few hours. During this period, characterized by acute headache, post - foam or stepwise increases focal symptoms in connection with the pro - continues to hemorrhage into the brain and progressive swelling of the brain. On the side of the hematoma, the zygomatic symptom of ankylosing spondylitis is usually caused; on the opposite side, hemiparesis or hemiplegia usually occurs. In this case, the possible manifestations of the syndrome of cerebral irritation about - lochek.

In the case of coma on the side hemiparesis or hemiplegia, almost always occur at a hemorrhage into the parenchyma of large - hemisphere on the side opposite the pathological focus, usual us "sluggish" upper lid "sail" cheek stop rotate outward are possible fixed or floating gaze strabismus, anisocoria, autonomic disorders, muscle tone disorders, often cardiac and respiratory disorders, increased protective reflexes, hormone.

With **hemorrhage into the large hemisphere,** usually quickly, sometimes deep central hemiparesis or hemiplegia develops. Muscle tone in paralyzed limbs may initially be reduced, often variable. The patient's face is purple, puffy, the mouth is half-open, the veins in the neck are tense, hyperhidrosis is common, consciousness is altered, and stunning, stupor, coma are possible; often vomiting, hyperthermia, blood pressure is more often elevated, the pulse is tense, bradycardia, collapse, breathing disorders are possible, while tachypnea, snoring, and sometimes periodic Cheyne-Stokes type breathing with difficulty breathing in and out (inspiratory or expiratory retention), possibly changing according to amplitude, rare breathing. The gaze in the most acute period can be turned towards the paralyzed limbs, then towards the focus, sometimes a divergent squint. Psychomotor agitation, anxiety, convulsive paroxysms, automated gestures in non-paralyzed limbs, anosognosia (inability to realize the severity of one's condition, the presence of paresis, paralysis and other clinical manifestations of the disease) are possible .

In cases of blood breakthrough into the ventricles of the brain, moving automatisms, manifestations of hormetonia, and protective reflexes are possible .

Specific hemorrhage into the ventricular system of the brain Tonia ***(gormetonicheky Davidenkov syndrome)*** represent repeats the range of this seizures tonic convulsions arising spontaneously or under the influence of external stimuli, thus expressed protective reflexive sy. Hormetonic convulsions appear more often against the background of a coma . They were described in 1919 by the Russian neuropathologist **S.N.** ***Davidenkov*** (1880-1961).

With hemorrhage in the brain stem **,** more often in the pons or midbrain, manifested in most cases in the form of hemorrhagic impregnation , a suddenly developing coma is characteristic. At the same time , autonomic disorders are especially pronounced: pallor, blueness of the face, ophthalmoparesis, strabismus, wide or (with hemorrhage into the bridge) very narrow, dotted marks, floating 'gaze, Hertwig-Magendie syndrome , anisocoria, loss of corneal reflex, nystagmus are possible. Possible "ocular bobbing" - periodic b movements of the eyeballs downward followed by their slow return to their original position, combined with restriction of eye movements to the sides (a sign of bilateral lesion of the bridge tire); from absent or grossly disrupted okulotsefalicheskie reaction is usually peri ferichesky paresis facial muscles elements pseudobulbar or bulbar syndrome symptoms pyramidal insufficiency, muscular tonus with low or variable, bradycardia, soon replaced by tachycardia, the pulse tense AD usually taller gi pergidroz, cold limbs, respiratory and cardiac dey telnosti, it is possible stupor, passing in a coma for several hours is often a hyperthermia.

Of additional methods in diagnosing intracerebral hemorrhage particularly informative echo EG (for intracerebral krovoiz - EFFECTS revealed considerable - shift of M-echo in a direction opposite localization hematoma), K T - and M P T uc repetition allows made more precise thread location and Prevalence - nennost hemorrhagic pro - cession.

Forecast. In most cases, hemorrhagic Institute - Soult has a severe course. The lethality with it is very high - from 60 to 90%. Death often occurs within the first 2 days. Especially great le - talnost with hemorrhages in the brain stem and cerebellum, as well as extensive hematomas in the cerebral hemispheres, complicated by a breakthrough of blood in the ventricular system.

Subarachnoid hemorrhage

Subarachnoid hemorrhages account for about 10% of CVA. Le - talnost with them on the first day up to 25%, and for the first 3 months after a hemorrhage in the shell - to 50% mortality rate is particularly high at repeated subarachnoid hemorrhage due to rupture of ap - ter aneurysm.

E t i o l o g y and p a t h o g e n e s i s . Subarachnoid hemorrhage - a consequence of vascular rupture, often in the area of internal elastic membrane defect, leading to the formation of saccular aneurysms which form - camping in the area of bifurcation or branching arteries located in subarahno idalnom space skull base. It is also possible gap spindles - noobraznyh, globular or diffuse

aneurysms located on the course branches of the internal carotid, or vertebral basilar artery, in arteriovenous malformations. There subarachnoid blood - effusions during exercise, but they can develop without any noticeable precipitating factors. Other causes of subarachnoid hemorrhages, pronounced manifestations of hypertension, blood diseases, infectious-toxic and blastomatous lesions of intracranial vessels, traumatic brain injury.

Aneurysms usually form in the vessels involved in the formation of the arterial circle of the large brain; the rupture of such aneurysms is more common in children or young people. In this case, hemorrhage due to rupture of the so-called supraclinoid aneurysm, usually developing from the posterior communicating artery, may be preceded by migraine- like headaches in the frontal-orbital region in combination with signs of paresis of the muscles innervated by the oculomotor nerve, which is rarely regarded as an attack of ophthalmoplegic migraine. Subarachnoid hemorrhage is also possible with rupture of its anterior connecting artery, proximal sections of the anterior and middle cerebral artery and their branches.

C l i n i c a l manifestations . Subarachnoid hemorrhage manifests itself as acute signs of irritation of the meninges. With physical or emotional stress, and often for no apparent reason, there is a sharp headache, pain in the neck, often spreading down along the spine, into the interscapular region. In this case, vomiting, disturbance of consciousness, psychomotor agitation, meningeal symptoms (stiffness of the occipital muscles, symptoms of Kernig, Brudzinsky, etc.), hypo- or areflexia, signs of intracranial hypotension, convulsions, autonomic disorders, in particular hyperthermia up to 38 -39 ° C. Focal symptoms in the most acute stage are not defined or expressed slightly. When basal arterial aneurysms rupture , signs of damage to the cranial nerves, more often the oculomotor, sometimes optic nerves and their intersection, may appear, hemorrhages into the retina of the eye are possible .

A manifestation of massive subarachnoid hemorrhage is its "tetanus" form - Shoffard's ***syndrome,*** in which meningeal symptoms are sharply expressed and there is a tendency towards predominant rigidity of the extensor muscles. Usually seen in children and young people.

The severe course of intrathecal hemorrhage most often leads to the development of respiratory and cardiac disorders, while situations arise that pose a danger to the patient's life.

In some patients , ischemic stroke usually develops on days 4-14 after subarachnoid hemorrhage. It, as a rule, arises in the basin of the artery, the rupture of the wall of which caused the hemorrhage. In this case, a cerebral infarction is formed, and focal neurological symptoms develop (hemiparesis, motor aphasia, etc.). Possible pronounced disorders of the functions of the hypothalamus due to irritation of its outpouring blood.

In 2-6 weeks after subarachnoid hemorrhage, repeated circulatory disorders of the same type are possible.

In the blood with subarachnoid hemorrhage, there is a moderate leukocytosis with a shift of the leukocyte formula to the left.

Late complications of subarachnoid hemorrhage can be resorptive hydrocephalus, persistent intracranial hypertension

D i a g n o s t i c s . Clarification of the diagnosis in case of subarachnoid hemorrhage can be facilitated by CT - or MRI - examination, diagnostic lumbar puncture, angiography. On CT on the first day in 90% of patients, blood is detected in the subarachnoid spaces; if an intravenous contrast agent is injected during a CT scan, then sometimes a source of subarachnoid hemorrhage is revealed, in particular aneurysm, arteriovenous malformation. With a lumbar puncture, the cerebrospinal fluid pressure is high (up to 500 mm of water). An obligatory symptom of subarachnoid hemorrhage is the presence of blood impurities in the CSF obtained with a diagnostic lumbar puncture (in the acute period, the CSF has the color of cranberry juice).

In the CSF with subarachnoid hemorrhage in the acute period, a pronounced admixture of blood staining it is noted, appearing after 3-6 hours; subsequently, usually after 3-5 days, xanthochromia, which lasts up to 3 weeks. -1 month From the 3rd day, CSF is detected within 4-19 days (on average, up to 9 days) after a stroke. In CSF (in a convenient approximation) 1 leukocyte corresponds to 700 erythrocytes, and every 1000 erythrocytes in 1 μl corresponds to 0.01 g / l of protein. During the 1st week in the CSF, there is usually a moderate decrease in glucose. In 3-4 weeks after hemorrhage in the CSF, lymphocytic pleocytosis up to 150 per 1 μL is noted.

Angiography data are the most informative for detecting aneurysm of cerebral vessels.

BASIC BRAIN STROKE THERAPY

Function regulation Respiratory analeptics

external respiration
is contraindicated!

Regulation of functions 1. Correction of systolic blood pressure
cardiovascular and maintaining it at the Joint level
systems 5-10mm Hg above
"workers" observation
digits neurologist and
2. Correction of
paroxysmal cardiologist

heart rhythm disorders
3. Therapy of concomitant pathology hearts

Regulation of water-salt and acid-base fortunes

1. Infusion therapy
2. Control: blood gases, pH-metry, osmolarity of urine and blood, blood electrolytes, biochemical blood test

A joint observation neurologist and reanimation logs

Combating cerebral edema and intracranial pressure

Dexazone 8 + 4 + 4 + 4 mg IV are preferred

prevention of an increase (in the absence of severe forms of sugar diabetes, internal bleeding, malignant arterial hypertension, torpid to therapy)

or osmotic diuretics (reogluman 2 times a day, mannitol 200 ml of a 15% solution intravenously drip, after 10-15 minutes lasix 20 mg intravenously)

Patient care. Prevention of hypostatic pneumonia

Prevention of pulmonary embolism, pressure ulcers, complications. purulent corneal ulcers, early contractures

Patients primarily are unconscious, without - need to be introduced liquids parenterally (2000-2500 ml per day in 2-3 doses). Solutions containing electrolytes

(isotonic sodium chloride solution, Ringer-Locke's solution), 5% glucose solution, polyglucin, rheopolyglucin, solutions of potassium nitrate or potassium chloride (up to 3-5 g per day) are introduced .

For correction of CBS should maintain adequate ventilation lay - FIR.

To eliminate acidosis, along with an increase in pulmonary ventilation with oxygen therapy, as well as measures to improve cardiac output and discharge, a 4% sodium bicarbonate solution is injected intravenously; In metabolic alkalosis, the water balance is replenished, hypokalemia and hypochloremia are corrected .

In order to **combat cerebral edema and prevent an increase in intracranial pressure,** it is possible to administer osmotic diuretics, in particular , glycerol is injected inside at a dose of 1 g / kg / day mixed with water or fruit juice in a ratio of 1: 2 or 1: 3; if the patient does not swallow, the mixture is administered through a tube. For the same purpose, a 15% solution of mannitol is injected intravenously at the rate of 0.5-1 g of dry matter per 1 kg of weight. The required dose of the drug is dissolved in 200 ml of isotonic sodium chloride solution or 5 glucose solution and injected intravenously in 2 divided doses. To accelerate the dehydrating effect, lasix can be administered intravenously in a dose of 20 mg after 10-15 minutes . Dexamethasone (dexone) can also be used at a dose of up to 20 mg / day, followed by a dose reduction of 4 mg / day, however, corticosteroids are not indicated in severe forms of diabetes mellitus , arterial hypertension, in the presence of internal bleeding

With hyperthermia up to 39-40 ° C and other autonomic disorders, a 4% solution of amidopyrine or 50% analgin is administered. 2-3 ml intramuscularly to increase heat transfer rub the body with alcohol, cool, use ganglion blockers in combination with seduxen, diphenhydramine intramuscularly or intravenously drip , lytic mixtures, for example isotonic sodium chloride solution 250 ml + 5% pentamine solution or benzohexonium solution 1-2 ml + 2% diphenhydramine solution 2 ml.

To maintain water and electrolyte metabolism, infusion therapy is performed.

It is necessary to pay attention to the organization of food; if necessary, nutrient mixtures are introduced through a tube.

Measures are required to prevent pressure ulcers pneumonia , control the functions of the pelvic organs. It is shown, in particular , to turn the patient in bed every 2-3 hours. 24-48 after the stabilization of the clinical picture of the stroke, passive gymnastics is performed up to 3-4 times a day.

Differentiated treatment in the acute period of ischemic stroke

The main goal of therapy in the acute period of ischemic stroke is to suppress the formation of cerebral infarction against the background of acute cerebral ischemia: improving the rheological properties of blood, correcting the functional state of the brain, aimed at reducing the expression of neurological deficit and metabolic protection of the brain from the fact of ischemia - hypoxia. In case of cerebrovascular insufficiency, which developed against the background of a fall in blood pressure and a weakening of cardiac activity, cardiac glycosides (korglucon, strophanthin) and pressor amines (mezaton, ephedrine), as well as corticosteroids are prescribed.

TREATMENT OF ISCHEMIC STROKE IN THE ACUTE PERIOD

The first 3-6 hours - 3-5 days

In accordance with the concept of the development of the "ischemic cascade , there are two main areas of treatment for ischemic stroke.

1)improved perfusion of brain tissue (impact on the 1st stage of the cascade).

2)neuroprotective therapy (impact on the 2-6th stage of the cascade).

Among the methods aimed at improving blood perfusion in tissue brain, an important place is occupied by hemodilution, mainly hypervolemic , with the help of low molecular weight dextrans (rheopolyglucin, rheomacrodex). Dextrans are administered every 12 hours by intravenous drip of 250-500 ml for 1-2 hours under the control of laboratory parameters of the state of the cardiovascular system. The main criterion for the effectiveness of hemodilution is a decrease in the hematocrit level to 30–35 %. With an increase in blood pressure against the background of hemodilution, it is necessary to reduce the rate of dextran administration (less than 20 drops per minute). The optimal course of hemodilution is usually 3-5 days.

Endogenous fibrinolysis activators (exogenous thrombolytics: urokinase, streptokinase, acetylated plasminogen-streptokinase complex) are contraindicated in ischemic stroke, which is primarily associated with frequent hemorrhagic complications and short-term drug action (only the first 3-4 hours of stroke).

However, the possibility of effective thrombolysis with complete vessel recanalization continues to attract attention. In this regard, at present , special significance is attached to endogenous thrombolytics (tissue activators of the plasminogen type), which, unlike substances synthesized and vascular walls, act only on fresh thrombi. The advantage of endogenous thrombolytics is also the absence of inactivation of coagulation factors V and VIII, which significantly reduces the risk of generalized anticoagulation and hemorrhages.

In arterial thrombosis medium and large diameter may ispol'uet - mations tissue activator ***of plasminogen*** in the first 3-6 hours after the onset yn - Counsel, sometimes allowing to achieve rapid recanalization of the affected vessel. Intra-arterial administration of this trobolytic at a dose of 0.9 mg / kg of body weight leads to a significant improvement in the condition of patients and the prognosis of the disease. However, in hemodynamic and microcirculatory (lacunar) strokes, the use of the drug is inappropriate.

The efficacy of ***antiplatelet therapy*** in acute ischemic - one stroke. Comparative analysis of the effect of drugs in this class - it seemed that the most advantageous and effective in patients of different age, irrespective of the level of blood pressure, is *pentoxifylline (Trental)*, providing comprehensive antithrombotic, antiaggregatory and reo - logical action (in contrast to the predominantly antiagregatsionnogo influence of other means). Prescribe pentoxifylline, 0.1 g (5 ml) in 350 ml of isotonic sodium chloride solution or 5 % glucose solution, increasing the daily dose to 0.2-0.3 g. At the same time, the choice of antiplatelet agent depends on the localization of vascular lesion, age patient, comprising - Niya cardiovascular system and other premorbid features. Patients of elderly and senile age are shown the use of ***antiplatelet angioprotective agents*** (prodectin, anginin, parmidin) inside, 250 mg 3 times a day during the entire acute period of stroke, indomethacin inside 25-50 mg 3 times a day after meals for 10- 14 days. In the presence of a

tachycardia in patients with severe resistant higher Nia blood pressure levels are preferred *beta-blockers,* eye - binding and antiplatelet effect: *obzidan* (Inderal) 10-20 mg four times a day for the first 5-7 days, then 20 mg of 4 once a day for 2-3 weeks under the control of the activity of the cardiovascular system. In the case of acute circulatory disorders in the vertebrobasilar system, *stugerone* (cinnarizine) is most effective along with the use of trental (pentoxifylline). With the localization of ischemic - one stroke in the hemisphere is better to use *cavinton* (vinpocetine). During the first 3-5 days Cavintonum be administered intravenously (honey - lenno) 10 mg (2 mL of 0.5% solution) in 500 ml of isotonic solution of sodium chloride up to 3 times a day. Subsequently possible to use pre - Paratov inwardly 5-10 mg 3 times a day for 3 weeks attraction. It should be on - imagine that this drug is contraindicated in severe ischemic slaughtering - Levan heart arrhythmias; in some patients, it worsens the venous outflow from the cranial cavity. Cavinton, like other antiplatelet agents, should not be used in combination with heparin.

As antiplatelet agents may also be recommended treatment *ac - Pirin* - strong irreversible inhibitor of cyclooxygenase - at a dose of 1-2 mg / kg / day; *tiklid* 250 mg 1-2 times a day, nicergoline (SERME oxazolidinone) 10 mg 3 times a day, *aminophylline* - 10 ml 2.4% solution in - venno isotonic sodium chloride solution (if no Ishe nomic heart disease, no lowering blood pressure and heart rhythm disturbances). When rapid abolition of antiplatelet agents may be a syndrome of - barter - the deterioration of rheological properties of blood. Therefore, the dose of drugs should be reduced gradually.

Vasodilators (papaverine, nicotinic acid and its derivatives) are ineffective in the acute period of ischemic stroke. On the contrary, the WHO - possible adverse effects of these drugs: the development of the syndrome of "on - kradyvaniya" due to the dilation mostly intact with - vessels and increase cerebral blood flow in neishemizirovannyh areas, as well as the development of systemic arterial hypotension.

T h e i n d i c a t i o n s for use ***of anticoagulant*** therapy within the first hours and days of ischemic stroke are buildup clinical pro - stroke events (typically due to thrombus formation) and the presence cardiocerebral embolism.

C o n t r a i n d i c a t i o n s to the destination anticoagulants are - are persistent increase in blood pressure (above 180 mm Hg) or, conversely, its significant reduction, deep coma consisting - of, seizures, severe liver disease, kidney disease, ulcerative - Naja ulcer and other hemorrhagic manifestations. It is one - to be noted that even if there are contraindications sometimes have to resort to anticoagulant therapy - namely, the development of DIC syn Dre.

Preferably the assignment of direct anticoagulant *(heparin)* in Techa - of the first 2-5 days of disease at a daily dose of 10 to 000 units under the skin of the abdomen (2500 IU 4-6 times a day) or intravenously in a daily dose of 10 000-24 000 units. Compulsory laboratory monitoring of the coagulogram and syncumar (by mouth, 4 mg 2-3 times a day), the reception of which is continued for the next 3-4 weeks.

Due to the frequent lack of antithrombin III in patients with ischemic stroke, it is advisable to inject *fresh frozen blood plasma* simultaneously with heparin (100 ml 1-2 times a day for 2-3 days).

The development of qualitatively new effects on cerebral microcirculation is considered to be a promising area of therapy that normalizes cerebral perfusion . Clinical trials application acute ischemic stroke ***chemical substitutes*** (flyuozolada, perfluorocarbon) having the ability to bind and carry oxygen effectively saturate them brain tissue, and ak tive natural vasodilator and antiplatelet prostacyclin (epoprostenol) and some bioreologicheskih drugs, including venom Malay swamp snake is an ancrod with a complex effect on the rheological properties of blood.

The next important direction in the treatment of acute ischemic stroke is ***neuroprotective therapy.***

Modern concepts of the mechanisms of the formation of cerebral infarction and the metabolic aspects of the pathogenesis of ischemic stroke have confirmed the importance of ***metabolic therapy for*** stroke. Results experi mental and clinical studies have shown that the maximum clinical effect of metabolic therapy is observed when she was appointed Research Institute of the first stroke of the clock, and is determined by an adequate choice of metabolically active drugs - under the control of multimodal mo nitorirovaniya functional activity of the brain.

Traditionally, the main direction of metabolic therapy is camping ***correction brain energy metabolism:*** reduction of the damaging dei Corollary dyscirculatory hypoxia on brain structure by inhibition of cerebral metabolism with a decrease in energy need NOSTA neurons (antioxidants), or stimulation of redox processes, enhancing glucose utilization (nootropics).

Antioxidants are used mainly in the most severe cases of stroke, accompanied by inadequate brain hyperactivity, occurring with "uneconomical" energy metabolism. The field clinical effect is manifested by regression of psychomotor agitation (if any), paroxysmal changes in muscle tone, vegetative-trophic disorders, faster recovery of consciousness, and disappearance of general cerebral symptoms.

The use of antioxidants in the acute period of ischemic stroke is also most effective in the first hours of the disease. Advantageously purpose antioxidants with a different mechanism of action: the drug destroying peroxides *(unitiol* 5 ml of intravenously), *tocopheryl la* (vitamin E) and *retinol* (vitamin A) or complex (e.g., Aevitum 2 ml, 2 times a day) linking catalysts that inactivate free oxygen during the first 3-5 days after the onset of a stroke.

As an antioxidant, sometimes *thiopental notes,* or *hexenal* (0.3-0.4 g intravenously jet slowly or drip 2-3 times a day), phenobarbital (0.1 g 2 times a day), benzodiazepines, in particular sibazone, are sometimes used (seduxen, relanium) 2 ml of 0.5% solution intravenously slowly 3 times a day. Use as antigipoksantov preparations from the group of barbiturates undesirable because the possible manifestations of nega - tive effects: cardiodepressive and hypotensive action, a method - NOSTA inhibit the activity of the respiratory center.

Experimental and clinical studies show - whether the effectiveness of the domestic drug *mexidol* (oksimetiletilpi ridina succinate). Intravenous drip administration in a dose of 100 to 800 mg per day of the drug has a significant antioxidant effect, on - vyshaya activity endogenous antioxidant system and reducing you - expressions of free radical processes. Clinical efficacy mexidol manifested significant regression in patients disorders consciousness and

significantly faster (compared to patients taking - yuschimi placebo) recovery of motor function and reduction of symptoms of vasomotor instability.

Another promising direction antiischemic protection brain is interrupted primary links glutamate calcium k'as - qada to correct the imbalance of excitatory and inhibitory neuro transmitter systems and activate the natural inhibitory processes. This may facilitate a direct impact on system neurotransmitters and neuromodulators brain neuronal receptors - ry leads to normalization correlation processes and exciting the torus - synchrotron-neurotransmission.

Natural ***activator inhibitory neurotransmitter systems*** is *glycine*, developed in PBMCs "Biotics" giving multicomponent - -component antiischemic effect. Glycine is an inhibitory neurotransmitter, as well as universal conjugate nizkomoleku - polar toxic compounds in large quantities produced during ischemia. Glycine is used in the first hours and days of stroke sublingually at a dose of 20 mg / kg of body weight (on average, 1-2 g per day). It is found that the gly - ching limiting toxic effect of excitatory aminatsidergicheskih neurotransmitters (glutamine, aspartate), promotes the binding of the released during cerebral ischemia aldehydes and pheno - fishing, resulting in attenuated damaging effects of ischemia on the brain tissue in patients with various localization of the ischemic lesion and reduced infarct area brain.

An important area of neuroprotective therapy is-Application - of drugs with neurotrophic properties and neuromodulator. For drugs ***neurotrophic*** number refers *Cerebrolysin,* prev - resents a protein hydrolyzate extracts from brain mle - kopitayuschih, active fraction action is due nizkomo - -molecular peptides expressed neurotrophic effect. The on - contrast to other natural neurotrophic factors cerebrolysin not cause antigenic anaphylactic reactions. Low molecular weight peptides cerebrolysin allows it to penetrate the BBB and ac - tively included in the metabolism of brain neurons. Protective dei - Corollary cerebrolysin brain tissue due to its stimulating dei - Corollary in brain energy metabolism and the synthesis of an intracellular protein deceleration processes kaldievogo glutamate cascade and peroxide lipid oxidation. At the same time, cerebrolysin has a pronounced neurotrophic effect. The use of Cerebrolysin in acute cerebrates - Noah ischemia promotes better survival of neurons in the ischemic area cal penumbra. In ischemic stroke of moderate severity, the optimal daily dose of cerebrolysin is 10 ml, in severe strokes - 20 ml intravenously drip in 100-200 ml of isotonic sodium chloride solution for 7-10 days, after which it is possible to continue the course of treatment in the form of intramuscular injections according to 5 ml for 21 s; diseases. Selection of optimal doses Cerebrolysin (and other meta parabolic active substances) is determined by not only the weight of the patient, but also the individual functional features ak ciency brain.

Polypeptide neurotrophic factors do not penetrate the BBB; therefore, much attention is paid to the study of the properties of low molecular weight neuropeptides that overcome the BBB, have a multifaceted effect on the central nervous system and are characterized by high efficiency and pronounced directionality, provided they are very low in concentration in the body.

At the Research Institute of Molecular Genetics, Russian Academy of Sciences, a synthetic analogue of the ACTH fragment, the drug *"Semax", has been created,* which is a neuropeptide devoid of hormonal activity. Semax is an endogenous

regulator; torus functions of the central nervous system and has neuromodulatory activity, and so " gives a pronounced nootropic effect. The inclusion of Semax in the complex of intensive therapy for acute hemispheric ischemic stroke reduces early mortality rates, has a beneficial effect on the severity and rates of recovery processes, promotes the acceleration of regression of cerebral and focal, especially motor, n; collapses. The optimal dose of the drug for strokes of moderate severity is 12 mg / day, for severe strokes - 18 mg / day intranasally. Treatment with Semax during the first 10-14 days of the disease makes it possible to alleviate the course of stroke and improve the recovery of impaired neurological functions.

In cases of prevalence in the clinical picture of a focal neurological defect, *neurotropic* ***drugs*** (predominantly GABA derivatives) are shown that activate energy metabolism and redox processes in the brain. Nootropic drugs are especially effective for limited cortical foci of ischemia, clinically manifested by disorders of higher mental functions (including speech) and motor deficits.

Stimulants of energy metabolism include *piracetam, gammalon, picamilon,* which have a nootropic effect . Piracetam (nootropil) and gammalon give a particularly significant effect in the group of patients with mild ischemic stroke, especially with superficial cortical localization of the focus. The optimal daily dose of drugs in the first 10 to 15 days is 0.1-0.2 g / kg of the patient's body weight, ie. on average 6-12 g per day (2-3 g 3-4 times a day intravenous stream or drip). Then the same drugs are administered orally at a dose of 4.8 g / day in 3 divided doses for 1 - 1.5 months.

Clarification of the pathogenesis of ischemic stroke in recent years has created prerequisites for the development of new methods of metabolic protection of the brain.They are aimed at interrupting the cascade of pathobiochemical reactions launched by energy deficiency of lactic acidosis and allow preventing the formation of persistent morphological changes in the brain tissue against the background of still reversible ischemia. With this purpose ***anta - gonisty calcium ions*** (calcium channel blockers).

It was found that among calcium antagonists in the acute period of ischemic stroke, dihydropyridine derivatives *(nipo dipine, nifedipine, nicardipine) are preferred.* Apart from their effects on the neuronal level (inhibition of intracellular calcium accumulation), these prep - you increase the elasticity of the erythrocyte, cerebral arterioles expand, i.e. improve postischemic reperfusion of brain tissues. Naibov - Lee efficient use of calcium antagonists in the first 12 hours after the onset of ischemic stroke. Single and daily doses of calcium channel blockers should be selected individually, taking into account the level of blood pressure.

In the acute phase of stroke is important to activate backup links Ener - energetically metabolism. To enhance the transport of fatty acids into mito - chondro and activation of fatty acid pool of acetyl CoA is expedient to use preparations carnitine *(aplegin).* Fatty acids, in contrast to glucose can be oxidized at a low oxygen tension in the blood, cerebral energy needs replenishing and maintaining of life activity - telnost neurons during hypoxia. Aplegin (10% solution Karnith - on chloride) injected intravenously at 1000 mg (15 mg / kg of patient weight) for 5-7 days. If necessary, after 2 weeks Provo - ditsya repeated

treatment with 500 mg (7 mg / kg) for 3-5 days [Kouzin VM Kolesnikov TI, 1996].

One of the treatments for ischemic stroke is ***Hyperbar - Ceska oxygenation,*** which can be used in com - the complex therapy of acute and periods of acute pathological protses ca.

It should be noted that the trend towards active treatment of a patient with ischemic stroke of various etiology is currently not shared by everyone. For example, in the UK and particularly in the United States in this form of pathology in the acute phase focuses on the maintenance of the vital functions of patients, carried out activities aimed at the prevention of complications of cerebrovascular pathology and general care in the future in survivors held physically active, logopediches - kai , psychological and social rehabilitation. Information about the conduct of patients with disorders of cerebral circulation in the United States found a reflection - of, in particular, in the book of CP Vorlou, MS Dennis, J. Van Gein et al. “Stroke. A Practical Guide for Patient Management ", published in Russian in 1998 in St. Petersburg.

Differentiated treatment in the acute period of hemorrhagic stroke

In identifying hemorrhagic stroke is needed is urgent - naya consultation of a neurosurgeon. When lateral hematomas in larger - brain lushariyah, hemorrhage in the cerebellum, as well as subarahnoi further bleeding due to rupture of an arterial anevriz - we, or arteriovenous malformation, can be shown neyrohi - rurgicheskoe treatment, in some cases it should be bezotlagatel - nym.

The patient should be provided with strict bed rest. In this case, it should be laid on that bed with a raised head end, the head should be given an elevated position, and its local cooling should be provided . The duration of bed rest is determined by the type and pathogenetic variant of hemorrhagic stroke. In the presence of aneurysms from the cerebral vessels, bed rest should be observed for 6-8 ns. The duration of this period is due to the fact that the vast majority of repeated hemorrhages from aneurysms occur within 1-1.5 months after the first. In addition, a significant period is required for the formation of strong connective tissue adhesions near the ruptured aneurism.

An important task of conservative treatment in the acute stage of hemorrhagic stroke should be **measures aimed at lowering, usually high in such cases, intracranial pressure and at the same time normalizing blood pressure, as well as normalizing the state of blood coagulation and permeability of the vascular wall, vital and vegetative functions.**

Intracerebral hemorrhage is usually accompanied by severe cerebral edema. The fight against cerebral edema and intracranial hypertension should be carried out taking into account the state of osmotic pressure and water-electrolyte balance. At low or normal osmotic blood pressure, it is advisable to use osmotic diuretics: glycerol (10% solution in isotonic sodium chloride solution at a dose of 1 g / kg of body weight per day regos or through a tube), reogluman (400 ml intravenously), mannitol (500 ml 10-20% solution intravenously drip) for 2-5 days with a gradual dose reduction to mitigate the phenomenon of recoil. It should be borne in mind that mannitol

is effective only when the osmotic regulation mechanisms are relatively intact . The absence of a decrease in osmolarity 2.5 hours after the administration of mannitol indicates the inexpediency of its further use. It is less desirable to use saluretics to reduce intracranial pressure due to their lower effectiveness, pronounced hypotensive effect and the likelihood of developing hypercoagulative syndrome. Nevertheless, it is possible to use lasix (2-4 ml of a 1% solution intravenously or intramuscularly) in combination with antiplatelet therapy.

Of the *corticosteroid hormones,* in the absence of contraindications, the drug of choice may be *dexamethasone, the* introduction of which does not lead to significant changes in the electrolyte composition of the blood. The drug has a moderate effect on blood pressure. Shown is the appointment of *dexazone* (under the control of hemorheological parameters) with increased or normal osmotic blood pressure intravenously or intramuscularly at a dose of 8 to 50 mg per day (in 3-4 portions) for 4-5 days with gradual withdrawal of the drug.

Control and correction of blood pressure is one of the main directions of treatment for hemorrhagic stroke. *Reserpine, clonidine* can be used to lower blood pressure . The decrease in blood pressure should not be sharp and excessive, it is desirable to bring it to the optimal value, which should be 15–20 mm Hg. exceed the patient's usual ("working") value

BP, slightly higher rates are acceptable.

If these drugs have the desired effect and blood pressure remains very high, ganglion blockers can be used under the control of blood pressure: pentamine (1 ml of 5% solution intravenously in 100-200 ml of isotonic sodium chloride solution or in 5% glucose solution slowly or drip), arfond (5 ml 5 % solution in 150 ml of 5% glucose solution or in isotonic sodium chloride solution intravenously drip from 30-50 to 120 drops per minute), benzohexonium (ml of 2% muscle solution).

In case of hemorrhages in the brain substance and intrathecal spaces, the hemostatic system, as a rule, shifts towards the activation of fibrinolysis and a decrease in the coagulation properties of the blood, and therefore requires the appointment of drugs that inhibit fibrinolysis and activate the formation of thromboplastin. In the first 2-3 days of hemorrhagic stroke (especially with hemorrhages in the subarachnoid space), epsilon-aminocaproic acid is used under laboratory control (50-100 ml of a 5% solution 1-2 times a day intravenously). It is also possible to administer a daily dose of the drug in 1 liter of isotonic sodium chloride solution using an infusion pump drip for 24 hours. Considering the possibility of a negative effect of epsilon-aminocaproic acid on the hemorheological properties and state of microcirculation, its administration can be combined with the use of pentoxifylline or other antiplatelet agents, as well as moderate hemodilution. In the next 3-5 days, an inhibitor of proteolytic enzymes is prescribed: aprotinin (gordox, cotrikal, trasilol) with the first injection of 100,000-500,000 KIE intravenously. With severe atherosclerosis, in order to avoid thrombotic complications, the administration of antifibrinolytics is combined with the appointment of low doses of heparin (2500 IU 4 times a day under the skin of the abdomen).

An effective hemostatic drug is dicinone (sodium ethamsylate), administered at 250 mg intravenously or intramuscularly 3-. once a day for 10 days. It activates thromboplastin, improves microcirculation and the condition of the vascular wall, and is also a strong antioxidant.

It is also advisable to administer calcium preparations (10-12 ml of a 10% solution of chloride or calcium gluconate intravenously), vikasol (1-2 ml of a 1% solution intramuscularly), ascorbic acid (1-5 ml of a 10% solution intravenously).

Since hemorrhage is often accompanied by spasm of cerebral arteries, it becomes necessary to use drugs that prevent spasm and improve collateral circulation. The drugs of choice in this case are calcium channel blockers, dihydropyridine derivatives, including nimodipine. Treatment with nimodipine begins with intravenous drip infusions of 15 μg / kg during the first hour, then - 30 μg / kg / h. Administration, if necessary, can be continued around the clock. After 5-10 days, they switch to taking nimodipine by mouth 60 mg 4 times a day.

Treatment of hemorrhagic stroke in the acute period

NEUROSURGICAL CONSULTATION AND RESOLUTION OF THE QUESTION ABOUT THE NEED FOR SURGICAL TREATMENT

"Cerebrovascular accident (CE - REBRALNYE crisis, transient ischemic attack and stroke)"

Control questions

1.How is the brain supplied with blood?
2. What is the etiology of acute cerebrovascular accidents?
3.Classification of cerebrovascular accidents.
4. What types of cerebral vascular crises do you know? 5 .. What is the pathogenesis of cerebral vascular crises?

6. The main clinical manifestations of cerebral vascular crises.
7. The main clinical manifestations of cerebral vascular crises in the carotid system.
8. The main clinical manifestations of cerebral vascular crises in the vertebral - basilar system.
9. Basic principles of treatment of cerebral vascular crises.
10. What is the pathogenesis of hemorrhagic stroke?
11. The main signs of hemorrhagic stroke.
12. The main signs of subarachnoid hemorrhage.
13. Determination of the side of the focus in apoplectic coma.
14. What is the pathogenesis of ischemic stroke?
15. Main characteristics in ischemic stroke (ratio of cerebral and focal symptoms)
16. Clinical differences between the defeat of the great vessels in the neck from thrombosis of intracerebral vessels.
17. Clinical manifestations of cerebral vascular embolism.
18. What are the main symptoms of circulatory disorders in the basin of the anterior cerebral artery?
19. What are the main symptoms of circulatory disorders in the middle cerebral artery system?
20. What are the main symptoms of circulatory disorders *called* back system cerebral artery.
21. What are the main symptoms of cerebrovascular accident in the vertebrobasilar system?
22. What are the main symptoms of cerebrovascular accident in the basin of the internal carotid artery?
23. Basic research in acute disorders of cerebral circulation.
24 . Changes of coagulation and anti-coagulation systems, aggregation and adge - sion platelet stroke.
25. Changes in cerebrospinal fluid in stroke.
26. Changes in cerebral blood flow prior to Doppler ultrasound in ischemic stroke.

27. Cerebral hemodynamics state according to rheoencephalography data in cerebrovascular accidents.
28. Diagnostic value of echoencephalography in cerebral strokes.

29 Changes detected by computed tomography *in* patients with hemorrhagic and ischemic strokes.

30. Indications and contraindications for angiography in cerebrovascular accidents.
31. What changes are detected by angiography in hemorrhagic ischemic stroke? |
32. What are the main criteria for the differential diagnosis of hemorrhagic and ischemic strokes?
33. The difference between an apoplectic coma and a coma of another origin.
34. Forecast for acute disorders of cerebral circulation,
35. Stages of specialized care for patients with acute cerebrovascular accidents.

36. General principles of undifferentiated treatment for brain disorders! th blood circulation.
37. Differentiated therapy for ischemic stroke.
38. Differentiated therapy of hemorrhagic stroke.
39. Rehabilitation therapy for cerebrovascular accidents.

40. Indications and contraindications for hospitalization of patients with acute cerebrovascular accidents.

41. Indications and contraindications for surgical treatment of hemorrhagic stroke.
42. Indications and contraindications for surgical treatment for circulatory disorders in the extracranial sections of the main vessels of the head and intracranial vessels.
43. Indications and contraindications for surgical treatment of cerebral aneurysms .
44. Examination and placement of patients after cerebral KRO - voobrascheniya?
45. What is the prevention of cerebrovascular accidents?
46. Spinal cord blood supply.
47. What are the etiological factors of circulatory disorders in the spinal cord?
48. What are the main signs of circulatory disorders in the spinal cord?

Tests of the I level of assimilation (1st option)

a) Identification tests

Are vegetative disorders associated with hemorrhagic stroke?
Could there be "flickering of symptoms" in ischemic stroke?

b) Tests for distinction

Indicate which of the listed symptoms are characteristic of the general cerebral vascular crisis? 1) Headache. 2) Dizziness. 3) Noise in the head. 4) Mono paresis. 5) Nausea or vomiting. 6) Short-term disorder of consciousness. 7) Aphasic disorders.

c) Classification tests

Which of the following symptoms are typical for: 1) carotid and 2) vertebrobasilar crises:

A) mono- or hemiparesis. b) systemic dizziness, c) anisoreflexia, d) paresthesia in the extremities of the same name, e) weakness and asthenia, f) visual disturbances, g) dysarthria and anarthria, h) dysphagia, i) aphasic disorders, j) Jacksonian epilepsy, k) temporal lobe epilepsy syndrome, m) cross optic-pyramid syndrome, m) memory disorders, o) diplopia, n) nystagmus, p) ataxia, c) alternating syndrome, t) auditory disorders, y) syncope, disorientation in space and time ...

Tests of the I level of assimilation (2nd **option)**

a) Identification tests

I Does a patient with subarachnoid hemorrhage have meningeal symptoms?

II Can ischemic stroke develop without blockage of cerebral vessels?

III. Can hormonal syndrome develop in hemorrhagic stroke?

IV. Is it possible to define hemiplegia in a coma?

V. It can be observed epileptiform seizures cerebral embolism suck - poisons?

b) **Discrimination tests**

Indicate what signs are characteristic of circulatory disorders in the middle cerebral artery basin . 1) Hemiplegia or hemiparesis. 2) Monoplegiya or mono - paresis of the legs. 3) Motor aphasia. 4) Astereognosis. 5) Apraxia. 6) Disorders scheme - we are the body. 7) Sensory disorders. 8) Monoplegia or monoparesis of the hand. 9) "Frontal psyche".

v) **Classification tests**

Which of symptoms characteristic of acute period different strokes: I) kro - voizliyaniya; 2) thrombosis; 3) embolism:

a) sudden onset, b) in the afternoon, after physical or mental stress, c) precursors, d) rapid development of focal symptoms, e) coma, f) short-term disorder of consciousness, g) the face is purple, cyanotic, hyperthermia, i) increased arterial pressure, j) meningeal symptoms, k) “symptoms at a distance” from the focus, m) hemorrhages in the retina, n) bloody or xanthochromic cerebrospinal fluid, o) leukocytosis, neutrophilia, an increase in the Krebs index to 6 or more, n) angiography: avascular zone with displacement of the arterial trunks.

Tests of the I level of assimilation (3rd option)

a) Identification tests

Can secondary brainstem syndrome be observed with extensive cerebral infarction of hemispheric localization?

Does hemorrhagic stroke occur due to rupture of a cerebral vessel?

Can hemorrhagic stroke develop due to diapedetic hemorrhage?

Is it possible to use hyperbaric oxygenation in the treatment of ischemic stroke? Can epileptic seizures occur with cerebrovascular accidents?

b) Tests for distinction

1. Specify what signs are characteristic of circulatory disorders in the anterior cerebral artery basin ? 1) Monoplegia or monoparesis of the *leg* 2) Akinesis 3) Hemianopsia 4) Apraxin of the left hand. 5) Visual agnosia 6) Grasp reflex, 7) Increased articular reflexes 8) Astasia-abasia 9) Amnestic aphasia 10} Mental disorders. 11) Urinary incontinence.

11. Indicate which pitchforks of undifferentiated division are used in acute cerebrovascular accidents? 1) Prevention and treatment of breathing disorders. *2)* Maintaining homeostasis. *3)* Anticoagulant and thrombolytic therapy. *4)* Treatment of disorders of general hemodynamics. *5)* Fight against cerebral edema and intracranial hypertension. *6)* Elimination of hyperthermia and other autonomic disorders. " *7)* Drugs that increase blood clotting and reduce vascular permeability. 8) Prevention of stroke complications. 9) Prevention of muscle contractures. 10) Treatment of some manifestations of cerebrovascular accident (repeated vomiting, hiccups, psychomotor agitation, status epilepticus)

Which of these additional research methods are characteristic for *I)* hemorrhagic, 2) ischemic stroke:

a) bloody or xanthochromic cerebrospinal fluid, b; mixing M - no more than 3 mm with echoencephalographic examination, c) leukocytosis over 9,000 with a shift to the left, d) signs of a local decrease or increase in the tone of cerebral vessels *with a* simultaneous decrease in blood circulation on the rheoencephalogram, e) gross and diffuse disturbances in the electrical activity of the brain, f) Filling of the vasculature in the basin of the vessel without displacement or compression of the surrounding areas of the brain during cerebral angiography, g) foci of reduced density in the brain according to computed tomography, h *)* foci of increased density in the brain according to computed tomography, i) Local disturbances in the electrical activity of the brain ...

Tests of the II level of assimilation (1st variant)

Preparation tests

List the pathogenetic mechanisms of ischemic stroke (1-6)

Constructive tests

I Name the types of ischemic stroke. II Nazovige principles of treatment of hemorrhagic stroke.

III What are the indications for surgical treatment of lesions of the carotid arteries.

Patient F., 45 years old, was admitted to the clinic of nervous diseases with complaints of headache in the left half of the head, weakness and numbness *in the* right limbs, especially in the arm. For the last 4 years he has been suffering from hypertension, was treated on an outpatient basis, does not remember blood pressure figures. The morning was awakened by the strong - Noah headache. While washing his right limbs suddenly weakened and the cabin boy

lost his speech. I could hardly pronounce the words and did not understand the speech of those around me, I did not lose consciousness. A day later, an ambulance was delivered to the clinic). P ri admission: general condition of the patient difficult, pulse 66 beats per minute, rit Michna. satisfactory filling. Blood pressure 170/90 mm Hg. Art. Rigidity of the occipital muscles, on the left, a positive Kernig symptom. The tongue deviates to the right when protruding. Right-sided hemiparesis with predominance in the hand, no active movements in the shoulder joint. Restricted flexion and extension are possible in other joints. In the hip and knee joints, the range of motion is almost complete; in the ankle and toes, the step is sharply limited. Right-sided hemihypesthesia. Tendon reflexes are higher on the right, Babinsky's reflex is found on both sides. Motor and sensory aphasia with spontaneous speech intact. Blood test: HB - 7.5 g%, l. 9350, ESR 3 mm per hour. Bleeding time 48 seconds, clotting 11 minutes. Cerebrospinal fluid: pressure 270 **mm of** water, Art., Xanthochromic fluid, protein *0.99 ‰,* cytosis 34/3, Pandey ++ reaction, Nonne-Apelta +. When examining the field of view; reveals right-sided hemianopsia. Echoencephalography: displacement of the M-echo from left to right by 4 mm. CT scan; high density focus 1. Where is the focus localized? 2. Establish a diagnosis. 3. Prescribe treatment.

a) Substitution tests

List the main signs of subarachnoid hemorrhage. (1-4)

b) Constructive tests

I. What are the basic principles of treatment of cerebral vascular crises?

II. What are the indications for surgical treatment of lesions of the carotid arteries.

III. What are the indications *for* surgical treatment of hemorrhagic stroke?

c) Task

Patient S., 58 years old. was admitted to the clinic of nervous diseases with complaints of weakness in the right arm and leg, difficulty speaking. During the last few years (every 2-3 kneading - ya), the patient developed weakness of the right arm, which was held without treatment. During work, she noted weakness in her right arm, added weakness in her right leg, and deterioration of vision in her left eye was noted. Upon admission, the general condition is satisfactory. Pulse - 82 beats *per* minute, rhythmic. Blood pressure -160 / 90-140 / 85 mm Hg. Art. The heart sounds are clear, the emphasis of the second tone is on the aorta. The pulsation of the left common carotid artery is weakened. Neurological status: slight paresis of the facial nerve on the right according to the central type, slight deviation of the tongue to the right. In Barre's test, the right extremities descend somewhat faster . Tendon and periosteal reflexes are higher on the right than on the left. The abdominal reflexes are on the lower right, the plantar reflex on the right is not triggered. Right-sided hemihypesthesia. Blood test: Ni - 106.2 units, Er. - 4,975,000, l, -9,000, ESR-6 mm per hour. Cerebrospinal fluid: pressure 140 mm of water, st., Protein 0.33 ‰ Pandi ++ reaction , cytosis 1/3, negative Wasserman

reaction, Lange reaction - 01121000. Urine analysis without features. On the ECG - sinus rhythm, signs of overload of the right atrium. Fluoroscopy of the chest organs - the heart is expanded to the left, the aorta is compacted. The fundus of the eye - the arteries are sharply narrowed. Rheoencephalography is a decrease in blood circulation in the left hemisphere of the brain. Angiography - stenosis of the left internal carotid artery in the neck. Computed tomography is a focus of low density .

1. Establish a diagnosis. 2. Prescribe treatment.

Tests of the 11 level of assimilation (3rd option)

a) Substitution tests

List the main pathogenetic mechanisms of cerebral vascular crises (1-4)

b) Constructive tests

1. What are the basic principles of ischemic stroke treatment?
2. Name the factors that determine the indications for surgery for cerebral aneurysm .
3. Name the types of cerebral vascular crises.

c) Task

And .. 35 years old, was admitted to the clinic of nervous diseases in serious condition with complaints of a sharp headache. The patient periodically suffered from a headache . In the afternoon, a severe headache, vomiting suddenly appeared, the patient lost consciousness. Within 4 days he was in a soporous state. Expressed Menin gealnye symptoms. The cerebrospinal fluid is bloody. On the 5th day of the illness, mental disorders developed, manifested by psychomotor agitation, aggressiveness, inappropriate behavior, impaired memory and criticism. Of the local symptoms, there was a predominance of tendon reflexes on the right, mitral paresis of the right facial nerve, positive Barre test on the right, mild paresis of the right foot.

1. Establish a diagnosis. 2. Prescribe treatment.

Damage to the meninges

The purpose of the lesson : to teach the student to examine a patient with membrane pathology, evaluate the information received, cause meningeal syndrome, carry out differential diagnostics between diseases accompanied by various physical, biochemical, morphological and biological changes in the cerebrospinal fluid.

The student should know:

1. What is the manifestation of meningeal syndrome?
2. General cerebral syndrome;
3. Symptoms of "stiff neck " Kernig, upper, middle and
4. Lower symptoms of Brudzinsky, hanging and pointing dog;
5. Physiological properties of cerebrospinal fluid;
6. Biochemical properties of cerebrospinal fluid;
7. Morphological properties of cerebrospinal fluid;
8. Biological methods for the study of cerebrospinal fluid;
9. Distinctive features of serous, purulent meningitis and subarachnoid hemorrhage.

The student should be able to:

- to identify signs of cerebral syndrome, syndrome of tonic tension of skeletal muscles or antalgic rigidity and to interpret physiological, biochemical, morphological and biological changes in the cerebrospinal fluid; to carry out differential diagnostics with a different nature of changes in the cerebrospinal fluid.

Lesions of the meninges of the brain. SPINAL FLUID CHANGES

The brain and spinal cord are covered with three membranes: hard, arachnoid and soft.

The dura mater (pachymeninx) consists of two sheets. The outer one is tightly attached to the bones of the skull and spine and is, as it were, their periosteum. The inner layer (actually the dura mater) is a dense fibrous tissue. In the skull, both of these sheets are adjacent to each other, only in places they diverge and form a special wall of the venous sinuses. In the vertebral canal, between the sheets, there is an epidural tissue - loose adipose tissue with a rich venous network.

The arachnoid membrane (arachnoidea) lines the inner surface of the hard membrane and is connected to the soft membrane by many cords. Arachnoidea is, as it were, draped over the medulla and does not sink into the furrows.

The pia mater (eptomeninx) covers the surface of the brain and spinal cord, follows their relief, fuses with the medulla.

The posterior and anterior spinal roots, moving away from the spinal cord to the sides and down, pass through the meninges. Therefore, meningitis and other meningeal lesions can involve roots in the process. There is a space between the soft and arachnoid membranes, called the subarachnoid. It circulates cerebrospinal fluid - cerebrospinal fluid. At the base of the brain, the subarachnoid space expands and forms large cavities filled with cerebrospinal fluid (basal cisterns). The largest of them is located between the cerebellum and the medulla oblongata - cisterna cerebellomedullaris. In the spinal canal, the subarachnoid space surrounds the spinal cord. From the level of its end (vertebrae L1-L2), this space increases in diameter and becomes the receptacle of the cauda equina (terminal cisterna - cisterna terminalis). Cerebrospinal fluid is also located inside the brain and spinal cord, filling the ventricular system: right and left lateral, third ventricle, sylvian aqueduct, fourth ventricle, central spinal canal. From the fourth
of the ventricle, it enters the subarachnoid space through the paired opening of Magendie and the unpaired opening of Lyushka in the posterior cerebral parus. Cerebrospinal fluid is formed in the cells of the choroid plexus of the brain. This was the reason for some authors to call it plexus coryoideus and tela coryoidea - the choroid gland of the brain.

The amount of this fluid in humans is relatively constant. On average, it is equal to 120-150 ml. Most are located in the subarachnoid space. The ventricles contain only 20-40 ml. It is produced continuously in an amount of 600 ml during the day and is also continuously absorbed into the venous sinuses of the dura mater of the brain through the arachnoid villi. The accumulation of such villi in the venous sinuses (there are especially many of them in the upper sagittal sinus) are called pachyon granulations. The inflow and outflow of this fluid ensure the constancy of its volume in the ventricles and in the subarachnoid space. The liquid is partially absorbed into the lymphatic system through the sheaths of the nerves, which are a continuation of the meninges. The movement of cerebrospinal fluid in different directions is associated with the pulsation of blood vessels, breathing, movements of the head and trunk.

The physiological significance of cerebrospinal fluid is diverse. First of all, it is a hydraulic cushion of the brain, which creates mechanical protection against shocks and concussions. At the same time, it also turns out to be an internal environment that regulates the processes of absorption of nutrients by nerve cells, maintaining osmotic and oncotic balance in them. Cerebrospinal fluid also has protective (bactericidal) properties, and antibodies accumulate in it. She takes part in the mechanisms of regulation of blood circulation in the closed space of the cranial cavity and spine. Cerebrospinal fluid circulates not only in the ventricles and in the sub-arachnoid space, it penetrates into the thickness of the medulla through the so-called perivascular fissures (Virchow-Robin space). A small amount of it also enters the peri-endoneural clefts of the peripheral nerves. It should be noted that in the first weeks of embryo development, the neural tube is supplied with cerebrospinal fluid before the development of the vascular system. As the microcapillary bed in the brain matures, its nutrition is provided mainly by blood. However, the cerebrospinal fluid route of delivery of nutrients to neurons is also preserved in adults. Some of the glial cells have a pumping function and within 1 min sucks up the cerebrospinal fluid from the ventricular system 8-10 times. Therefore, the dimensions of the ventricular system change the same number of times per minute: they decrease during the absorption phase of the cerebrospinal fluid and increase when the cerebrospinal fluid returns to the ventricular system with the products of neuronal metabolites. In manual medicine, this liquor phase of nutrition is referred to as the “primary respiratory mechanism of the brain” and can be detected with cranial manual therapy techniques. In addition, when examining the cerebrospinal fluid extracted even with lumbar puncture of the dural sac, it is possible to detect pathological changes in the fluid in various diseases of the brain substance.

Meningeal Syndromes

With meningitis of various etiologies, as well as with hemorrhages in the subarachnoid space and with some other pathological conditions, a clinical picture develops, called the meningeal irritation syndrome, or, in short, meningeal syndrome. Its frequent components are: headache, vomiting, pain during percussion of the skull and spine, hypersensitivity (general hyperesthesia) to light, sound and skin irritations. Typical signs of irritation of the meninges are tonic tension of some groups of skeletal muscles: 1) muscles that extend the head; 2) muscles - flexors of the hip and knee joints. In severe forms of meningitis, persistent tonic tension of the listed muscle groups leads to the formation of a peculiar posture. The patient lies on his side, the head is thrown backward, the thighs are pressed to the abdomen, the shins to the thighs. Sometimes tonic tension extends to the muscles that extend the spine, which is indicated by opistho-nus. The described forced position of the body in such a pronounced degree occurs relatively rarely, however, increased tension of the listed muscles a constant phenomenon with meningitis. An attempt to passively bend the head anteriorly (to bring the chin to the chest) with irritation of the meninges meets resistance due to the emerging reflex tension of the posterior cervical muscles. This symptom is called the stiffness of the occipital muscles. It is more correct to talk about the rigidity of the posterior muscles.

For meningitis, the symptom described by the St. Petersburg clinician VM Kernig in 1882 is characteristic. Kernig's symptom is detected as follows. In a patient lying on his back, the doctor bends the leg at the hip and knee joints at a strictly right angle, and then, in this initial state, tries to straighten the leg at the knee joint, which, in the case of meningeal syndrome, meets resistance. The flexors of the lower leg are tonically tense; it is usually not possible to straighten the leg at the knee joint. Sometimes pain appears in the muscles (flexors of the lower leg), less often in the lower back and along the entire spine.

In the study of the tone of the posterior cervical muscles (test for the stiffness of the muscles of the occiput), as well as in the Kernig test, in addition to the above, there are also distant reflex-motor phenomena. They are called Brudzinsky's meningeal symptoms.

Bending the head forward causes slight flexion of both legs in the hip and knee joints - the upper symptom of Brudzinsky, and the same flexion movement in the contralateral leg during the Kernig test - the lower symptom of Brudzinsky. The same movement of the legs can cause pressure on the pubic articulation - the average symptom of Brudzinsky. When a child is lifted by the armpits, reflex bending of the legs and bringing them to the stomach is observed - a symptom of "hanging" according to Le Sage.

What is the mechanism of movement (tonic) disorders during irritation of the meninges? There is a widespread point of view that tonic tension of the posterior cervical muscles and muscles - flexors of the lower leg in meningitis is a reflex defense reaction that reduces the tension of the posterior roots and relieves pain.

Currently, in the above-described motor nine
disorders, you can see an increased tonic reflex of the muscles to stretch. One has to think that when testing the rigidity of the occiput, when Kernig's symptom is detected, additional stretching of the corresponding muscles occurs and the most dramatic manifestation of the tonic myotatic reflex.

In conclusion, it should be said that if there are symptoms of irritation of the meninges and inflammatory changes are found in the cerebrospinal fluid, then a diagnosis of meningitis is established. Blood in the cerebrospinal fluid indicates subarachnoid hemorrhage. However, with various diseases (pneumonia, appendicitis, etc.), especially in children, symptoms of irritation of the meninges can be detected without any changes in the cerebrospinal fluid. In such cases, they speak of meningism.

It is very important for the clinic to study the circulation of the cerebrospinal fluid and changes in its composition in pathological conditions.

There are three ways to obtain cerebrospinal fluid from a patient: 1) lumbar puncture of the final cistern; 2) suboccypital puncture of the cerebellar-medullary cistern; 3) puncture of the lateral ventricle through the milling hole in the skull.

LUMBAL PUNCTION

The most accepted in the clinic is a lumbar puncture, or lumbar puncture. This procedure is relatively safe, its technique is simple. Puncture can be done in the patient's position and sitting and lying. They prefer the latter. The patient is placed on a firm bed (trestle bed, table, bed with a shield) in a lateral position.

The legs should be bent at the hip and knee joints. The head is tilted until the chin touches the sternum. The body is strongly bent forward so that the spinous processes protrude and the gaps between them increase. The surface of the back should be vertical in relation to the bed, and the spinous processes should be in a horizontal line. The head can be laid on a flat pillow. In the given position, the patient is held by an assistant (orderly, nurse). The patient must be warned to lie still, not make any movements during the procedure.

To determine the puncture site, the most elevated points of the iliac crests are probed, marked and connected with a straight line using an iodine solution. This is the so-called Jacobi line: it runs at the level of the spinous process of the L4 vertebra (according to some authors, in the interval L3-L4.). The puncture is performed between the spinous processes L3-L4 or L4-L5 (in adults, in addition, between L2-L3). At these levels there is no longer a spinal cord, the cauda equina roots are washed by the cerebrospinal fluid.

move away from the needle and are usually not injured during the puncture. Special needles are used to carry out a lumbar puncture. The doctor treats his hands in one of the ways recommended in surgery. After that, you can only touch sterilized items, and the patient's skin - after it has been treated with alcohol, 5% iodine tincture. The skin around the puncture site is twice treated with 70-degree alcohol, then smeared with iodine tincture. Excess iodine is removed with a gauze ball moistened with alcohol. Then local anesthesia is performed with 0.5% novocaine solution. It turns out "lemon peel". It is not recommended to introduce novocaine into the subcutaneous tissue, as it impairs the palpation orientation of the spinous processes. Novocaine in an amount of 3-5 ml is infiltrated along the course of the future puncture to a depth of 2-4 cm.Waiting for about half a minute, the doctor injects a puncture needle directly under the spinous process of the superior vertebra (usually 3 or,) strictly along the midline with a very slight upward slope (the angle between the line of direction of the needle and the surface of the back should be approximately 80 °). The needle should be injected slowly and smoothly. Piercing the skin, ligaments, the doctor experiences tangible resistance. After passing through the dura mater (in adults it happens at a depth of 4-7 cm, in children - up to 3 cm), the resistance ceases and there is a feeling of "failure" of the needle. After that, the mandrel is carefully removed (usually not completely) from the needle. Noticing the release of fluid, immediately insert the tip of the connecting tube of the manometer into the cannula of the needle and measure the cerebrospinal fluid pressure. In the supine position, it is equal to an average of 100-180 mm of water. Art. In the sitting position, the cerebrospinal fluid pressure is slightly higher - 200-300 mm of water. Art. A simple water pressure gauge is usually used to measure CSF pressure.

Pathological processes localized in the cranial cavity and in the spinal canal can disrupt the patency of the cerebrospinal fluid. This is especially pronounced in the spinal canal (with complete or partial spinal block). The block can be caused by a tumor, herniated intervertebral disc, bone compression with a compression fracture of the vertebra, adhesions with adhesive leptopachymeningitis.

Under normal conditions, there is a close relationship between venous and cerebrospinal fluid pressure. The liquorodynamic tests described below during the block are based on the registration of a violation of this ratio.
Kweckenstedt test. The assistant doctor covers the lower part of the neck with the index and thumb of both hands and squeezes the cervical veins for 5-10 seconds. The onset of an increase in venous pressure B of the cranial cavity is judged by the swelling of the facial and temporal veins, by redness with a bluish tint of the skin of the face and the sclera of the eyeballs. Venous congestion of the head
the brain leads to an increase in intracranial pressure and an increase in cerebrospinal fluid pressure. The height of the cerebrospinal fluid column in the manometric tube increases significantly. After the cessation of the compression of the veins, the cerebrospinal fluid pressure rapidly decreases to the initial level. All this happens with a passable subarachnoid space. With a complete block of the cerebrospinal fluid pathways within the spinal cord, compression of the cervical veins does not increase the cerebrospinal fluid pressure. With a partial block, the cerebrospinal fluid pressure rises slightly and slowly decreases after the cessation of compression.
Pussep's test . The patient bends his head to his chest. In this case, there is a partial compression of the cervical veins. The cerebrospinal fluid pressure at this moment rises by 30-60 mm of water. Art. Returning the head to its original position lowers the cerebrospinal fluid pressure to the previous figures. With a block of the subarachnoid space, Pussep's test does not increase the cerebrospinal fluid pressure.
Stukey's test . The doctor's assistant squeezes the abdominal wall with his hand at the level of the navel for 20-25 seconds. As a result, the abdominal veins are compressed and stagnation occurs in the venous system of the spinal canal. The cerebrospinal fluid pressure rises by 1-1.5 times. It decreases to the initial level after the cessation of compression. This reaction of CSF pressure persists in the presence of a block of the subarachnoid space at the level of the cervical or thoracic spine.
It is customary to depict the results of liquorodynamic tests graphically: the phase of one or another moment of the experiment is marked horizontally, and the pressure height is marked vertically.
After completing the measurements of the pressure dynamics, the cerebrospinal fluid is collected in test tubes, usually in an amount of 3-8 ml for further analysis in the laboratory. The amount of extracted cerebrospinal fluid is commensurate with the patient's condition, the nature of the disease, the level of pressure, the tasks of the upcoming study. At the end of the above manipulations, the puncture needle is quickly removed, the puncture site is lubricated with iodine and closed with a ball of sterile cotton wool (preferably moistened with collodion). The patient is prescribed bed rest for 2-3 days. The first 1.5-2 hours after the puncture, it is recommended to lie on your stomach without a pillow.
Suboccipital and ventricular punctures are usually performed by a neurosurgeon. In most cases, 5-8 ml of cerebrospinal fluid is extracted without
complications. Occasionally, post-puncture meningism is observed (lasting for several days, headache, vomiting may appear). However, there are diseases in

which a lumbar puncture is life-threatening and requires special care. These include brain tumors, especially when located in the posterior fossa. If a tumor of the posterior cranial fossa is suspected, with large intracranial hypertension with congestive nipples, the cerebrospinal fluid is released in a minimum amount (1-2 ml) in slow drops (the lumen of the needle is reduced with a mandrel). It is necessary to have a well-ground syringe and a warm saline solution ready for forced administration when signs of wedging appear. Lumbar puncture in such patients is recommended to be performed in a neurosurgical department. Caution requires the appointment of a puncture to patients with a tumor of the spinal cord (possibly increased paresis and sensitivity disorders - the so-called "wedging" syndrome). After extracting the required amount of cerebrospinal fluid, it is advisable to repeat the measurement of the cerebrospinal fluid pressure, it is almost always below the initial level.

Of practical importance can be the determination of the Ayala index (Aua1a) according to the following formula: the amount of liquor taken (ml) is multiplied by the value of the residual pressure of the cerebrospinal fluid (mm of water column), the resulting product is divided by the value of the initial pressure (mm of water column). In healthy people, the index ranges from 5.5 to 6.5. An index of more than 7.0 indicates hydrocephalus or serous meningitis, an index of less than 5.0 is typical for a block of the subarachnoid space.

SPINAL FLUID

Under normal conditions, cerebrospinal fluid has a specific gravity of 1.005-1.007, its reaction is weakly alkaline, pH, like blood, is close to 7.4, the amount of protein is from 0.15 to 0.45 g / l. Inorganic substances in the cerebrospinal fluid are contained in a proportion approximately the same as in the blood: chlorides - 720-740 ml%, potassium - 16-20 mg%, calcium 5-6.5 mg%. The concentration of glucose in the cerebrospinal fluid is about 70% less than in the blood serum, and is equal to 3-3.5 mmol / L of glucose. The composition of the cerebrospinal fluid is highly dependent on the function of the blood-brain barrier. Apparently, this biological septum, separating the bloodstream from the cerebrospinal fluid, is represented, on the one hand, by the capillary wall, and on the other, by glia, in particular astrocytic. For the existence of such
barrier indicates the difference in the composition of blood and cerebrospinal fluid. Many substances circulating in the blood do not enter the cerebrospinal fluid. This also applies to many drugs. Therefore, when treating neurological patients, it is sometimes necessary to inject drugs (in particular, antibiotics) not into the blood, but directly into the cerebrospinal fluid (intrathecal injection). Normally, there are some differences in the composition of the ventricular and lumbar cerebrospinal fluid.

Analysis of cerebrospinal fluid in the laboratory is carried out according to the following program: 1) analysis of physical properties; 2) morphological analysis; 3) biochemical analysis; 4) bacteriological and virological analysis; 5) immunological analysis.

Analysis of physical properties. Normally, transparent and colorless (indistinguishable from water in appearance), cerebrospinal fluid with meningitis can become cloudy due to the presence of a large number of cellular

elements. Sometimes the fluid acquires a greenish-yellow color - this so-called xanthochromia is observed in some meningitis, after subarachnoid hemorrhage, in brain tumors. Xanthochromia can be combined with a tendency of the cerebrospinal fluid to massive coagulation - this congestive Frouant's syndrome, or Nonne's compression syndrome, is more often observed with volumetric processes in the lower half of the spinal cord.

Morphological analysis. Normal cerebrospinal fluid contains in 1 ml up to 5 formed elements such as lymphocytes. Under pathological conditions, their number can increase to tens, hundreds, and even thousands. This phenomenon is called pleocytosis. To count the number of cells in 1 ml of cerebrospinal fluid, a mixer for blood leukocytes and a Fuchs-Rosenthal or Goryaev counting chamber are used. Cells are counted in all 256 small squares of the chamber. The volume of liquid above the mesh is 3.2 ml. It is customary to do this: the denominator 3 is added to the number of white cells counted in the entire grid of the chamber (erythrocytes are not included in the count). The counting result takes the form: 4/3 or 84/3, etc. These samples correspond with sufficient accuracy for practice the number of white cells in 1 ml of cerebrospinal fluid. Normal fluid contains only cells of the lymphocyte type. In pathological conditions, the cerebrospinal fluid is found in addition to lymphocytes

Cerebrospinal fluid composition at different levels

Puncture	Pressure. mm. water column	Protein count g / l	Cell count Mcmol / L	Glucose count
Lumbar	70-200	0.15-0.45	0-5	3.0-3.5
Suboccipital	70-190	0.25-0.30	0-3	3.0-3.5
Ventricular	70-190	0.50-0.15	0-1	3.5-4.4

plasma cells, monocytes, mast cells, neutrophilic and eosinophilic leukocytes, histiogenic cells (including macrophages). Sometimes, tumor cells are found in the cerebrospinal fluid (sarcomatosis, carcinomatosis of the meninges, cerebellar

medulloblastoma, other brain neoplasms located close to the cerebrospinal fluid pathways). Such "findings" are of great diagnostic value.
Biochemical analysis. It is convenient to start the study of cerebrospinal fluid with the Pandy protein test. The reagent is a saturated solution of carbolic acid. Reaction technique: adding 1 drop of cerebrospinal fluid to 0.5-1 ml of reagent. With a positive reaction, turbidity occurs, the degree of turbidity is indicated by the number of crosses (from 1 to 4). The reaction indicates an increase in the protein content in the cerebrospinal fluid.
The quantitative determination of the total protein content is carried out according to the Roberts-Stolnikov method by adding strong nitric acid to different dilutions of the cerebrospinal fluid with saline. Determine the maximum dilution that will be the first to form a white ring at the border of the two liquids. According to the found critical level, the figure for the total protein content is recognized from the table. Normal cerebrospinal fluid contains 0.15-0.45 g / l - an amount that is many times less than that of blood serum.
The Nonne-Apelta reaction approximately reveals the content of the globulin fraction of the protein in the cerebrospinal fluid. The essence of the reaction is the precipitation of this protein fraction with a saturated solution of ammonium sulfate. In a test tube, mix equal volumes of this solution and cerebrospinal fluid. The increased content of globulins is revealed by the turbidity of this mixture. There are four gradations of turbidity, expressed by the number of crosses.
In the study of cerebrospinal fluid, in addition to the above, colloidal-chemical methods are also used. They are based on the property of pathological cerebrospinal fluid to disrupt the stability of an artificially prepared colloidal solution of gold (the so-called goldsol - gold sol). The transition of a sol into a gel during this reaction is clearly visible with the "naked eye". From red, the solution turns to red-violet, violet, red-blue, blue, light blue, or becomes colorless.
The reaction of colloidal gold is carried out with different dilutions of the cerebrospinal fluid with a NaCl solution. It should be borne in mind that artificially prepared colloidal dispersed systems can be disturbed by the introduction of certain concentrations of sodium chloride solution. To dilute the cerebrospinal fluid, it is necessary to take such a concentration of the solution of this salt, which leaves the colloidal solution stable, retaining its red color in the reaction being carried out. This concentration is usually a 0.4 percent solution. The reaction of colloidal gold in cerebrospinal fluid, proposed by Lange in 1912 and designated by his name, is carried out with different dilutions of sodium chloride solution (from 1: 10 to 1: 20,000-1: 32,000). It turns out a series of 12-16 test tubes with an increasing degree of dilution of the cerebrospinal fluid. Each of them is then poured into 5 ml of freshly prepared colloidal gold. The reaction results for each dilution of CSF are plotted graphically on a pre-prepared grid. On the horizontal, the degree of dilution of the solution is noted, on the vertical, the gradation of the change in the color of the reagent. The intersection points of the vertical and horizontal lines are connected, a curve is obtained that depicts the result of the reaction. The curve formed on the grid shows the ratio of the degree of change in the colloidal solution at different dilutions of the cerebrospinal fluid. In order to save space, the graphic image is replaced with a conventional row of numbers. The number denotes the

gradation of the color change of the colloidal solution, and the ordinal position of the number in the row is the degree of dilution of the cerebrospinal fluid.
The reaction of colloidal gold is positive in neurosyphilitic diseases, in brain tumors, meningitis of various etiologies. The maximum change in the reagent does not always coincide with the highest concentration of cerebrospinal fluid. With tumors and meningitis, a so-called shift of the curve to the right is observed, the maximum change in the color of the reagent falls on the average degree of dilution of the cerebrospinal fluid. In neurosyphilitic diseases, the colloidal state of the reagent is more disturbed within the left half of the grid (shift of the curve to the left). With progressive paralysis in five to six test tubes on the left, the reagent turns out to be completely discolored, with other neurosyphilitic diseases, the curve in this zone forms a tooth in the downward direction. Its maximum change falls on dilutions of 1: 40-1: 80.
Bacteriological and virological analysis. In a number of infectious diseases of the nervous system, it is necessary to conduct a bacteriological examination of the cerebrospinal fluid in the form of bacterioscopy and sowing it on nutrient media. Only in this way is it possible to establish an etiological diagnosis with complete reliability and prescribe the most rational treatment. This primarily applies to the diagnosis of purulent meningitis.
In some cases, it is necessary to send the cerebrospinal fluid to the virological and immunological laboratory for a special study (polymerase chain reaction - PCR, determination of the level of autoantibodies to non-NMDA type glutamate receptors, sensitivity of immunocytes to neurospecific antigens, etc.). The main fractions of immunoglobulins were found and quantified in the cerebrospinal fluid: JgA is a carrier of specific antibodies, its concentration is 1-5 mg / l; JgG - its biological function is to neutralize viruses and toxins, it comes from blood plasma and has a concentration of 5-50 mg / l; JgM - has a large molecule size (M = 800,000 nm) and passes little through biological barriers, therefore its concentration in the cerebrospinal fluid is only 0.1-0.6 mg / l.
Finally, if a syphilitic disease of the nervous system is suspected, a Wasserman reaction and any of the specific serological reactions (RIF, RIBT) should be performed in the cerebrospinal fluid. It should be borne in mind that the Wasserman reaction in the cerebrospinal fluid is carried out according to a special technique in which more cerebrospinal fluid is required than serum (up to 1 ml).
In conclusion, let us dwell on the description of some characteristic syndromes of changes in the cerebrospinal fluid observed in the clinic (table and). In some cases, patients have an isolated increase in the amount of protein (hyperproteinosis) in the cerebrospinal fluid, the number of cells remains unchanged. This picture is called protein-cell dissociation. It is found in tumors of the brain and spinal cord, in the spinal cicatricial adhesive process with a block of the subarachnoid space.
More often, however, a simultaneous increase in the number of cells (pleocytosis) and an increase in the amount of protein (hyperproteinosis) are observed in the cerebrospinal fluid. This picture of cerebrospinal fluid occurs with meningoencephalitis, meningomyelitis and meningitis of various etiologies. Already the sight of cerebrospinal fluid (turbidity) makes one think of meningitis. Cell counting reveals pleocytosis. Globulin reactions are positive, the

total protein content is increased. Examination of a smear from the cerebrospinal fluid sediment can reveal predominantly neutrophilic pleocytosis, the presence of gram-negative diplococci. Especially their intracellular localization raises suspicion of meningococcal meningitis. If gram-positive extracellular diplococci are found, one has to think about pneumococcal meningitis. To clarify the diagnosis, additional studies are required, in particular, cerebrospinal fluid cultures, which should be carried out

Features of cerebrospinal fluid in some diseases

Disease	Pressure, Mm water column	Color	Amount cell	Amount protein g / l	Amount of glucose, Mcmol / l
Norm	70-200	Colorless	0.15-0.45	0-5	3.0-4.4
Brain tumor -	Increased up to 600	Colorless or xanto to 10 lame	Increased	Rate (5-6)	Rate
Meningitis:					
serous	Increased	Colorless	Norm	Pleocytosis (lymphocytes)	Reduced
purulent	Increased	Muddy (whitish, up to 0.9 greenish)	Increased	Pleocytosis (neutrophils)	Reduced
Subarachno- distant outburst	Increased blood	Bloody (color of fruit drink)	Increased up to 1.0	Fresh or you- alkaline (erythrocytes)	Increased

according to all the rules of bacteriological technology. To avoid contamination, they are best done so that the tube with the culture medium is directly placed under the drops of liquid from the needle during lumbar puncture. If purulent meningitis is suspected, culture media containing blood or blood serum are most suitable. It should be mentioned that by bacterioscopy of the cerebrospinal fluid and sowing on Sabouraud's medium, a rare form of cryptococcal (torulosa) meningitis can be detected, caused by one of the types of yeast fungus.

With tuberculous meningitis, the cerebrospinal fluid may remain clear. However, in many cases, after 12-24 hours, a thin cobweb-like film appears in the cerebrospinal fluid in the test tube. From this film, mycobacteria can be sown, protein samples are positive. The number of formed elements is increased, but to a lesser extent than in purulent meningitis, lymphocytes usually predominate. In a smear from a film when stained according to Ziehl-Nielsen, tubercle bacilli can be found. Sowing the cerebrospinal fluid on a nutrient medium special for Koch's rods can give growth only after a long period of time, measured in weeks. In the composition of the cerebrospinal fluid in tuberculous meningitis, there is another peculiar feature: the amount of sugar tends to decrease.

In some diseases, pleocytosis in its severity prevails over the degree of protein increase - cellular-protein dissociation. This occurs in many neuroinfections.

A very important symptom may be the presence of erythrocytes or their decay products in the cerebrospinal fluid. This sign indicates the penetration of blood beyond the vessel wall and is important in the differential diagnosis of cerebral stroke. The same must be said about a significant admixture of blood to the cerebrospinal fluid, which happens with subarachnoid hemorrhage. In both cases, the question arises whether the admixture of blood is accidental, depending on the trauma of the vessel with a puncture needle, or the patient has a hemorrhage in the cranial cavity or in the spinal canal.

A technique has been developed with the help of which it is possible to establish that the blood entered the cerebrospinal fluid accidentally. To do this, collect the liquid dripping from the puncture needle into several test tubes. If in each subsequent test tube the liquid is cleared of blood more and more, it means that its source was close and the incoming new portions of the cerebrospinal fluid do not contain blood. In such cases, erythrocytes in test tubes quickly settle, the above sedimentary liquid remains colorless. Quickly and clearly, the so-called path blood can be distinguished from blood from the subarachnoid space by applying a drop of cerebrospinal fluid flowing from the puncture needle onto white filter paper. If the spreading spot has a uniform pink or red color, this indicates hemolysis due to prolonged contact of blood with cerebrospinal fluid in the subarachnoid space. In case of accidental ingestion of blood, the stain has two zones: red (with aggregates of erythrocytes) in the center and colorless (from diffusion of normal cerebrospinal fluid) along the edges.

If an admixture of blood in the cerebrospinal fluid is associated with hemorrhagic stroke, the fluid above the sediment has a yellowish color (xantho-chromium). In a smear from the sediment under a microscope, you can see the collapsed erythrocytes, sometimes macrophages, loaded with blood pigment.

Control questions

1. What is included in the concept of "meningeal syndrome"?
2. What are the differences between the symptoms of meningitis and meningism?
3. What is a subarachnoid spinal block and how to identify it?
4. What are the normal values of protein and cells in the cerebrospinal fluid?
5. What is protein-cell dissociation and under what diseases does it occur?

Infectious diseases of the central nervous system.

The purpose of the lesson : to study infectious diseases of the nervous system, their classification, clinical manifestations of meningitis, encephalitis, myelitis, methods of diagnosis, treatment, disease prevention.

The student should know:

1. The concept of infectious diseases of the nervous system;
2. Etiopathogenesis of certain nosological forms;
3. The course and clinical characteristics of diseases;
4. Clinical features of individual forms and their treatment.

The student should be able to:

1. Examine a neurological patient;
2. Identify neurological syndromes and establish the localization of the pathological process;
3. Establish a clinical diagnosis;
4. Prescribe adequate treatment.

General Provisions

Title of infectious diseases of the nervous system usually orders - INDICATES any of its sections preferably impressed in this case. PLAYBACK - Lenie meninges referred to as meningitis, arachnoiditis, pahime Ning; inflammation of the brain - encephalitis, inflammation of the spinal cord - myelitis, selective loss of gray matter of the spinal cord - Polio; a combination of lesions of the meninges and brain brain - menigoentsefalit, brain and spinal cord damage - Enz - falomielit, peripheral nerve damage - neuritis, etc. More op - definiteness topical diagnosis of infectious lesions of the nervous ICU - themes reflect the terms such as "basal meningitis", "stem en - Cefalu ', etc. It is important to specify the nature of the disease has led to the title - Sheha infective disease, such as viral meningitis, tank - ter encephalitis. If refined to

cause infectious SHOCK Nia nervous system, its name may reflect the etiology Zabolev - Nia, such as tuberculous meningitis, encephalitis, measles, etc.

The clinical picture of infectious diseases of the nervous system is determined by the nature of the causative agent and is particularly striking Institute - fektsiey tissues, as well as a response to infection: immune system orga - nism. Features of the clinical picture of the disease make it possible to suspect an infectious lesion of the nervous system and the nature of the morphological structures involved in the pathological process; clarifying the diagnosis of CSF provided with lumbar puncture, as well as serological and microbiological tests on - allows one to determine the nature of the causative agent of an infectious process. All this serves as a basis for carrying out in each case yn - infectious lesions of the nervous system and the most efficient eff - cient treatment tactics

Men and ng and you

Meningitis is an inflammation of the lining of the brain. They can take the form of leptomeningitis (inflammation of the soft and arachnoid membranes), arachnoiditis (inflammation mainly of the arachnoid membrane), and pachymeningitis (inflammation of the hard membrane).

The term "meningitis" meningitis generally denoted at koto - rum infection spreads by the liquor paths, the process involved in soft and arachnoid membrane; if the infection enters the ventricles of the brain, the process is complicated by inflammation of their walls - the ventricle lithium. When ventricular primarily occurs inflammation vystilayusche - th layer intracerebral liquor spaces ependymal cells (ependymoma, or ependymitis) and the choroid plexus of the brain ventricles (choroiditis); the usual combination of both may be referred to as choreoeppendymitis, or chorioependymatitis.

Chorioependymatitis is usually accompanied by an increased secretion of 1DSF, which predisposes to the development of hypersecretory hydrocephalus, and with narrowing and even more overlapping of the apertures of the ventricular system of the brain , occlusive hydrocephalus is also possible. If the inflammation spreads to a large extent on the substance of the brain, the Global Developing - etsya meningoencephalitis.

Classification. Meningitis classified according to *the nature of the WHO - arouser.* This makes it possible to divide them primarily on meningitis of bacterial - nye, viral, fungal, etc. Clarification of the cause meningitis excitaton. - turer infection makes it possible to include in the name of the process and mixed - Niya about its etiology, such as meningococcal meningitis, sifiliti - cal meningitis, etc. By *nature of the inflammatory process* meningitis differentiate on *purulent and serous,* for *clinical course* menin - HIT may be *acute, subacute* , or *chronic;* with meningococcal meningitis, its fulminant form is also possible.

Meningitis can be primary or secondary. Primary menin - git develops without previous overall infection or previously originated - Sheha infectious focus in the body, while the secondary is the oc - complication in the other infectious diseases, inflammatory SHOCK - Nij organs and systems. Atrium at primary IU - Ning are the mucosa of the nasopharynx, bronchi, gastro-ki - antiplaque tract. Secondary meningitis is metastatic, contact or traumatic. Infection of the fetus is possible through the parade ground - one way or during passage through the birth canal.

Ways of spreading the infection. The causative agents of meningitis enter the meninges in different ways. The most common is the hematogenous route, at which the primary **infectious** focus (e.g., furuncle person or purulent wound in the paravertebral region) has direct suck - kyanite connection (arterial, venous, or both) with shells th - Karlovna or spinal cord. Possible introduction of infection in the brain is about - span of from the primary pathological focus and *lymphatic system.* In addition, the infection can spread *by contact,* if the primary focus of infection (purulent sinusitis, etmoidit, mezatimpa filaments, etc.) is in close proximity to the brain hull - Cam. Purulent meningitis may also be due to the open Prony - jacent traumatic brain or spinal trauma, in particular trauma, complicated liquorrhea (the liquor fistula). It may be a consequence of infection in the cerebrospinal fluid introduction paths when Punk - tion or their large neurosurgical operations.

C l i n i c a l m a n i f e s t a t i o n s . The clinical picture of any form of meningitis is formed on the background of signs common to all infectious diseases (general malaise, pyrexia, signs yn - intoxication). Moreover, in the case of the development of meningitis, manifestations of intracranial hypertension and meningeal syndrome are typical .

D i a g n o s t i c s . Almost always, clinical symptoms make it possible to recognize the presence of meningitis, however, only that diagnosis of meningitis, which is confirmed by the results of a liquorological study, is considered reliable. CSF study - the only way to - conductive confirm the diagnosis of meningitis and determine its originator. The investigations - dovanie CSF is suspected, patients have meningitis should be made immediately. Already by color CSF obtained by dia - gnostic lumbar puncture (see chap. 32), it can usually be otdif - ferentsirovat from meningitis and subarachnoid hemorrhage purulent meningitis of serous. With purulent meningitis, CSF is cloudy, milky white or greenish yellow; with serous it is transparent, sometimes opalescent. Pressure CSF in all forms of meningitis is high, and it usually detected cell-protein dissociation due expressed - Nogo increasing number of cellular elements. The number of cells in CSF in purulent meningitis in the thousands and tens of thousands, with lo - discord meningitis -. More hundreds thousand to 1, and occasionally a few larger in 1 ul. With purulent meningitis, predominantly neutrophilic pleocytosis is characteristic; macrophages and reticular cells are also detected. Serous meningitis is usually dominated by mononuclear cells, mainly lymphocytes. When purulent meningitis as well as in tuberculous meningitis in CSF increased protein content (up to 1 - 10 g / l in connection with - Rushen BBB permeability), while dramatically positive (++++) sedimentary samples Pandey and Nonne-Apelt ... The glucose content in the CSF with bacterial meningitis (especially with purulent, tuberculous and fungal meningitis) is reduced, with viral meningitis it is within normal limits or increased. Reduced chloride content and increase lactate (lactic acid) in CSF is observed in purulent forms IU - ningita. The most important diagnostic value is the detection of meningitis pathogens in the CSF .

In the blood with purulent meningitis, as a rule, there is a neutrophilic pleocytosis with a shift of the leukocyte formula to the left, an increase

in ESR. When serous meningitis in the blood changes are less typical, the WHO - are possible leukopenia, lymphocytosis. Verification meningitis pathogen can contribute to the results of bacteriological or virusologiches - who studies. But sow microorganisms from the blood of patients with purulent meningitis often (more than 25% of cases) can not, as of - in the blood or CSF naruzhenie viruses involves great technical assistance with - Kimi difficulties and can not always informative.

Since the bacteriological diagnosis requires a considerable expenditure of time, suspected meningococcal meningitis may be employed methods express diagnostics - detecting CSF spe - cific antigens (protein components meningitis pathogen) with using group precipitating sera using counter immunopheresis or by latex agglutination.

The appearance of brain with meningitis and focal neurological symptoms and paroxysms of convulsive indicates involved - SRI inflammatory process in the brain substance, the effect on it infection Onno-toxic factors (encephalitic reaction or meningoencephalitis). In this case, cerebral edema is usually noted, and with meningoencephalus , displacement of individual cerebral structures, cerebrovascular disorders, in particular thrombus formation, hemorrhage into the brain tissue are possible .

If a patient is suspected of meningitis, his urgent hospitalization in a specialized department is necessary , where isolation in a box is possible. In severe clinical manifestations of meningitis usually req - Dimo intensive care and sometimes intensive care Mero - enterprises. Before transporting it is advisable to enter a patient guy - teralno analgesics, dehydrating agents. Thus on dogospi - Talnoye stage desirable to begin treatment with antibiotics. However, in severe cases of bacterial meningitis occurring with the high - perature, disturbance of consciousness, with signs of infection- toxic shock urgent need administering to a patient intravenously 3 000 000 IU benzylpenicillin crystalline sodium salt and 60 mg prednisolone. It is very desirable that prior to the introduction antibioti - ka doctor ambulance patient was taking blood samples for bacteria - ologicheskogo research.

In the hospital, a diagnostic lumbar puncture is urgently carried out with measurement of cerebrospinal fluid pressure, after which CSF is slowly collected through a mandrel into different test tubes for general, biochemical and bacteriological research. For suspected tuberculous meningitis collected in a separate tube CSF to identify ha - acteristic for him gentle fibrin film; for this test tube mouth - navlivaetsya state and remain there for at least 12 hours, then carefully (avoiding shaking) can be seen on a dark background.

General p r i n c i p l e s o f t r e a t m e n t . When purulent meningitis should be as soon as possible to begin antibiotic therapy: intravenously administered - but large doses of antibiotics and then clarify the nature of containing - camping in the CSF of bacteria and their sensitivity to antibiotics and sulfanyl - amides possible correction of antibiotic treatment.

In viral meningitis on individual indications apply - Xia interferon preparations RNase or DNase, immunoglobulins. Antibiotics and sulfonamides

are prescribed in such cases only to prevent and treat bacterial complications or concomitant lesions.

All forms of meningitis are accompanied by intracranial hypertension, therefore, dehydration therapy is prescribed. Furthermore, when meningitis - those typically shown analgesics and detoxification treatment. At cha - severe forms of meningitis are used corticosteroids.

By individual indications prescribed anticonvulsants pre - Paratov, protease inhibitors, noootropnye, vitamins of group B are used extracorporal methods of detoxification.

Of great importance are nutrition (if necessary through a tube or parenterally), skin care and mucous membranes, monitoring of pelvic organs (indication Provo - DYT bladder catheterization, cleansing enemas).

F o r e c a s t . Depends on the etiology of meningitis, severity bolez - no, the degree of involvement of the pathological process of brain matter, comprising - Niya liquorodynamics, as well as the adequacy of the remedial measures and the usefulness of measures of care. With timely diagnosis and adequate treatment, in most cases it is possible to achieve almost - of recovery, but there may be very serious complications (ventilation rikulit, encephalitis, brain abscess), persistent residual symptoms (gidrotse - faliya, movement disorders, seizures, dementia) in children, except for those the same neurological manifestations, delayed physical and mental development. In late diagnosis and treatment inadequate in cases cha - severe forms of meningitis, especially in tuberculous and various wa - Rianta purulent meningitis death possible.

Purulent meningitis

A patient with suspected purulent meningitis claimed by the first - power, maximum attention and vigorous measures to clarify the diagnosis and treatment.

E t i o l o g y . Many pathogenic bacteria can be the causative agent of purulent meningitis . Most often these are meningococci, streptococci, hemophilic bacillus type B, pneumococci.

Newborns are more likely to become infected while passing through the birth canal. Clinical manifestations of meningitis occur in such cases, usually in the first week of life. In this case, the causative agent of purulent meningitis is more often conditionally pathogenic gram-negative enterobacteria , group B streptococci, listeria, and fungi. If the cause of meningitis in the newborn is a sepsis infection foci in the lungs or on the skin, the symptoms of meningitis usually occur no earlier than the second week, and the causative agent of meningitis are staphylococcus, streptococcus or conditionally pathogenic microflora.

After 2 months of life the child is deprived of maternal antibodies and becomes particularly sensitive to hemophilic stick and meningo - cocci, which are the most common cause of a cart - penetration of purulent meningitis in children. In this case, children under the age of 5 years is particularly common causes of meningitis is Haemophilus pas - span of. In children older than 5 years, the most common cause of purulent meningitis is the meningococcus, at least - the pneumococcus, staphylo - coccus, Escherichia, Salmonella, Pseudomonas aeruginosa. In adults, rev - Lemma purulent meningitis usually is pneumococcus, meningococcus rarely, even more rarely Streptococcus, Staphylococcus aureus,

Haemophilus influenzae. Thus pus - ny meningitis often develops in the presence of predisposing factors, such as immunosuppression, trauma and neurosurgical the intervention - Properties. In people with impaired health, in particular in patients alcoholysis - IOM and the elderly, the cause of purulent meningitis can be conditionally pathogenic bacteria: Escherichia coli and Haemophilus influenzae.

The incidence of purulent meningitis increases in the cold season, especially in the winter-spring period. Prevalence and ramp - of the frequency of purulent meningitis are associated with a common epidemiologists - cal atmosphere.

P a t h o g e n e s i s . The pathogenesis of bacterial purulent meningitis oc - novnoe value deployment inflammation attached exo- and endotoxins microbes and cytokine response. The production of cytokines by blood cells and vascular endothelium, as well as astroglia, leads to the deployment of a cascade of inflammatory reactions, the final result of which is the development of a purulent inflammatory process in the pia mater, which is accompanied by neutrophilic pleocytosis, an increase in the protein content in the CSF, and hyperthermia up to 39-40 " C, . signs of intracranial hypertension, severe meningeal syndrome this characteristic disturbance of consciousness, convulsions, it is possible - on the focal neurological symptoms.

An important pathogenetic factor purulent meningitis is time - vitie edema pial and adjacent brain tissues fuss - repentieth in response to the accumulation of toxic substances in CSF - produk - the breakup of microorganisms and leukocytes, lactate acidosis followed - conductive development of cerebral hypoxia. The resulting disorders of cerebrovascular microcirculation, autoaggregation of blood cells and brain metabolism can also have a pronounced effect on the patient's condition and lead to various neurological disorders.

Clinical manifestations. Purulent Meningitis can be a primary (meningokokkozy) or secondary arising at already existing outbreak of bacterial infection (suppurative otitis media, mastoiditis, B - nusit, pneumonia, bacterial endocarditis, etc.) Or in connection with ported - hydrochloric open penetrating traumatic brain injury. Predispose - conductive factor is immunosuppression, particularly in patients receiving you - sokih doses of corticosteroids, as well as diabetes mellitus, chronic alcoholism, HIV infection. ...

In infants, psychomotor restlessness, tremors, regurgitation, hyperesthesia, tension of fontanelles, head tilting are common . Already in the first 48 hours, general or local tonic-clonic seizures often appear. In older children and adults for - bolevanie begins with an intense headache, repeated vomiting agitation. A delirious state, depression of consciousness are possible, pronounced signs of meningeal syndrome appear early . On the 2nd-4th day, especially with late diagnosis and lack of adequate treatment, there is an increase in cerebral and development of focal symptoms.

With purulent meningitis, leukocytosis is detected in the blood with a shift of the leukocyte formula to the left, a high COP. When septicopyemia WHO - can identify the pathogen in blood smears infection stained by Gram.

D i a g n o s t i c s . In the diagnosis of purulent meningitis important zna - chenie have changes in CSF obtained by lumbar puncture [1]. With purulent meningitis, pleocytosis is characteristic (from several hundred to 10 thousand or

more cells in 1 μl) with a predominance of neutrophils. At the same time, it should be borne in mind that in the CSF obtained in the first hours of the disease, these features may still not be so clearly pronounced, and then pleocytosis will be relatively small, and the predominance of neutrophils may be unconvincing. However, after a few hours, pleocytosis in the CSF acquires characteristic features in most cases.

The protein content in the CSF is increased to 2-10 g / L, the glucose content in the CSF is usually reduced to a greater extent than in the blood. It is not enough ha - acteristic part of the CSF in purulent meningitis may be in cases where the patient before the lumbar puncture has already received antibiotics, as well as with meningitis development against the backdrop of severe leukopenia, and in cases when pathogens are bacteria that cause CSF advan - nificant lymphocyte reaction. Sometimes, CSF pleocytosis can be relatively minor. So is - is flowing when rapidly during septic and attenuated in the pre morbid period of patients with symptoms of immunosuppression that in some cases difficult to diagnose.

Identification of bacteria by microscopic examination of sediment CSF obtained after centrifugation produced immediately after its preparation, sometimes facilitates detection and identification WHO - arouser meningitis. Staining of the material under study is usually Provo - ditsya Gram. At the same time, to intensify the cryptococcal capsule, it is contrasted with ink.

CSF should be cytologically examined as early as possible after CSF is removed. If the CSF is at room temperature, then after 4 hours 10% of the cells contained in it are destroyed, after 18 hours - 70%, and after 46 hours - all cells.

Important diagnostic value may have bacteriological ICs - following the CSF. For this, the taking of the appropriate material should be carried out before the start of antibiotic therapy. However, it is believed that the sow from CSF in purulent meningitis bacteria is often possible for the first 4 hours of antibiotic treatment and bacterial antigens can be detected in it for at - how many days of starting treatment.

To clarify the causes of purulent meningitis needed rentgenolo - based surveys, lung, otorinolaringelogicheskoe examination of the patient, craniography, a CT scan of the brain, in some cases, MRI of the head. These studies reveal ve - royatnye causes suppurative meningitis: pneumonia, sinusitis, middle otitis media, cholesteatoma, brain abscess, as well as complications such oc - novnogo pathological process, such as swelling of the brain, offset and impaction brain tissue, brain infarction or hemorrhage foci in the cavity skull, hydrocephalus. To identify CSF fistula, the most informative research method is CSF scintigraphy . When searching for the primary site of infection may help big eye - to show a thorough physical examination of the patient.

Meningococcal infection

E t i o l o g y . The causative agent of meningococcal infection is meningococcal (gram negative diplococcus Veykselbauma - Neisseria meningitidis), the peak of the disease usually occurs in winter-spring ne - IRS. When this infection may be localized or generalized - hydrochloric. Localized forms of meningococcal infection are meningococcal disease, acute nasopharyngitis, isolated meningococcal pneumonia, meningococcal endocarditis, meningococcal arthritis (synovitis) or

polyarthritis, meningococcal iridocyclitis. By gene - ralizovannym forms include meningococcal septicemia - Neisseria meningitidis kemiya (acute or chronic), meningococcal meningitis, meningococcal meningoencephalitis, a combination of meningococcemia and meningitis [Pokrovsky VI et al., 1992].

E p i d e m i o l o g y . Meningococcus has the shape of a coffee bean, with a size of 0.6 to 1 micron. In Gram-stained blood smears and CSF sediment, meningococci are arranged in pairs, each of which is framed by a capsule. The cell wall contains endotoxin meningococcus is - schiysya lipopolysaccharide. According to the antigenic structure, 13 serotypes of meningococcus are distinguished , of which serotypes A, B and *C* are of the greatest importance . Meningococci die under the influence of direct sunlight, as well as at temperatures below 22 ° C and above 50 ° C; they cannot withstand long-term storage in a refrigerator at a temperature of ı rheO-4 ° C.

Meningococcal infection is sporadically seen everywhere, on this background there are outbreaks and epidemics that are not - which countries are repeated periodically usually 10-15 years. In the overpopulated states of Africa, from time to time, more often from November to May, epidemic outbreaks of cerebral meningitis occur almost every year . At the same time, the mortality rate in them reaches 80%, and among the deceased, 50% are children under the age of 15. It is noted that the largest epidemics of meningococcal disease caused by meningococcus gray group A, less frequent outbreaks caused by meningococcus serogroup B and C.

The only source of infection is meningococcal brow - century - the patient or meningokokkonositel. The majority of those infected have no clinical manifestations of the disease, acute meningococcal nasopharyngitis occurs in approximately 10% of cases of infection, and only in rare cases do infected people develop a generalized form of meningococcal infection, mainly meningococcemia, meningitis, or a combination thereof.

Meningococcus is transmitted by airborne droplets. Infection spo - Property crowd of people, especially close, prolonged contact with meningokokkonositelem.

Meningococcal meningitis (cerebrospinal epidemic meningitis) develops at any age, more often in children from 6 months to 10 years. Jn - fektsiya transmitted by droplets from patients or batsillono - CITEL. Entrance gate - nasopharynx, henceforth propagation occurs by hematogenous sometimes pathogen directly pro - arises from the nasopharynx in the meninges through perforatum plas - Tink ethmoid bone.

Acute onset, rapid: sudden headache, vomiting, giperter - mia to 39-40 "C and above, the general hyperesthesia, after a few hours you'll wish to set up and quickly increases in the degree of meningeal syn core, climaxing at 2-3 day In young children, manifestations of meningococcal meningitis are usually accompanied by bulging fontanelles.In older people, the onset of the disease may not be so acute, the temperature in some cases is subfebrile, meningeal syndrome appears only on the 3-4th day of illness.

Tendon reflexes in meningococcal meningitis are first increased, then fade away. Pyramidal pathologically] symptoms are often caused (Babinsky's symptom,

etc.). Of the cranial nerves, II, III, IV, VI and VIII are more often affected , and purulent labyrinthitis may develop. The cause of focal cerebral symptoms can be cerebral edema, dislocation, sometimes wedging of cerebral structures, manifestations of encephalitis or hemorrhage into the brain tissue. Pronounced irregular changes in body temperature are common , with toxic shock hypothermia is possible, as well as unstable heart rhythm, while tachycardia is more common, but pulse reduction is possible . Heart sounds are muffled, a sign of myocardial dystrophy is noted on the EEG . The clinical picture of meningococcal meningitis can be complicated by layered herpes infection, pneumonia, manifestations of respiratory, heart, adrenal insufficiency.

The results of a lumbar puncture with meningococcal meningitis: high pressure, CSF is cloudy, greenish, during the culmination of the disease contains 10-30 thousand leukocytes in 1 μl, mainly (more than 90%) neutrophils, the content of glucose and chlorides is reduced, the amount of protein is increased to 1 - 16 g / l, and the reaction Pandey Nonne- Apelt sharply positive - tion (++++). In bacteriological diagnostics, mucus is sown from the posterior wall of the pharynx, CSF, and in meningococcemia, blood is sown. Isa - the benefits and methods of rapid diagnosis.

Needed to clarify lumbar puncture diagnosis, however Nali - PIR manifestations intracranial hypertension, in particular congestive dis - Cove optic nerves, and especially in cases of suspected offset and vkli - nenie brain tissue is primarily shown to hold a CT scan of the brain to identify possible contraindications to the lumbar puncture. If the CT scan revealed marked swelling headache - of the brain, and the more the risk of herniation of brain structures, conducted by - Niya lumbar puncture should be temporarily avoided. In such cases, to avoid the loss of time, treatment with antibiotics should be on - Chato to obtain information on the composition of the CSF.

As the rare variant describe cases of meningococcal meningitis, at which CSF osta - etsya transparent or slightly opalescent. In this case, it is revealed moderate pleocytosis with a predominance of lymphocytes, but usually with an increased content of neutrophils, as well as with an increased content of protein.

In meningococcal meningitis in the background lightning forms IU - ningokokkemii already in the debut of the disease observed pronounced Institute - toxication, increasing signs of infectious and toxic shock syndrome with manifestations of the syndrome Uoterhauza- Friderichsen possible partial and generalized seizures, sometimes hyperkinesis, psychomotor WHO - excitation of delirium, changing the state of stupefaction , stupor, coma.

Hypertoxic meningococcal infection, which usually occurs against the background of a combination of a hyperacute, fulminant form of meningococcemia, which can be combined with meningococcal meningitis, manifests itself as a clinical picture of infectious toxic shock. Its pathogenesis is complex and contains the combination of signs gapovolemicheskogo, cardio gene (arising from a decrease in cardiac output, decreased cardiac output) and the distributor (Nara consequence - sheniya vascular tone) shock.

There are 4 degrees of *infectious toxic shock* (ITSh).

And T Sh-1 (phase of w a r m normotonia). The patient's condition thee - said heavy, pink face, but the rest of pale skin; skin su - hai, warm, but it is possible and

profuse sweating, vomiting, central hyperthermia (38,5-40,5 ° C), tachypnea, tachycardia, blood pressure in pre - affairs norms or increased (giperkateholemii consequence in connection with - Chin hypovolemia) ... Observed psychomotor agitation, general hyperreflexia, in infants, possible convulsions, metabolic acidosis with a partially compensated alkalosis, hyper - coagulation.

And T Sh-2 (w a r m h y p o t e n s i o n phase) . The patient's condition is very serious, he is lethargic, inhibited, the face and skin are pale, with a grayish tinge, acrocyanosis, the skin is cold, moist, the temperature is normal or subnormal. Expressed tachycardia, tachypnea, weak pulse - of filling, heart sounds are muffled. Blood pressure and cardiac output, central venous pressure is reduced, peripheral vasodilation, reduced ne - rifericheskogo resistance relative hypovolemia, metaboliches - cue acidosis, DIC 2nd degree.

And T SH-3 (phase of c o l d hypotension). The patient's condition is extremely serious, consciousness is usually absent, mask-like face, peri - fericheskaya vasoconstriction, total cyanosis, multiple Hemorrhoids - cally-necrotic elements, venous stasis by type cadaver spots limbs damp, cold. The pulse is threadlike or not detected, tachycardia, shortness of breath, blood pressure is very low, does not change with an increase in the volume of circulating blood. The reaction of pupils to light is reduced, it is possible - us squint, hypertension, muscle, tendinous hyperreflexia, pathologists - cal stopnye reflexes may occur periodic spasms, the usual - us meningeal symptoms, anuria. Uncompensated metabolite - cal acidosis, DIC 3-4-th degree, diffuse bleeding, multiple organ failure.

And T SH-4 (terminal phase). The patient is unconscious, marked - are paralytic mydriasis, muscle atonia, areflexia, tonic convulsions, collapse, irregular breathing, apnea, respiratory arrest. Cardiac activity after this can persist for another 10-15 minutes.

Meningococcal meningoencephalitis characterized by severe flow - it, in this case since the early days of the disease along with obscheinfektsionnymi, general - cerebral and meningeal symptoms and signs Ocha - traction brain lesions:. Local convulsions, paresis, paralysis, etc. With the development ventriculitis marked somnolence, expressed higher - of muscle tone , sometimes opisthotonus, convulsions, stunning, stupor, coma. In the CSF, along with pleocytosis, there is a lot of protein; xanthochromia is possible.

Meningococcal endocarditis has a long course, but usually proceeds without the formation of valvular heart disease. Dull heart sounds, tachycardia, sometimes cyanosis and shortness of breath are noted. May be accompanied by periodic rises in temperature, skin rashes, manifestations of meningococcal arthritis.

Meningococcal arthritis (synovitis), or arthritis more often Serozem - nym, usually affects metacarpophalangeal and interphalangeal, rarely large joints. Affected joints are swollen, enlarged, upon them a cart - mozhna flushing of the skin, floating, palpation and movement marked tenderness. Adequate treatment leads to restoration of the function of the affected joints in 3-6 days.

Meningococcal iridocyclitis is one of the most severe manifestations of meningococcal infection. The iris acquires a rusty color and bulges forward in a ball shape, while the pupil is narrowed, the anterior chamber of the eye is shallow, and intraocular pressure is low. Characteristically fast, sometimes during the day, an

increasing decrease in vision up to blindness.In the future, a gradual development of atrophy of the eyeball is possible

D i a g n o s i s o f m e n i n g o c o c c a l i n f e c t i o n . An outfit with the data of the anamnesis and clinical examination of the patient is given important information by the results of blood and CSF tests. However, the etiological diagnosis can be absolutely reliable only after the identification of the pathogen. For example, in cases of suspected meningococcemia in the early days of the causative agent of the disease can be detected by microscopy painted - Noah Gram thick drops of blood. If meningococcal nasopharyngitis is suspected, mucus from the back of the pharynx is examined for the same purpose. In generalized meningococcal infection importance can have rapid serologic diagnosis by conducting a reaction passive hemagglutination or counter-immunoelectrophoresis and Drew - GIH immunological methods. Sowing of appropriate tissues and fluids (blood, CSF) and subsequent determination of the response of the grown bacterial colonies to various antibiotics can provide valuable data for clarifying the diagnosis and treatment .

T r e a t m e n t . In generalized forms of meningococcal infectious - tion shown urgent hospitalization of the patient in the profiled otde - Leniye. With pronounced manifestations of cerebral edema, infectious-toxic shock, hospitalization in the intensive care unit is indicated.

With meningococcal meningitis and meningococcemia carried ethio anisotropic component & therapy penicillin rate of 300 000 IU / kg per day within - musculo intervals between injections administered either 4 hours polusinte - cal penicillins at the same doses. Brain edema and infection-toxic shock considered the drug of choice chloramphenicol females tsinat rate of 80-100 mg / kg body weight per day (less than 2 g / d) vnut - Riven at intervals of 6 h for 24-48 hours (toxicity !), followed by - conductive transition to penicillin. Antibiotic treatment course without reduced - Niya dose is 7-10 days. In severe forms of meningococcal infection, crystalline sodium salt of penicillin can be administered endolumbally once. Criterion cancellation antibiotics in IU - Ning - cytosis reduction in CSF and 100 cells in 1 mm, the mono nuclears should be at least 70%, normal ana indicators - blood Lisa, satisfactory overall.

In addition to meningococcal infection antibiotics are used also sulnilafamidnye sustained release formulations: sulfa flax or combined preparations - Bactrim, a maximal sulfate - GOVERNMENTAL doses. The duration of antibacterial treatment is usually 5 to 10 days. For the purpose of detoxification performed polyionic infusion solutions , glucose solutions, protein drugs available expediency - NOSTA plasmapheresis hemosorption. When severe complications during the slaughtering - Levan on the testimony injected corticosteroids, heparin, antiplatelet agents.

Pathogenetic therapy includes detoxification, dehydration, normalization of the homeostasis system and metabolic processes.

Possible complications of meningococcal meningitis: hydrocephalus, intracranial hypertension, epileptic seizures, status epilepticus , autonomic lability, decreased attention, memory, mental retardation in children, and dementia in adults.

Secondary purulent meningitis

E t i o l o g y . Secondary purulent meningitis occurs when bacteria enter the meninges from a purulent focus in the body (sinusitis, otitis media, bronchiectasis, etc.) or as a result of the involvement of the meninges in a purulent inflammatory process that develops in neighboring tissues (osteomyelitis, otitis media, sinusitis, etc.) .), as well as in the case of infitsirova - Niya meninges with an open penetrating craniocerebral injury. The causative agent can be a Staphylococcus, Streptococcus, Streptococcus pneumoniae, Haemophilus influenzae Afanasyeva-Pfeiffer, at least Salmonella, blue- pus bacillus, etc. In infants most likely to excite. - telyami purulent meningitis are group B streptococcus, Listeria, Gram-negative enterobacteria, in children older than 2 months of festering Menin - git is more often caused by Afanasyev-Pfeiffer hemophilic bacillus.

C l i n i c a l m a n i f e s t a t i o n s . The clinical picture and course are very similar to the manifestations of meningococcal meningitis. It is usually difficult expressed cerebral and meningeal Symp - toms, CSF turbid pleocytosis, usually within a few hundred or thousands of cells in 1 mm, preferably neutrophilic. To clarify the diagnosis, a bacteriological examination of the CSF is carried out and the sensitivity of the seeded infection to antibiotics is revealed .

Below is information about some forms of secondary purulent meningitis.

Serous bacterial meningitis

Tuberculous meningitis

In recent years, there has been an increase in the incidence of tuberculosis (in Russia up to 70 per 100,000 inhabitants), annually 3 million people die from tuberculosis in the world . Tuberculous meningitis accounts for 3% of all diseases of tuberculous etiology, while its percentage is especially high among children with tuberculosis. Only in 30-40% of patients it occurs with an active tuberculous process, more often in the lungs, in other cases - against the background of a latent primary process. It is believed that in children and young people, tuberculous meningitis may be a manifestation of a primary infection. Thus tuberculin test in 50% of cases may be negative, as in primary infection with Mycobacterium tuberculosis propagated - Xia intracellularly within 2-8 weeks after exposure and before the lane - O manifestations of the disease is approximately 10 weeks.

E t i o l o g y a n d p a t h o m o r p h o l o g i c a l p i c t u r e . Tuberculous meningitis - a manifestation of hematogenous dissemination of the tuberculosis bacillus - can be recognized as one of the most severe and prognostically unfavorable forms of infectious lesions of the nervous system, and among the serous forms of meningitis, it is probably the most insidious disease. Typically occurs in the presence of tuberculosis in the body chamber (pulmonary tuberculosis, lymph glands, bone tuberculosis), although the cases of tuberculous meningitis occur in which g 'of primary focus remains undiagnosed.

The causative agent of tuberculosis infections, in particular tuberculosis IU - ningita is tubercle bacillus (Mycobacterium tuberculesum, wand Koch). TB infection usually occurs in children WHO - to grow, often through the respiratory

tract. People can get tuberculous meningitis at any age, but children and the elderly with insufficient immune system functions are more likely to get sick .

It affects predominantly basal membrane (basal meningitis), ependyma ventricles, particularly IV and III, and their suck - kyanite plexus, cranial nerves may involvement in the process vessels and brain parenchyma. Specific inflammation foci consist of miliary tubercles formed cluster epithelioid, plazmati - gal and lymphoid cells.

C l i n i c a l manifestations. The clinical picture is stolen - Chiva subacute prodromal period after extending an average of 2-3 weeks, sometimes up to 8 weeks. During this period, there are a total of ALS - Bost, lethargy, fatigue, anorexia, low-grade topics - perature. From the first day of illness, there is a growing headache, repeated vomiting, hyperthermia up to 38-39 ° C; meningeal syndrome - melt away within a few days, and usually soon reaches a pronounced degree, especially in children, sometimes taking a pose "cocked" or "setter", while often the symptom "navicular" belly, opisthotonos. Involvement of cranial nerves in the process is characteristic . Tendon reflexes decrease and then disappear, radicular pain is possible . Usually deafness, cognitive disorders, **pronounced** autonomic disturbances **(pronounced sweating** of the Trousseau spot, increased blood pressure, brady- or tachycardia, disorders of the functions of the pelvic organs) develop. Of the cranial nerves, the optic, oculomotor, abducers, facial, vestibular cochlear nerves are more likely to suffer, manifestations of bulbar syndrome, epileptic seizures, and sometimes *1* cerebral rigidity are possible. If the patient does not receive specific treatment, then after 2-3 weeks he dies.

For the diagnosis of tuberculous meningitis, along with the data of the anamnesis of the neurological and somatic examination of the patient, the results of a lumbar puncture are very important. CSF is secreted under high pressure (300–500 mm of water column), transparent or opalescent . In the CSF, mixed pleocytosis is noted (several hundred cells 1 μl), usually neutrophilic in the first days, and soon with a predominance of lymphocytes, the amount of protein is increased to 5-10 g / l, the glucose content is below 40% of its content in the blood with a slight decrease in the level nya chlorides and vitamin C; the content of the residual is slightly increased; nitrogen. After 12-24 hours, a delicate cobweb-like fibrin film usually appears in a test tube with CSF (before examination, the test tube should not be shaken; it is examined in transmitted light on a black background), from which the causative agent of tuberculosis (Koch's bacillus) is sown in 20-40% of cases. It can be found in a smear from this film or from the sediment of centrifuged CSF, stained according to Ziehl-Nielsen. When examining the patient, a chest X-ray is required, tuberculin tests, although their information content is relative.

Poor prognostic signs are a deep depression of consciousness already at the beginning of treatment, miliary tuberculosis, the presence of hydrocephalus and stroke, a pronounced decrease in glucose in the CSF; very high protein levels.

Diagnostics. Differential diagnosis is sometimes provided; very difficult. It is carried out with other forms of serous meningitis, mainly viral and fungal.

The most reliable sign of tuberculous etiology of the disease is the detection of tuberculous mycobacteria, which is achieved by microscopy of stained blood smears and culture of sputum and CSF. Though exciter sown in about 75% of the cases of tuberculous meningitis - one requires from 2 to 6 weeks for its cultivation. The CSF identify mikobak - terii TB hard smear is better to cook from it formed a fibrin film.

Certain difficulties clarify tuberculous origin IU - ningita often dictate the need to start the appropriate leche - of in relation to the severity of the patient ex juvantibus until re - results of microbiological research.

It is believed that in difficult to diagnos tics any cases of subacute meningitis with fever and low containing - should be regarded as tuberculous until Niemi glucose in the CSF proved otherwise. This position is justified by the fact that tuberculous meningitis is a form of serous meningitis, in which the saving of the patient's life is largely determined by the timing of the start of his treatment, but even with adequate treatment of tuberculous meningitis, its course is protracted, while the likelihood of persistent residual effects is high.

Viral meningitis

Acute lymphocytic choriomeningitis

E t i o l o g y and E p i d e m i o l o g y . Acute lymphocytic ho - riomeningit (meningitis Armstrong) - zoonotic acute infectious disease whose causative agent belongs to the group of adenoviruses, races - prostranennyh everywhere. The causative agent was isolated in 1934 from the CSF of patients. Discovery occurred accidentally in the pro - cession study of St. Louis encephalitis. The main natural rezervua - rum virus are domestic and laboratory mice, at least - Other Plays - Zun, in particular, guinea pigs and hamsters, in the body where the virus persists for a long time.

Acute lymphocytic choriomeningitis transmitted to humans by INFI - -skilled rodents through the respiratory tract or upon contact with ekskre - cops infected animals. It is assumed the presence of not - as possible pathways: airborne, alimentary, transmissible (arthropod bites). Reliable data on the re - giving of infection from person to person there. In the human body the virus races - uted hematogenically, penetrates the blood-brain barrier

(BBB).

C l i n i c a l m a n i f e s t a t i o n s . The disease manifests itself in the form of primary serous meningitis, edema and lymphocytic infiltration of the pia mater and choroidal plexuses occur . The duration of the incubation period is from 6 to 13 days. Along the course , acute, chronic and asymptomatic forms of lymphocytic choriomeningitis are sometimes distinguished [Vasiliev V.S. et al., 1994].

In typical cases, the disease starts with an acute hyperthermia 38- 40 ° C , continuing 1-2 weeks, at least - 3 weeks, fever, general weakness, not - domogany, headache, especially orbitofrontal localization - tion, myalgia, especially in the lumbar area, photophobia, anorexia, nausea, vomiting. Usually the pain in the throat, pharynx congestion, perversion Chuv - and void, pain in the chest, in the region of the parotid glands, taken away - chenie cervical, axillary lymph nodes. Sometimes, after

1-3 weeks. From the onset of the disease, there are signs of orchitis, hair loss, in rare cases - up to baldness. Meningeal symptoms are characteristic ; in the early days, psychomotor agitation is possible. There may be convulsions , unstable paresis of muscles innervated by cranial seals III and VI (strabismus, diplopia), tendon hyperreflexia, pyramidal signs, congestion in the fundus.

With a lumbar puncture: CSF pressure is high, it is colorless transparent, in 1 μl usually 100-300, less often up to 1,000-1500 cells, of which. 90 - 95 % - lymphocytes, the protein content is normal or moderately increased (up to 1-2 g / l), the level of glucose and chlorides is within the normal range.

Acute lymphocytic meningitis gradually regresses. The elevated temperature persists for 1-2 weeks, somewhat longer - with signs of irritation of the meninges. CSF sanitation occurs within 2-4 weeks. The state of health of patients usually improves after 7-10 days from the onset of the disease, and clinical recovery gradually begins. However, sometimes, due to the formation of an adhesive process in the meninges, impaired CSF dynamics and increased intracranial pressure, the headache persists for a long time. In rare cases, already in a standing position close to clinical recovery, a relapse of the manifestations of meningitis occurs: fever, cephalalgia, meningeal symptoms and other signs of the disease. In such cases, recovery can take up to 5-6 weeks. Meningeal signs persist for 2 weeks or more.

The virus from the blood of patients can be isolated in the first days of the disease, complement- binding antigens in the blood persist for 6-8 weeks, and the virus neutralizing specific antibodies detected by means of neutralization reactions, immunofluorescence and complement binding for several years. The prognosis is often favorable. In some cases, the development of persistent intracranial hypertension is possible .

Treatment. There is no specific treatment. In order to reduce intracranial pressure, saluretics are prescribed, sometimes unloading lumbar punctures. Symptomatic and restorative therapy is carried out, vitamin complexes are prescribed, general care is required.

Prevention. Specific prophylaxis has not been developed. To prevent infection, it is recommended to deratize living quarters, food warehouses, keep food in places accessible to rodents, and observe the rules of personal hygiene.

Enteroviral meningitis

Currently, more than 70 enteroviruses are known, among which they can cause various diseases in humans. In neurology, enteroviruses of poliomyelitis, Coxsackie and ECHO are of particular importance. The last two groups of viruses are ubiquitous. At the same time, many practically healthy people are the carriers of the virus . Among the diseases of the nervous system caused by the Coxsackie and ECHO viruses, serous meningitis is the most common.

Primary serous meningitis caused by enteroviruses Cox (the name of the American city in which the virus was isolated in 1948 I *ECHO* (an abbreviation formed from the initial letters "entero cytjpatj human orphan" - intestinal cytopathogenic human orphan viruses.

Encephalitis

Encephalitis is an inflammatory brain lesion. caused by infectious or infectious-allergic Process - catfish. Concomitant inflammatory brain damage and his Obolo - checks is called meningoencephalitis, brain and spinal cord - Enz - falomielitom, and if the inflammatory process such as encephalomyelitis involved and the meninges, then we are talking about meningoentsefalomieli ones. The causative agents of encephalitis can be bacteria, viruses, rickettsia, prions, protozoa.

Classification. There are different classifications Enz - falitov. As with meningitis, encephalitis can be differentiated on the *lane - between primary and secondary,* as well as the etiological basis.

Most primary encephalitis caused by viruses, in particular - NOSTA arboviruses (RNA-containing viruses transmissible dwell - conductive body bloodsucking arthropods and transmitted teplokrov - nym animals and man bites). Encephalitis caused by neurotropic viruses can be contagious, endemic , seasonal, and climatic and geographic. Encephalitis can be *diffuse and focal.* Focal encephalitis in for - depending on the preferential localization of lesions of the brain is divided into the stem, cerebellar, diencephalic, hemispheric. By preimuschest - venno damages the brain structures is isolated from dominating encephalitis - yuschim white matter lesions - *leykoentsefality* (group subacute progressive leykoentsefalitov) and with a predominance of encephalitis - expressions gray matter - *polioentsefality* (acute polioentsefalomie- lit., epidemic encephalitis lethargic); encephalitis with diffuse lesions of gray and white matter of the brain - *panencephalitis* (tick-borne encephalitis, a mosquito, Australian, American encephalitis). By the nature of exudate encephalitis can be divided into *pus - nye and purulent.*

P a t h o g e n e s i s . The pathogenesis of encephalitis is diverse. The reason for - expressions brain tissue may be in its penetration agents, and the development of toxic and allergic processes. In the CLM - n reactions occur in the form of perivascular infiltrates, edema, brain tissue disorders, hemodialysis and liquorodynamics, dot blood - effusions, reversible and irreversible degenerative changes of nerve cells, regressive or progressive glial reaction, demieliniz pathways tion. Possible involvement in the process of brain about - lochek, formation of pus or serous infiltrates, foci not - Crozat.

C l i n i c a l m a n i f e s t a t i o n s . The clinical picture is characterized - ized obscheinfektsionnymi signs, cerebral and focal neurological symptoms, the nature and severity of which can vary within wide limits depending on the characteristics of the pathogen, the prevalence of preferential localization and characteristics of pathological changes in the brain, as well as on the state of them - immune systems are ill and somatic status in the premorbid period.

Primary encephalitis

Tick-borne spring-summer encephalitis

H i s t o r i c a l b a c k g r o u n d . Undoubtedly, the tick-borne encephalitis, characterized by severe natural foci was Prevalence - nen in certain areas since ancient times. However, the first description of the clinical picture of tick-borne spring-summer Enz - falita was done in 1935, domestic neurologist

AG Panov, who gave this name to the disease. A detailed study of the spring-summer encephalitis carried out complex expeditions in taezh - nye places of Siberia and the Far East, and is reflected in the works EN Pavlovsky, L.A. Zilber, M.P. Chumakova, A.A. Smorodintseva, V.D.-Solovyova, A.N. Shapoval and others.

E t i o l o g y ; Tick-borne, or taiga, spring-summer encephalitis is a primary acute viral disease. The causative agent is a neurotropic filterable arbovirus from the Togaviridae family, a genus of flavoviruses; it can be represented by subspecies of western and eastern tick-borne encephalitis viruses. On the basis of the antigen affinity to the core - GIM arboviruses and common pathways it enters the group of viruses of tick-borne encephalitis, which also includes the viruses Omsk Hemorrhoids - cal fever, Scottish encephalomyelitis, Lyme disease.

Epidemiology. Infection with tick-borne encephalitis pro - comes in endemic natural foci of infection transmissible (after the tick bite) or alimentary route (through infected Molo - co). A reservoir for human pathogenic neurotropic filter - camping arbovirus tick-borne encephalitis in nature are rodents, nekoto - rye poultry, goats. An additional reservoir of the virus and its carrier are mainly ticks (Ixodes persulcfnus, less recinus Ixodes), dwelling in the forest softwood, as well as in broadleaf and mixed - GOVERNMENTAL forests, sometimes in the steppe areas.

Seasonal morbidity in humans is closely related to the periods of greatest biological activity of vectors of infection. In spring and summer ne - IRS (May-July) clamp-carriers of the virus reach sexual maturity and are being infected, can infect human saliva containing the virus when they bite and blood sucking. After the initial replication on - domain entrance gate viruses penetrate the blood of man, in the future they will continue to multiply, by hematogenous spread throughout the body. At the same time they get to the lymph nodes, the internal op - Ghana and in the central nervous system, where developing inflammatory and degenerative changes - Niya. Much less frequently there alimentary infection with the human virus of tick-borne encephalitis virus by eating infected milk virus goats and, less frequently, cows. In such cases, the virus enters the bloodstream, proho - AJ through the mucous membrane of the digestive tract.

The disease is particularly prevalent in areas of the tick habitat: in taezh - GOVERNMENTAL regions of Primorye Territory, Siberia, the Urals. Much less mite - howling encephalitis is found in the northern regions of the European part of Russia. Gradually halo spread of the disease is expanding, and now time, it is also found in some areas of central Russia (Jaro - Slavska, Smolensk, Tver). People who come to the taiga from other regions are especially susceptible to tick-borne spring-summer encephalitis , often their disease is especially difficult. This can be explained by the lack of immunity among visitors, which is developed among the indigenous people. Incidence tick encephalo - that in endemic areas varies from 5-10 per 100 thousand yarn. - teley. The severity of clinical symptoms of the disease is dependent on the characteristics of the endemic focus: more severe in foci where circulating strains of the virus and eastern smaller - in foci where propagation - the countries of Western type TBE virus.

P a t h o g e n e s i s . Depending on the route of infection, the multiplication of the tick-borne encephalitis virus begins in the skin or in the intestinal mucosa. Generalization process occurs lymphogenous or heme - togennym means. Viremia is of two-wave nature, - wherein the second wave is generated at the end of the incubation period and coincides with the multiply - Niemi viruses in internal organs and their penetration into the CNS. The neurotropism of the virus manifests itself in the predominant lesion of the anterior horns of the spinal cord, more often the cervical thickening, and the motor nuclei of the brainstem. After an acute period of the disease, the virus can persist in the central nervous system for many years , and under unfavorable conditions for the body and with a decrease in immunity, it can be activated, which leads to the development of chronic forms of tick-borne encephalitis, the course of which can be progressive, leading to death or permanent disability .

P a t h o l o g i c a l picture. Tick-borne spring-years - Nij encephalitis can be characterized as acute purulent menigoentse falomielit. Pathological changes are defined as in gray and white matter of the brain, but predominantly in the gray matter of the brain stem and spinal cord, as well as in the cerebral cortex. Histological examination reveals massive death of nerve cells, the phenomenon of neuronophagy with the formation of nodules from astrocytes. The inflammatory reaction in the early period of the disease is represented by tissue infiltration by neutrophils and lymphocytes, in later periods lymphocytes and macrophages prevail. With the development of chronic - iCal forms of tick-borne encephalitis has a combination of degenerative processes in neurons, swelling and proliferation of glial cells, perivascular infiltrates, indicating the different stages of the inflammatory pro - cession in the CNS.

Clinical manifestations. The incubation period after the virus enters the body after a tick bite lasts from 2 to 30 days, more often from 7 to 14 days. With alimentary infection, it is shorter: 2-7 days.

The clinical picture is diverse. Distinguish inapparent, Sr. - fifth, feverish, meningeal (meningoencephalitic) and hearth - -hand (paralytic) forms of the disease. Depending on the prevailing symptoms, non-focal and focal forms of tick-borne encephalitis can be distinguished . Non-focal forms include febrile and meningeal forms; to focal - poliomyelitic, polioencephalomyelitic and encephalitic.

The inapparent (asymptomatic, subclinical) form of the disease appears to be the most common *form of the* disease in natural foci in the local population.

The erased form is characterized by a short (1-3 days) and not always pronounced febrile state, short-term manifested by meningeal syndrome, while changes in the CSF are always detected . It occurs 10-14 times more often than clinically expressed forms of the disease .

With *febrile* and *meningeal forms, the* disease begins acutely, the onset of the disease is especially severe in children. A sudden rise in temperature to 38-40 ° C is characteristic ; hyperthermia persists for 3-7 days. As a rule, signs of general intoxication are expressed: diffuse headache, dizziness, chills, repeated vomiting. In about half of the patients, the temperature curve has two waves, between which it turns out to be significantly reduced within 2-5 days (two-

wave temperature reaction). The second febrile wave of the disease occurs in those persons who have a delay in the production of antibodies. In especially severe cases, the disease can debut with status epilepticus.

In the acute stage of febrile and meningeal forms of spring- summer encephalitis, attention is drawn to hyperemia of the face and conjunctiva. The pulse often does not correspond to body temperature and is relatively slow. The upper body is usually hyperemic. Catarrhal phenomena, gastrointestinal disorders are possible . From the very first days of illness, patients note pain in the muscles of the neck, shoulder girdle, calves of the leg muscles, confusion, lethargy, stupor, stupor . In the meningeal form, radicular and meningeal symptoms can be expressed to one degree or another . The manifestations of the meningeal syndrome persist for 2-3 weeks. Possible, more often in children, focal, mainly cerebral, neurological symptoms: anisoreflexia, ataxia, muscle tone disorders, hemiparesis, cranial nerve lesions. There may be (usually in adults) signs of lesions of the anterior horns of the spinal cord in the form of flaccid, mainly proximal paresis.

In the blood of patients, leukocytosis, aneosinophilia, and lymphopeta increase in ESR are characteristic . CSF with a lumbar puncture flows out under increased pressure, colorless, transparent. In the febrile form of the disease, a small pleocytosis is common (up to 30 cells in 1 μl). The content of protein in CSF may also be increased . With the meningeal form of tick- borne encephalitis, manifestations of serous meningitis or meningeal encephalitis are characteristic , meningeal symptoms are expressed. In the CSF, at the first lumbar puncture, the number of cells is from 100 to 600 per 1 m, while approximately equal numbers of mono polynuclear cells are usually observed. Subsequent punctures reveal a gradual predominance of mononuclear cells of lymphocytes and monocytes in the CSF, typical of pink meningitis . At the same time, the protein level is increased, sometimes up to 6 g / l. The course of the meningeal form of tick-borne encephalitis is often severe. Rehabilitation of CSF occurs in 3-5 weeks.

Focal (polioencephalomyelitic , ralitic) forms of the disease are the most difficult . They develop sharply after a short prodromal period. At the same time, the temperature rises to 39-40 ° C above and stays at this level from 3 to 12 days. Characterized by severe headaches, pain in the limbs, in the lower back, relative bradycardia, shortness of breath, severe cerebral symptoms and autonomic reaction. Enlargement of the spleen and liver is possible.

Encephalitis in influenza and other acute respiratory infections

Etiology and pathomorphological picture. These forms are encephalitis viral respiratory complication yn - fektsy due, in particular, influenza viruses *(A* \ A_2, A_3, B) viruses and parainfluenza, adenoviruses, causing various forms of acute respiratory disease and SARS. The pathways for the infection to enter the brain have not been specified. You can pre - suppose hematogenous spread may, in some cases, the infection enters the cranial cavity through the ethmoid bone at obonyatel - nym threads. The penetration of pathogenic viruses into nerve cells has been established, although the presence of actually inflammatory changes in the exchange tissue is disputed by some authors. Characteristically plethora suck -

poisons the brain, trombovaskulity, perivaskudyarnye infiltrates, dot hemorrhages. Among the pathogenetic factors that disrupt the functions of the brain, an important place is occupied by discirculatory phenomena and severe intoxication.

Clinical manifestations. Signs of toxic effects on the nervous system structure and meninges arise in all cases of influenza and influenza-like state, but in some cases during the climax of respiratory disorders may expressed worsened - of the general condition, the appearance or increase in symptoms of irritation meninges expressed cerebral disorders (headache, vomiting, dizziness) and focal, usually little pronounced, neurologists - cal symptoms, lethargy, drowsiness. There are signs of irritation of sensitive spinal roots, sometimes marked manifestations - Niya trigeminal neuralgia. Usually, there are marked Vegeta - tive disorders. With a lumbar puncture, an increase in intracranial pressure is detected, lymphocytic pleocytosis in the CSF, and a slight increase in the level of protein is possible.

Sometimes the flu more often during the climax of brain sympto - mov, develops acute sharp deterioration with chills, raising the temperature to 40 ° C and above, confusion, rezchayshey head pain, recurrent vomiting, convulsions, polymorphic focal neuro - logical symptoms. Increases rapidly swelling of the brain, thus deepening - wish to set up impairment of consciousness up to coma. CSF usually contains erythrocytes, and the CSF color is pinkish or xanthochromic. In such cases, we are talking about the development of hemorrhagic influenza encephalitis or meningoencephalitis. This form of complication of influenza is very difficult and can be fatal; in the case of recovery often remain residual neurological disorders and mental Naru - sheniya, leading to disability.

T r e a t m e n t . Comprehensive treatment includes antiviral pre - Paratov and glucocorticoids. Great attention should be paid Dezin - toxication intravenously administered in divided doses throughout the day n modez to 500 ml, 500-1000 mL of 5% glucose solution, administered ascorbic - new acid. B vitamins, ascorutin , essential, unitiol, anginine (prodectin), decinone, calcium chloride or gluconate are used.

Epidemic encephalitis

(Economo disease, encephalitis A, lethargic encephalitis)

Historical b a c k g r o u n d . In 1917, at the Vienna Neuve society - ropatologov and psychiatrist Karl Economo ETS - lal message about an epidemic outbreak of a new infectious bolez - no, he watched from the autumn of 1916 until the spring of 1917 in the military state - Pitalev Austria. The reason for the selection of the disease in a particular nozologiches - kuyu form was the originality of its clinical picture. After to - Rothko prodromal period evolved increasing headache pain, nausea, vomiting, and against this background arose expressed deep drowsiness, often in combination with delusions and followed by immersion in a lethargic sleep. In severe cases, usually ends lethal - but deepened drowsiness to coma. When favorable - rated current of disease drowsiness gradually disappeared. We mention often - chalis paresis of the muscles

innervated by cranial nerves, especially frequent eye movement. At the beginning of the disease is usually mild arose - nye meningeal symptoms, in most cases, the increased - the body perature, although pyrexia was not an obligate feature of the disease . The temperature reaction, if it manifested itself, did not have any typical features. The disease was named Economo's lethargic encephalitis.

Epidemic outbreak of the disease, observed since 1915 , on the fronts of the First World War to 1924 was transformed into a panda - the mission, to cover not only the whole of Europe, but many countries in other conti - nents from Canada to the Philippines and New Zealand. When this became known - but that in some cases the disease becomes chronic and the clinical symptoms are similar to symptoms of Parkinson's disease, which served as a basis to consider due to their clinical picture like Parkinson's. It is assumed that until 1927, the total quantitative - to recover from encephalitis epidemic has exceeded 80,000 people [Stern R., 1936]. Since these epidemics were observed, and the diagnosis of the disease is put only in rare sporadic cases, the clinical picture of the disease is usually reduced and verify it in such cases is difficult, since the pathogen was still neutochnen - nym.

At the same time, as A.G. Panov (1962), the presence of sporadic SLE - teas encephalitis economy and the absence of reliable means of preventing the disease pose a potential threat to its propagation of the epidemic - roub. This argument is still valid until now, although the epi - -pandemic encephalitis is now rare.

E t i o l o g y and p a t h o g e n e s i s . It is assumed that the causative agent of epidemic encephalitis is a filterable virus and infect - vanie occurs through direct contact with sick or infected with HIV. However, the contagiousness in such cases is relatively low, which casts doubt on the airborne method of transmission of infection. The spread of the pathogen in the body, appears to be the blood - the gene. During the epidemic encephalitis are acute and of chronic - ical stage.

P a t h o - m o r p h o l o g i c a l p i c t u r e . Advantageously, - reflects certain brain regions - primarily structures otno - syaschiesya to limbic-reticular complex, basal ganglia, and the black substance and core barrel cranial nerves located in the stem of the brain on mesencephalic level. In the acute stage of brain preobl adayut signs of inflammation: infiltration of mononuclear marrow tissue, the accumulation of leukocytes, and plasma cells around vessels, sometimes the - Chechnev hemorrhage in brain tissue, its swelling phenomenon. Over time, the WHO - are possible degenerative changes of cellular structures in the tire medium of the brain. In the chronic stage of the disease, degenerative- dystrophic changes predominate . It celebrated the death of individual neurons, the odds - miruyutsya glial scars. In this case, the cranial substance suffers most significantly. In 1919, this fact was established by K.N. Tretyakov. Of the internal organs, the liver suffers more than others.

C l i n i c a l m a n i f e s t a t i o n s . In typical cases of epidemic encephalitis, its *acute stage* begins after the incubation period, the duration of which can vary from 1 to 14 days. Acutely a headache, marked state, afflicted - Nia, general weakness, catarrhal phenomena, the temperature rises more often up to

38 ° C. Patients become lethargic, drowsy. From focal Neuve - rologicheskih symptoms of the earliest and constant are at - signs the defeat of the nuclei of the oculomotor nerve: bilateral, usually part-time, one- or two-sided ptosis, infringement of convergence and accommodation, paresis or paralysis of external eye muscles and is due - Noah this double vision (diplopia) gaze disorders, first of all. Turning the gaze up. *Athanassio's syndrome* (*inverted* Argyll Robertson's syndrome) is also considered characteristic : the lack of reaction of "crustaceans" to accommodation and convergence, while their response to light is preserved . Fever, sleepiness and oculomotor disorders compiled dissolved Economo triad typical of an acute stage of epidemic Enz - falita. Less often, the nuclei of the facial and vestibular cochlear nerves are affected, which leads to the development of paresis of facial muscles, dizziness, sometimes with nausea, vomiting, horizontal or rotatory nystagmus. Asthenia, weakness, abulia are characteristic .

Abnormal sleepiness in typical cases of epidemic Enz - falita so pronounced that the disease was named lethargic encephalitis. Patients fall asleep in any position: while eating, talking, etc. A sleeping patient can always be woken up if desired, after which he can adequately answer questions, perform persistently presented simple tasks, but then falls asleep again. Sometimes the disorder of sleep manifests persistent insomnia that occurs at the beginning of Zabol - Bani or at the end of a pathological sleep. An inversion of the sleep formula is also possible : the patient sleeps during the day and cannot fall asleep at night. Naru - sheniya sleep, more drowsiness can last 2-3 weeks, and sometimes *have* more.

For epidemic encephalitis, pyramidal and other conduction disorders are not characteristic. However, manifestations of subcortical pathology in the form of hyperkinesis of the type of chorea, athetosis, tremor or myo] CLONUI, gaze cramps, blepharospasm are POSSIBLE . Somewhat less marked element cops akinetic-rigid syndrome: hypokinesia, oligokineziya we - antiplaque rigidity, susceptibility to catatonia. As a rule, expressed Vega - -commutative violations: vasomotor lability cutaneous vascular reactions, hyperhidrosis, hypersalivation, overproduction of sebaceous glands ("sal Noah" person). Possible polymorphic disorders of the psyche of patients, more often changes in the level of consciousness, hallucinations, delirium.

In the blood, minor changes are common: erythropenia, decreased hemoglobin, a slight shift in the leukocyte formula to the left, eosinophilia , increased ESR. Urine was normal. CSF with a lumbar puncture flows out under slightly increased pressure. Its composition is not changed, or it reveals a small (up to 40 cells in 1 μl) lymphocytic pleocytosis, sometimes the protein content is slightly increased (up to 0.5-1 g / l)

ACUTE MYELITIS

Myelitis is an inflammation of the spinal cord that affects both white and gray matter.

Etiology and pathogenesis. Allocate Institute - infectious, intoxication and herbs - matic myelitis. Infectious Mie - lites may be primary, are caused by - E neyrovirusami *(Herpes zoster,* viruses, polio, rabies) caused - E tuberculous or

syphilitic lesions. Secondary myelites fuss - cabins as a complication of diseases obscheinfektsionnyh (measles, scarlet fever, typhus, pneumonia - Monia, flu) or any purulent focus in the body and sepsis. If your primary - GOVERNMENTAL infectious myelitis infection spreads hematogenically, the defeat of the brain precedes viremia. In the pathogenesis of secondary infectious myelitis, the allergic factor and hematogenous transport of infection into the spinal cord play a role . Intoxication myelitis is rare and can develop as a result of severe exogenous poisoning or endothelial intoxication. Traumatic myeitis occurs with open and closed injuries of the spine and spinal cord with the addition of a secondary infection. Cases of post-vaccination miele litas are not uncommon .

Pathomorphology. Macroscopically, the brain substance is flabby, edematous, bulging , on the cut, the "butterfly" pattern is smeared, microscopically , hyperemia, edema, minor hemorrhages , infiltration of formed elements , cell death, myelin breakdown are found in the area of the focus

The clinical picture of myelitis develop camping acute or subacute within the context of John infectious symptoms: increase the perature to 38-39 ° C, chills, Celje - Niya. The neurological manifestations of myelitis begin with moderate pain and parasthesia. In the lower limbs, back and chest , which are radicular in nature. Within 1-3 days, motor sensory and pelvic disorders appear, increase and reach their maximum .

The nature of the neurological symptom is determined by the level of the pathological process. In myelitis of the lumbar spinal cord, peripheral paraparesis or paraplegia of the lower extremities is observed with atrophy, birth reaction , absence of deep reflexes , and pelvic disorders in the form of true urinary and fecal incontinence. When myelitis thoracic spinal cord occurs spastic paralysis of the legs with hyperreflexia, clonus, pathological Ref Lex, loss of abdominal pgg owls, pelvic disorders as urinary retention and fecal incontinence in passing. In case of suddenly developing transverse myelitis, muscle tone, regardless of the localization of the focus, can be low for some time due to the phenomena of diachysis. When the spinal cord is affected at the level of the cervical thickening, the upper flaccid and lower spastic paraplegia develop. Myelitis in an upper portion of the spinal cord is characterized by spastic tetraplegia, lesion phrenic nerve disorder rows Haniya sometimes tabloid violations - E. Sensory disturbances in the form of hypesthesia or anesthesia are of a conductive nature, always with an upper limit corresponding to the level of the affected segment. Quickly, sometimes during the first days, pressure sores develop on the sacrum, in the region of the greater trochanters, femurs, and feet. In more rare cases, the inflammatory process covers only half of the spinal cord, which gives a picture of Brown-Séquard syndrome.

Forms of subacute necrotizing myelitis are described , which is characterized by a lesion of the lumbosacral part of the spinal brain, followed by the spread of the pathological process upward, the development of bulbar disorders (lethal outcome. In cerebrospinal fluid in myelitis, an increased content of protein and pleocytosis is found. to be on linuclears and lymphocytes. With

liquorodin amic tests there is no block. In the blood there is an increase in ESR, leukocytosis with a shift of the formula to the left.

Course and forecast. The course of the disease is “three”, the process reaches its highest intensity after a few days, and then remains stable for several weeks . The recovery period lasts from several months to 1-2 years. The fastest and earliest recovery is sensitivity, then the functions of the pelvic organs; motor disorders regress slowly. Often, minor paralysis or paresis of the limbs remains . The most severe downstream and about - are cervical myelitis due to e tetraplegia, proximity vitally important centers, respiratory disorders, the prognosis is favorable when myelitis thoracicoinferior and lumbosacral localization due to severe lesions , poor recovery of the pelvic organs, joining secondary inf projection (bedsores, urosepsis) ... *Diagnosis and differential diagnosis.* Acute onset with the rapid development of cross-spinal lesion to background obscheinfektsionnyh Symposium volumes, the presence of inflammatory changes - Nij in the cerebrospinal fluid in the absence of the block sub arachnoid space makes dia - prognosis sufficiently clear. However, it is important - but to diagnose epidemiological rit, the clinical picture is in pain - most cases indistinguishable from myelitis, but that requires urgent hi - rurgicheskoe intervention. The dubious - GOVERNMENTAL cases should resort to Explora tive laminectomy. When diagnosing epidurita should bear in mind the presence of purulent focus in the body, to the appearance - reshkovyh pain syndrome increasing compression of the spinal cord. Acute poly radiculoneuritis Guillain-Barre syndrome is characterized by the absence of conductive myelitis sensitivity disorders spastiches - FIR phenomena and pelvic disorders. Swelling - if the spinal cord characterized by slow-flowing, with a clearly defined stage to - reshkovyh pain, the presence of protein-cell dissociation in the cerebrospinal fluid, the block at likvorodinamiches FIR samples. Hematomyelia and hematorchis occur suddenly, are not accompanied by a rise in temperature; with hematomyelia , it is mainly the gray matter that is affected; hemorrhage under cladding originated - dissolved meningeal symptoms. The history often reveals indications of trauma.

In acute transverse lesions of the spinal cord is necessary differential - tion from acute disorders spinnomozgo - Vågå circulation. It is suspected multiple sclerosis, but for him ha - acteristic electoral defeat and white - of the substance; rapid and significant reg - res symptoms after a few days or weeks, signs scattered lesions brain and spinal cord. Chronic meningomyelitis differs slower development, lack of temperature rise and often Obus - catching syphilitic lesions that set using serologiches - FIR reactions.

Treatment. In all cases, should be desig - chat dei broad-spectrum antibiotics - Corollary to the highest possible doses. To reduce pain, and at the high - perature shown antipyretics. Prima - nyayut glucocorticoid hormones in a dose of 50-100 mg per day (or equivalent to PS dexamethasone or triamcinolone) CRF in a dose of 40 IU twice daily for 2-3 weeks. with a gradual dose reduction. Particular attention should be paid to preventing the development of pressure ulcers and ascending urogenital infection. For the prevention of pressure ulcers, part of the WHO - arising, over bony prominences, pain - Foot to be installed in a circle, under the heel underlay cotton proklad - ki, a daily wipe the body of

camphor spirit, to change the position. When the appearance - SRI bedsores necrotic tissue Isse - cabins and bandage with penicillin or tetracycline ointment, salve Wisniewski. To prevent Obra - mations bedsores and post-emergence is carried out ultraviolet irradiation Iago - Dietz, rump, feet.

The first delay period of the disease urine can sometimes be overcome use - it anticholinesterase drugs; if is it is not sufficient, req - Dima catheterization with lavage urinary - of bladder antiseptic solutions.

To prevent the development of kontrak - tour on the first day of the disease should be a passive exercise therapy and put the patient in the bed, straightening his legs in the hip and knee joints, and bent at the ankle - GOVERNMENTAL, what used rollers and spe - cial tires. After the acute period (. 2-4 weeks depending on the severity zabole - Bani) needs to move to a more active remediation: mass - Ms, passive and active exercise therapy, iglotera - FDI, physiotherapy. Showing vitamins of group B, Neostigmine, dibazol, galantamine, biostimulants, absorbable prep - you. With a sharp spasticity, seduxen, elenium, melliktin, baclofen, midocalm, sirdalud are used. In the future recom - mended spa treatment.

Ability to work. Determined by locale - zatsiey and prevalence of the process, the degree of movement disorders and pelvic - O functions, sensory disorders. In the acute and subacute periods, patients are temporarily disabled. With a good recovery of functions and the possibility of returning to work sick leave mo - Jette be extended to practical vyzdo - rehabilitation pro gram. In case of residual effects in the form of a slight lower paraparesis with weakness of the sphincters, the patient is assigned a III disability group. When he died Mr. lower paraparesis, violating campaign ki and statics patients can not work in a normal production conditions and recognized disability group II, patients in need of a permanent post ronnem care (paraplegia, tetrapareses disorders of pelvic organs), set the I group of disability. Whether during dynamic observation of MSEC and medical institutions for 4 years, the restoration of the impaired functions does not occur, the disability group is established indefinitely.

POLIO

Poliomyelitis (infantile paralysis, Heine-Medin disease) is an acute infectious disease caused by a virus that has a tropism for the motor neurons of the anterior horns of the spinal cord and motor neurons of the brain stem (nucleus of the cranial nerves), the destruction of which leads to paralysis of muscles and their atrophy.

Epidemiology. Until the middle of the XX century. the outbreak of the poliomyelitis epidemic, nowadays, thanks to the widespread immunization of children with the polysmyelitis vaccine, only sporadic cases are encountered . The number of healthy carriers and abortive cases, when recovery occurred before the development of paralysis, significantly exceeds the number of patients in the paralytic stage. It is healthy carriers and abortive cases that are the main carriers of the disease, although it is possible to get infected from a patient in the paralytic stage. The main routes of transmission are personal contact and fecal contamination of food. This explains the seasonality with the maximum incidence in late summer and early autumn. At the age of 5 years, the susceptibility decreases

sharply. The incubation period is 7-14 days, but it can last up to 5 weeks. In the XXI century. a sharp decrease in the incidence rate is observed in those countries where prophylactic inoculations with the Salk, Seibin and A.A. Smorodintsev vaccines are carried out.

Etiology and pathogenesis. Three are highlighted; virus strain: types I, II and III. The virus can be isolated from the mucous membrane; nasopharynx of patients in the acute stage, zdo - rovyh of virus, recover, and from feces. In humans, the most common route of infection is through the digestive tract. The virus reaches the nervous system via vegetative fibers and perineurally along axial cylinders in peripheral nerves and in the central nervous system. It is believed that it can spread through the blood and lymphatic system. The site of introduction of the virus can be the pharynx, especially the tonsil bed after tonsillectomy . The virus is resistant to chemical agents , but sensitive to heat and desiccation. It can be grown in monkey kidney cell culture . Specific serological tests are used, including complement fixation and antibody neutralization tests.

Pathomorphology. The spinal cord is re - complete blood count, Patriotic, soft, gray The substance in TBE - small areas of hemorrhage. Histological changes are most pronounced in the gray matter of the spinal cord and medulla oblongata. The basal nuclei and cortex are slightly affected . In the motor cells of the anterior horns, various changes are noted - from mild chromatolysis to complete destruction with neuronophagy. The essence of inflammatory changes consists in the formation of perivascular, predominantly from lymphocytes, with a SMALLER amount of polymorphonuclear cells , and diffuse infiltration of gray matter by these cells and cells of neuroglial origin. Recovery is characterized by the return to normal of those ganglion cells that were not very grossly damaged. cells do not disappear completely. In the anterior horns of a small number of detected cells with secondary degeneration anterior roots and peripheral nerves, n orazhennyh muscles are varying degrees of neurogenic atrophy with increasing connective and adipose tissue.

The clinical picture. There are 4 types of reactions to the poliomyelitis virus: 1) the development of immunity in the absence of symptoms of the disease ; *2)* symptoms at the stage of viremia , which have the character of a general moderate infection without involvement of the nervous system in the process (abortive cases); the presence in many patients (up to 75% during the epidemic) of fever, headache, malaise; there may be meningeal phenomena, pleocytosis in the cerebrospinal fluid. Paralysis does not develop; 4) the development of paralysis (in rare cases).

In the subclinical form, there are no symptoms. When abortive form about - events are indistinguishable from any sheathe Institute - fektsii. Serological tests positive - tion, it is possible to isolate the virus. In the remaining - GOVERNMENTAL patients unable to allocate two hundred - di- clinical picture: preparalitiche ical and paralytic.

Preparalytic stage. During this stage, two phases are distinguished. In the first phase, with fever, malaise, headache, drowsiness or insomnia, sweating,

redness of the throat, ventricular - but-intestinal disorders (anorexia, PBO - one diarrhea). This phase of the "small illnesses" long - camping 1-2 days. Sometimes after it comes time improvement with a reduction in rate - ture for 48 hours or disease passes into the second phase - the "big disease", while to - Torah headache is stronger and with - accompanied by back pain, limbs, fatigue muscles. Symp - toms resemble other forms of viral meningitis. In the absence of paralysis, the patient recovers. In tserebrospi - pressure tional liquid increased, pleocytosis (50-250 in 1 mm). Initially have - camping and polimorfonukleary, and lymphocytes, but after 1 week - only lymphocytes . The level of proteins and globulins is moderately increased . The glucose content is normal. During the 2nd week of the level of white - ka in the cerebrospinal fluid increases. Preparalitiches - kai stage lasts for 1-2 weeks.

Paralytic stage. Spinal odds - ma. Development of paralysis precede expression - conjugated fasciculations. There have been a pain in the extremities, increased sensitivity - Nosta muscle pressure. Paralysis may be widespread or localized - GOVERNMENTAL. In severe cases of possible motions - zheniya, except for a very weak (in the neck, trunk, limbs). In less cha - severe cases, attract the attention of asym - metrical paralysis: muscle may be severely affected on one side of the body and stored on another. Usually, paralysis reaches its maximum within the first 24 hours, less often the disease progresses. When "ascending present" embodiment paralysis of legs Prevalence - nyayutsya up and threaten life because when - respiratory disorders compound. Meeting - are descending and options for the pas - ralichey. It is necessary to monitor the function of the intercostal muscles and the diaphragm. Test to detect respiratory paresis - loud counting in one breath. If the patient is not able to count to 12-15, there are expressed - zhennaya respiratory insufficiency and follows - blowing forced measured breathing volume to determine the need SUB - gatelnogo respiration.

Improvement usually begins by the end of the 1st week after the onset of paralysis. As with other lesions Peripheral - Sgiach motoneurons, says the loss or reduction of skin and deep reflexes. No violations of sensitivity, rarely upset the function of sphincter pelvic - O bodies.

Stem form (polioencephalitis). On - are observed paralysis of facial muscles, tongue, pharynx, larynx, and rarely - glazodviga - Telnyh muscles. Golovokruzhe possible - of nystagmus. Great danger of involving the vital centers in the process of (respiratory - tion, cardiovascular). It is very important to distinguish between respiratory problems due to the accumulation of saliva and mucus when paralysis of the pharyngeal muscles of the true pas - ralichey respiratory muscles.

Diagnosis and differential diagnosis. ACT - radicheskie cases should be differentiated - Vat from myelitis other etiologies.

In adults, poliomyelitis should be differential - rentsirovat with acute transverse myelitis - if and Guillain-Barré-Shtrolya. However, in the first case, flaccid paralysis of the legs is combined with pathological foot marks, sensory disorders, and loss of control over the sphincters of the pelvic organs. In infectious and allergic poliradikulomielonevrite Guillain-Ba - PPE-Shtrolya paresis asymmetric and transformations - possesses in the distal extremities, in the cerebrospinal fluid

increased protein content, however pleocytosis is rare. Bulbar polio form should be differentiated from other Enz - falitov that is specified using serolo - cal tests and virus isolation.

Treatment. If you suspect a polyomavirus - lit. should immediately create the pain Nome complete rest, as physical ak ciency in preparaliticheskoy stage of Witzlaus risk of severe paralysis when there is paralysis treatment tactics depends on their prevalence in the respiratory tion and bulbar muscles. When treating patients without respiratory disorders , intramuscular administration of ribonuclease and convalescent serum is indicated . In the acute stage, give a sufficient amount of liquid. Lumbar puncture is necessary for diagnostic purposes and can also relieve headache and back pain. Analgesics and sedative drugs (diazepam) are used for about legcheniya pain and reduce anxiety. The only acceptable form of activity is light passive movements. Antibes tics prescribed only for the prevention of pneumonia in patients with respiratory races - stroystvami. Immunoglobulins are useless , since the virus, after connecting to the nervous tissue, is unattainable for antibodies.

Treatment after the development of paralysis is divided into stages.

In the acute stage, with pain and increased muscle sensitivity (3-4 weeks), it is important to prevent stretching of the affected muscles and contracture of the antagonists, which may require longer treatment. Pain - Noah must lie in a soft bed, finite Nosta should be put in such SRI to paralyzed muscles were relaxed (not stretched). For this, pads and sandbags are used.

In the convalescent stage with continued build-up of muscle strength (6 months - 2 years), physical exercises are important , which the patient performs with outside help, in a bath or in apparatus with support with straps and straps.

In the residual stage, in the presence of contractures, tenotomy or other surgical intervention is performed . They mean proserin, dibazol, vitamins, metabolic agents, physiotherapy.

With the threat of respiratory failure STI sometimes for weeks or even months is needed mechanical ventilation, when the normal con - centration PCO_2 and PO_2 can be subtree Jean only excessive, exhausting effort of the patient.

With bulbar paralysis, the main danger is the ingress of fluid and secretions into the larynx, sucking them in when inhaling. Difficulties in feeding patients are aggravated by diphagia. The correct position of the patient (on his side) is important , and every few hours he should be turned on the other side *; the foot end of the bed is* raised 15 °. This position can be changed for grooming or other purposes, but not for long. The secret is removed by suction. After 24 hours of fasting , the patient should be fed through a nasogastric tube. *Forecast.* The mortality rate during epidemics was 5-25%. The cause of death is usually respiratory distress in bulbar forms or ascending paralysis, when the intercostal muscles and the diaphragm are involved . The mortality rate has become significantly lower with the introduction of mechanical ventilation. At the mo aschenii progression of paralysis feasible to recover. Favorable conditions are the presence of voluntary movements, deep reflexes and muscle contraction caused by nerve stimulation for 3 weeks. after the development of paralysis. The incipient improvement can last for a year, sometimes more. *Prevention.* Due to the fact that the

virus of polio found in saliva, urine and feces, the patient is required not less insulation - over 6 weeks. In feces, the virus after a week. found in 50% of patients, and in 1 5-6 weeks. - at 25%. Children who have had contact with a polio patient should be separated from other children for 3 weeks. from the moment of isolation of the patient. Modern immunization is a more successful measure of limiting the spread of epidemics. Attenuating ovannaya polio vaccine 1 to 2 drops on a piece of sugar) produces immuno ordre for 3 years or more.

Test questions on the topic: Meningitis, myelitis, poliomyelitis .

1. Classification of meningitis.
2. What are the general symptoms of meningitis.
3. What are the clinical manifestations of meningococcal meningitis.
4. What are the treatment and prevention of meningococcal meningitis.
5. What are the clinical manifestations and treatment of secondary purulent meningitis.
6. What is the recognition and treatment of tuberculous meningitis.
7. What are the clinical manifestations and treatment of acute and serous meningitis.
8. What are the clinical appearance of myelitis.
9. What are the methods of myelitis treatment and prevention of complications.
10. What are the etiology of poliomyelitis and how the infection spreads?
11. What are the clinical manifestations and periods of the course of poliomyelitis?
12. What are the principles of poliomyelitis treatment in different periods?
13. What is polio prevention?

<u>Tests of the I level of assimilation *(1st* option)</u>

a) Identification tests

I. Is a recurrent course of meningococcal meningitis possible?

II. Are tuberculous tubercles located at the base of the brain?

III. Does CSF Sugar Decrease in Primary Serous Meningitis?

IV. Is a fibrinous film in the cerebrospinal fluid characteristic of syphilitic meningitis?

V. Is the fulminant development of tuberculous meningitis characteristic?

Vi. Is it possible to damage the cranial nerves with meningitis?

Vii. Is a decrease in chlorides in the cerebrospinal fluid typical for tuberculous meningitis?

b) Tests for discrimination

I .. Indicate at what meningitis there is a decrease in blood sugar in the cerebrospinal fluid?

a) Meningococcal. b) Secondary purulent, c) Tuberculous, d) Primary serous.

I of I of . Indicate in what forms of poliomyelitis breathing is impaired?

a) Abortive, b) Bulbar, c) Spinal, d) Encephalitic.

II I. Specify how the cerebrospinal fluid changes in the preparative period of poliomyelitis?

a) Lymphocytic pleocytosis. b) Neutrophilic pleozotosis.

c) Classification tests

I. Which of the following symptoms: a) conductive disorders sensitivity - telnosti b) spasticheekaya paraplegia in) slack monoplegiya d) dysfunction of the pelvic organs, etc.), peripheral facial paralysis, d) a pair of bulbar Lich - relate to: 1) myelitis and 2) poliomyelitis?

II. Which of the following indicators of the study of cerebrospinal liquid STI: a) an increase in pressure, b) opalescent, c) dairy, d) formation of captive - ki, d) the formation of clots, e) lymphocytes, g) the majority of polynuclear-stand - and, h) a moderate increase in protein i) a significant increase in protein, j) a moderate decrease in glucose - characteristic for 1) tuberculous and 2) meningococcal meningitis?

TESTS of the <u>I level of assimilation (2nd variant)</u>

a)* Identification *tests

1. Is lymphocytic pleocytosis observed in meningococcal meningitis?
2. Is ataxia possible with arachnoiditis of the posterior cranial fossa?
3. Are segmental disorders typical for myelitis?
4. Are general cerebral symptoms typical for meningitis?
5. Are conductive sensory disturbances typical for poliomyelitis?
6. Can peripheral facial nerve palsy occur in poliomyelitis?
7. Are gastrointestinal disorders possible during the preparative period of poliomyelitis?

b) Tests for discrimination

I. What are the symptoms of meningitis? a) Headache b) Vomiting c) Paraplegia d) Claude-Bernard-Horner syndrome e) Stiff neck f) Kernig's symptom g) Brudzinsky's symptom h) General skin hyperesthesia.

II Indicate what signs are characteristic of myelitis? a) Hemiplegia b) Disorders of the functions of the pelvic organs c) Itsenko-Cushing's syndrome d) Conductive disturbance of sensitivity e) Spastic para- or tetraplegia.

III Specify what symptoms are characteristic for arachnoiditis of the pons-cerebellar angle?

a) acalculia b) in nystagmus) Scanning speech g) Ataxia d) Paraplegia e) Hypotension muscles g) Reduction corneal reflex, s) Ptosis, u) Lowering cl ear k) peripheral facial nerve paresis.

c) Classification tests

Which of the following symptoms: a) Claude-Bernard-Horner syndrome. b) conduction disturbance of sensitivity from the D10 level, c) disorders of the functions of the pelvic organs, d) conduction disturbance of sensitivity from the C5 level, e) lower spastic paraplegia, f) flaccid paralysis of the upper and spastic paralysis of the lower extremities -

refers to myelitis: 1) the thoracic spinal cord and 2) the area of the cervical thickening of the spinal cord?

Tests of the I level of assimilation (3rd option)

a) Identification tests

I. Does purulent meningitis occur with inflammation of the paranasal sinuses?

II. Can there be radicular pain with arachnoiditis of the brain?

I.II. Are attacks of Jacksonian epilepsy observed in meningitis? b) Tests for discrimination

b) Identification tests

I.. What are the symptoms of fulminant form of meningococcal meningitis? a) Violent onset, b) disturbance of consciousness, c) transient membrane syndrome, d) sharp rise in temperature, e) disturbance of respiration and cardiac activity, f) pronounced changes in cerebrospinal fluid.

II. What are the symptoms of posterior fossa arachnoiditis? Ataxia, b) adiadochokinesis, c) attacks of Jacksonian epilepsy, d) muscle hypotonia, e) aphasia, f) nystagmus, g) dizziness, h) headache, i) nausea, vomiting, j) congestive nipples, k) hemiplegia.

c) Classification tests

I. Which of the following signs: a) a long prodromal period, b) acute development, c) a sharp rise in temperature towards the end, d) minor meningeal symptoms, e) opalescent cerebrospinal fluid, f) positive protein reactions, g) lymphocytic pleocytosis, h) a film of fibroids , and) a small lymphocytic leukocytosis in the blood -

typical for: 1) tuberculous and 2) acute serous lymphocytic meningitis?

II. What are the symptoms of: a) flaccid paralysis of the legs, b) a spastic tetraplegia, c) races - stroystva breathing d) violation of the sensitivity level of D12; e) violation of sensitivity from the C1 level, f) possible tabloid symptoms - are characteristic of myelitis: 1) the lumbar spinal cord and 2) the upper cervical segments of the spinal cord?

Tests of the II level of assimilation (1st variant)

a) Substitution tests

List the features of the clinical picture of epidemic spinal meningitis. (1-3)

List the common symptoms characteristic of all forms of meningitis. (1-7)

List the preferred localization of the pathological process in poliomyelitis. (1-5)

b) Constructive tests

1. Name the features of cerebrospinal fluid in meningococcal meningitis.

2. Name the nature of acute inflammatory changes in poliomyelitis from the nervous system.

c) Task

A 21-year-old patient was admitted with complaints of severe headaches, vomiting, double vision . From the anamnesis it is known that he fell ill 10 days ago, when he felt unwell , general weakness, and a slight headache. There was a subfebrile temperature. The headache for 6 days gradually increased sharply to unbearable and appeared double vision, vomiting. Objectively: rigidity of the occipital muscles, Kernig's and Brudzinsky's symptoms, anisocoria, the left pupil is wider, ptosis on the left, divergent squint (there is no movement of the left eyeball inward) In the cerebrospinal fluid there is lymphocytic pleocytosis, the sugar content is low, a film has formed when the quor stands.

Mouth novite diagnosis. 2. Prescribe treatment.

Tests of the II level of assimilation (2nd variant)

a) Substitution tests

I. Pe rechislite the main symptoms of meningitis. (1-3)

II. Pe rechislite principles of treatment of purulent meningococcal meningitis (1 -9)

b) Constructive tests

I. What are the main signs of secondary purulent meningitis.

II. What are the complications of purulent meningitis?

c) Task

Patient N., 6 years old, fell ill 5 days ago, when the temperature suddenly increased to 39 C, severe malaise, general fatigue, sore throat, cough, runny nose appeared, after 2 days a severe headache, vomiting, back pain began. On the 4th day of the disease, paralysis of the lower extremities developed. Objectively: flaccid paralysis of the lower extremities, especially the proximal part. Peripheral paralysis of the facial nerve on the left. Sensitivity is not changed. CSF: lymphocytic pleocytosis.

1. Establish a diagnosis. 2. Prescribe treatment.

Tests II. **assimilation level (3rd option)**

a) **Substitution tests**

List the types of clinical course of poliomyelitis. (1-3)

List the features of changes in cerebrospinal fluid in meningococcal meningitis. (1-4)

List what changes in the internal organs in poliomyelitis can lead to the death of the patient? (1-4)

b) Constructive tests

I. What are the main methods of treatment of meningococcal meningitis.

II Name the pathomorphological changes in myelitis in the acute and late period.

c) Task

A 26-year-old patient was admitted to the clinic with complaints of girdle pain in the chest. Ill during the week, when the temperature rose to 37.6 ° C, there were catarrhal symptoms. The last 2 days - radicular pain, then the day of admission to the hospital it became difficult to walk due to weakness *in the* legs, difficulty urinating. Objectively: lower spastic paraparesis. Conductive disturbance of all types of sensitivity from the level of DZ on both sides. Naru - shenie pelvic organs. CSF: lymphocytic pleocytosis of 20-30 cells; protein - 0.6%.

1. Establish a diagnosis. 2. Prescribe treatment

TOPIC: "ENCEPHALITES"

Control questions

1. Definition of the concept of "encephalitis;
2. Classification of encephalitis.
3. In which parts of the central nervous system changes prevail in epidemic encephalitis?
4. What are the clinical manifestations of the acute period of epidemic encephalitis?
5. What are the clinical manifestations of the chronic stage of epidemic encephalitis
6. Treatment in acute and chronic periods of epidemic encephalitis.
7. Surgical treatment of parkinsonism and other diseases of the extrapyramidal system by the stereotaxic method.
8. Tick-borne encephalitis, periods of the course of the disease.
9. What are the clinical forms of tick-borne encephalitis?
10. Treatment of patients with tick-borne encephalitis.
11 Prevention of tick-borne encephalitis.
12. Comarine encephalitis and its clinical manifestations.
13. Treatment and prevention of mosquito encephalitis.
14. What are the clinical manifestations of lesions of the nervous system in influenza?
15. Treatment and prevention of neurological disorders in influenza.
16. In what disease does small chorea occur, what are its symptoms and treatment?

Tests of the 1st level of assimilation (1st option)

a) Identification tests

1. Is the acute development of the disease characteristic of tick-borne encephalitis?

2. Is it possible to develop Kozhevnikovskaya epilepsy with tick-borne encephalitis?

3. Is serotherapy (administration of convalescent serum) effective in the late period of tick-borne encephalitis?

4. Is oculoletargic syndrome typical for encephalitis lethargy ?

b) Tests for discrimination

I) What symptoms are characteristic of the chronic stage of epidemic encephalitis? 1) Paralysis of the limbs. 2) Violations of sensitivity. 3) Parkins onism.

II) What symptoms are characteristic of tick-borne encephalitis? 1) Bulbar disorders. 2) Violations of sensitivity. 3) Flaccid paralysis of the upper limbs. 4) Central paralysis.

c) Classification tests

I. Specify the preferred localization: a) the nucleus of the brain stem; b) the anterior horns of the spinal cord; c) the lining of the brain; d) cerebral cortex and subcortical white matter

- pathomorphological changes in case of: 1) poliomyelitis; 2) meningeal; 3) polioencephalomyelitis; 4) meningoencephalitis forms of tick-borne encephalitis.

I I. What are the main syndromes: a) spinal segmental paralysis; b) combined damage to the cranial nerves and the segmental apparatus of the spinal cord; a) meningeal , cerebral and focal syndromes; d) serous meningitis syndrome

- typical for clinical forms of tick-borne encephalitis: 1) meningeal, 2) meningoencephalitic, 3) polyencephalomyelitis, 4) poliomyelitic ?

III. What are the signs: a) seasonal diseases (spring-summer), b) an acute onset of the disease , c) giperkinezy d) drowsiness, etc.), eye movement disorder, e) akinesia, g) plastic muscle tone, h) hanging head, and) upper flaccid pair paresis, j) hiccups, l) vestibular disorders, m) bulbar disorders, n) hypersalivation -

have diagnostic value in: 1) tick-borne encephalitis and 2) epidemic encephalitis?

Tests of the I level of assimilation (2nd **option)**

a) **Identification tests _**

I. Is it possible the occurrence of encephalitis against the background of infectious diseases?

II. Is the lesion of the white matter of the brain characteristic of epidemic encephalitis
?

III. Are there specific agents for the treatment of the acute period of epidemic encephalitis?

IV. Is the chronic stage of epidemic encephalitis manifested by Parkinson's syndrome
?

V. Is it necessary to administer convalescent serum or gamma globulin to people who have been bitten by ticks in the taiga?

VI. Is Kozhevnikov's epilepsy a characteristic symptom of tick-borne encephalitis?

b) Tests for discrimination

I. What clinical forms are characteristic of tick-borne encephalitis?
1) Poliomyelitis. 2) Lathargic. 3) Meningeal. 4) Oculocephalic . 5) Meningoencephalitic. 6) Polioencephalomyelitis. 7) Poly diculoneuritic.

II. Indicate what stages of the course are characteristic of epidemic encephalitis?
I) Sharp. 2) Chronic. 3) Remitting.

Sh. Indicate which cells can be affected in the acute stage of tick-borne encephalitis : I) Motor neurons of the upper cervical segments of the spinal cord. 2) The neurons thalamo ca, 3) motoneurons motor nuclei XII cranial ngrvov, 4) motor neurons of the cortex, 5) motoneurons XI cranial nerves.

c) Classification tests

I. What are the signs: a) moderate fever, b; drowsiness, c) oculomotor disorders, d) flaccid paralysis, e) boulevard disorders, f) acute onset - are characteristic of the acute period: 1) epidemic encephalitis and 2) tick-borne encephalitis?

II. Which of the following features: a) glazodvigatelnye disorders, b) Vega - - commutative violations c) hemiparesis, g) the progression of extrapyramidal races - stroystv influenced by various stressful situations, myocardial infarction, decompensation of diabetes, cerebral vascular crises and strokes - characteristic for parkinsonism: 1) postencephalitic, 2) atherosclerotic

I II. Which of the following symptoms: a) lower spastic paraparesis, > nystagmus, c) ataxia, d) choreiform hyperkinesis characteristic for: 1) multiple sclerosis, 2) rheumatic encephalitis?

<u>Tests of the I level of assimilation (3rd option)</u>

a) Identification tests

1. Is the alimentary route of infection possible with tick-borne encephalitis?
2. Can parkinsonism be a manifestation of the chronic stage of the epidemic encephalitis?
3. Is the causative agent of epidemic encephalitis isolated?
4. Is Kozhevnikovskaya epilepsy a sign of chronic tick-borne encephalitis?
5. Can vestibular disturbances be observed in epidemic encephalitis?
6. Do lingering paralysis arise when poliomieliticheskoy form tick e ntsefalita?

b) Tests for discrimination

I. Indicate which of the symptoms are characteristic of epidemic encephalitis? 1) Sleep disorders. 2) Oculomotor disorders. 3) Imperative urge to urinate. 4) Hypersalivation. 5) Disappearance of abdominal reflexes. 6) Plastic muscle hypertension. 7) Hypomimia. 8) Vestibular disorders. 9) Retrobulbar neuritis.

II. In what clinical forms of tick-borne encephalitis is the prognosis more difficult? 1) Polioencephalomyelitis. 2) Meningeal. 3) Polyradiculoneuritic. 4) Meningoencephalitic.

III. Indicate with what diseases in the acute period tick-borne encephalitis is differentiated? 1) Amyotrophic lateral sclerosis. 2) Poliomyelitis. 3) Rheumoencephalitis. 4) Cerebrospinal epidemic meningitis. 5) Japanese encephalitis.

6) Epidemic encephalitis.

IV. What are the most important ways of treating the acute period of tick-borne encephalitis ? 1) Antibiotics. 2) Vitamins. 3) Convalescent serum. 4) Gammaglobulin.

c) Classification tests

Which of the following symptoms: a) stiff neck muscles, Kerny's symptom ; b) disorders of consciousness; c) epileptic seizures; d) central paresis or paralysis; e) kozhevnikovskaya epilepsy; f) flaccid muscle paralysis; g) bulbar disorders -

characteristic for each of the clinical forms of tick-borne encephalitis: 1) meningeal , 2) meningoencephalitis, 3) polioencephalomyelitis. 4) poly myelitis?

Tests of the II level of assimilation (1st variant)

a) Substitution tests

I. List the methods of therapy for post-encephalitic parkinsonism. (1-2)

II. List the main types of prevention of tick-borne encephalitis. (1-3)

III. List the methods of treatment in the acute period of tick-borne encephalitis. (1-5)

b) **Constructive tests**

I. Name the clinical types of the acute period of epidemic encephalitis a.

II. What are the clinical manifestations of influenza encephalitis?

III. What are the methods of treating influenza encephalitis?

c) Task

A 30-year-old patient has low-grade fever, diplopia, ptosis on the left, hyperhidrosis, hypersalivation , drowsiness, increased muscle tone according to the plastic type. In the cerebrospinal fluid: lymphocytic pleocytosis; in the blood - leukocytosis, accelerated ESR. EEG: desynchronization of the activity of cortical neurons.

1. Establish a diagnosis. 2. Prescribe treatment.

Tests of the II level of assimilation (2nd variant)

a) Substitution tests

I. List the clinical forms of tick-borne encephalitis. (1-6)

II. List the common symptoms characteristic of all forms of meningitis. (1-7) **b) Constructive tests**

I.. What are the clinical syndromes that can be observed in all forms of encephalitis?

II. ... Name the triad of symptoms of the acute period of lethargic encephalitis Econo mo

III. What are the clinical manifestations of influenza encephalitis?

c) Task

Patient, 12 years old, after tonsillitis there was a headache, gesticulations of grimaces, bizarre finger movements. Twitching worse with movement and disappear in sleep. Gordon's symptom is positive. Muscular hypotension. In the blood: leukocytosis and lymphocytosis. EMG shows salvo activity.

1. Establish a diagnosis. 2. Prescribe treatment.

Tests of the II level of assimilation (3rd option)

Substitution tests

1) List the clinical forms of tick-borne encephalitis. (1-6)

2) List the methods of treatment in the acute period of tick-borne encephalitis. (1-5)

Constructive tests

What are the methods of treating influenza encephalitis?

c) Task

A 12-year-old boy has become inattentive in the classroom, his working capacity has decreased, he gets tired quickly. Then involuntary polymorphic twitching appeared in the left hand, began to grimace, irritability increased. For several days, there is a rise in body temperature to subfebrile, once pain in the joints bothered . The examination revealed choreic hyperkinesis, diffuse hypotension, positive rheumatic tests ..

1. P leave the diagnosis, 2. What is the treatment plan.

Diseases of the peripheral nervous system.

The purpose of the lesson : to study the diseases of the peripheral nervous system, their classification, etiopathogenesis, clinical picture, individual forms, treatment features.

The student should know :

1. Ideas about diseases of the peripheral nervous system and their classification;
2. Etiopathogenesis of various forms of diseases;
3. Features of clinical forms and methods of their diagnosis, principles of treatment.

The student should be able to:

1. Examine a neurological patient;
2. To identify neurological symptoms, to determine the main clinical syndromes of damage to neurological systems and the localization of the pathological process;
3. Establish a clinical diagnosis;
4. Prescribe adequate therapy.

Classification

In accordance with the classification of peripheral neuropathies, based on the etiological and pathomorphological signs of WHO, 1982, all neuropathies are divided into axonopathies, myelinopathies and other types of neuropathies . In this case, *axonopathies* include genetically determined diseases: hereditary sensory neuropathies, peroneal muscular atrophy, Fabry, Bassen-Kornzweig, Leig disease, Tangier disease, Friedreich's ataxia, ataxia-telangiectasia. K acquired; axonopathy forms include: 1) neuropathies caused by exposure to or consumption of exogenous poisons and drugs, such as various metals - mercury, arsenic, zinc, aluminum, thallium; solvents - hexane, carbon tetrachloride, carbon disulfide, acetyl acetone; hi mical compounds belonging to different groups, - carbon monoxide; nitrogen dioxide, acrylamide, leptofox, organic phosphates; medicines - vincristine, phenytoin, amitriptyline, dapsone, isoniazid, food products - alcohol, lentils; invertebrate venoms - botulinum toxin, tick and black weaver spider venom; 2) neuropathies caused by metabolic disorders, such as diabetes mellitus, hypoglycemia, renal and hepatic failure, porphyria, mix deme; 3) neuropathies associated with a deficiency of thiamine, vitamin B, pyridoxine, pantothenic acid, riboflavin, food proteins 4) neuropathy from other causes: malignant neoplasms, myeoma , "senile" neuropathy.

By *mielinopatiyam*, genetically determined, attributed syndrome Sharko-Marie Dezherina- Sottas disease, Russi- Levy syndrome, bolez Refsum, adrenoleukodystrophy, Krabbe disease and Pelitsiusa- merzbach dick. In the group of purchased. myelinopathies included: 1) idiopathic, infectious or post-infectious neuropathies: acute (Guillain -Barré syndrome), chronic, recurrent, post-vaccination; 2) toxic neuropathies: diphtheria caused by lead, hexachlorophone , tellurium, acetylethyltetramethyltetralin - AETT, cyanides, etc .; 3) metabolic: with diabetes mellitus, dysproteinemia.

For *other types of neuropathies* include: 1) infectious neuropathies (neuritis in leprosy, shingles, viral neuropathies); 2) ischemic neuropathies; traumatic neuropathies; neuropathies from various reasons: neoplasms, amyloidosis, mucopolysaccharidosis, thermal lesions and electrical trauma, perineuritis, idiopathic Bell's palsy.

Acute demyelinating polyradiculoneuropathy

Acute or subacute peripheral, in typical cases ascending - conductive, paresis or paralysis in combination with protein-cell dissociation in CSF (Guillain-Barre syndrome, GBS) described in 1916, the French doctors G. Guillain (1875-1961) and J. Wagga (1880-1967).

E t i o l o g y . The reasons have not been specified. Almost 2/5 cases GBS devel - INDICATES soon after undergoing a common infection (ARI, SARS, influenza, gastrointestinal infections). In some cases, the disease occurs against the background of mononucleosis, acute exanthema, surgical intervention. Naib - Leah probable cause of the disease is an autoimmune disorder, occurring in 1-3 weeks after undergoing respiratory or zhelu - zling-intestinal infection, sometimes after exhaustion, hypothermia, and in some cases, and for no apparent reason.

C l i n i c a l manifestations. It is characterized by acute demya by linearization of the anterior nerve roots and by the motor portion of the trunks of

the spinal and cranial nerves. Less common is the axonal variant of the disease, which is more severe than its demyelinating form. In patients usually detected antibodies to various pathogenic bacteria and viruses (for Campylobacter, mycoplasmas, cytomegalovirus, a virus Epstein Barr et al.).

The disease is characterized by the syndrome of polyradiculoneuropathy, more often occurs in people in 25-50 years, although it can be observed at almost any age; according to A. Asbury et al (Asbury A. et al, 1987.), WHO. - tensile patients from 8 to 81 year.

The main early clinical manifestation of the syndrome Guillain - Barre syndrome is weakness, usually first appearing in the lower finite Nost, more proximal leg, rarely in the opening Zabolev - Niya notes and weakness of hands. Sometimes signs of peripheral paresis appear almost simultaneously in all limbs. At the same time, in the very first days, tendon reflexes decrease and disappear. Described movement disorders may not be accompanied by any Naru - sheniyami sensitivity, however, approximately half of the patients almost simultaneously with the appearance of weakness in the limbs there is numbness of distal department may aching pain in the hips, and the muscles of the pelvic and shoulder girdle; subsequently there may be re - tsidivy pains are usually already in bed-ridden patients, the pain is deep, aching, mainly in large muscles (in Jagodic - GOVERNMENTAL, calf, trapezoidal, in the quadriceps muscle). Pain can appear or intensify with muscle tension, often at night, sometimes with a burning sensation in the legs, similar to the "restless legs" syndrome.

Symmetrical flaccid paresis or paralysis of the limbs are the leading symptom of the disease. Movement disorders can increase in severity and prevalence within 2 to 4 weeks. At the same time , the diaphragm and other muscles involved in the act of breathing are often involved in the process . Sensitivity disorders are possible in the form of confident polyneuritic hypesthesia, sometimes disorders of proprioceptive sensitivity, which leads to a combination of muscle weakness with sensitive ataxia; GBS is often observed at positive Symp - toms relief (symptom Lasegue et al.). Approximately 15% of patients show severe autonomic disorders: orthostatic hypotension. accompanied by dizziness, tachycardia, arrhythmia, decreased sweating, bronchorrhea; sometimes - disorders pelvic functions - O organs, usually a delay of urination, the chair. Half of patients with paresis of upward movement extends to portions of Th - Repnev nerves (Landry's syndrome).

Landry's syndrome is an ascending polyradiculoneuropathy with the involvement of motor portions of the cranial nerves, primarily of their bulbar group, in the process: in this case, bulbar syndrome develops. Characterized by paresis or paralysis of the pharynx, soft palate ~ 7 ~ tongue, facial muscles; in this case , breathing disorders, swallowing, dysarthria occur. Sometimes paresis of the muscles that provide movement of the eyeballs, primarily their movement in the horizontal plane, develops . In a severe course of acute demyelizing polyradiculoneuropathy, manifestations of Landry's syndrome may occur soon after the first signs of the disease appear. The

syndrome was described in 1856 by the French physician J. Landry (1826-1865) and in 1859 by the German physician A. Kussmaul (1822-1902).

The most dangerous manifestation of the syndrome Guillain-Barré syndrome (GBS) is - by the expression *of respiratory muscle weakness,* occurring when - blizitelno y '/ s patients. In the case of bulbar disorders may occur and heart disorders, aspiration pneumo - Nia, where it may be necessary to use mechanical ventilation, cardiac facilities, other methods of intensive care, tube of feeding. The risk of developing respiratory disorders persists until the 21-28th day of illness.

The basis of GBS is polyradiculoneuropathy with segmental demyelination. In the acute phase of the disease in the blood plasma increases con - centration of immunoglobulins. Antibodies to peripheral nerve myelin can also be detected in the blood, in 20% of cases - an increase in the content of creatine phosphokinase (CPK). In the CSF, from the 5-10th day of illness, as a rule, protein-cell dissociation is detected, due to an increase in the protein content, usually up to 1-2 g / L, which is essential for clarifying the diagnosis of GBS.

Speed reduction of the seals on the pulse often proyav - wish to set up only after 7-10 days from the onset of the disease. Transient proteinuria is observed in approximately 5% of patients. In some cases, WHO - Nick manifestations intracranial hypertension may be due - hydrochloric difficulty venous outflow from the cranial cavity.

On the 3-4th week of illness, in most cases, there are signs of stabilization, continuing for 2-4 weeks, and on - next - the gradual regression of neurological symptoms. Vos - stanovitelny period is long: from a few months to a year and the pain - Shae. The majority of patients occurs almost complete vyzdorov - Lenie. Relapses observed in approximately 5% cases - s. In some patients who have had the disease in a severe form, preserves - nyayutsya residual effects, most peripheral elements tetrapa cut.

A variant of Guillain-Barré syndrome is a *Fisher syndrome,* described - ny in 1956, the American neurologist SM Fisher. For this sin - Droma characterized by chronic demyelinating poliradikulonevropa - ment to the development of the disease in the opening two-way outer (often *V* internal) ophthalmoplegia possible manifestations bulbar syndrome - ma and ataxia. Proximal peripheral paresis of the extremities when it develops later and spread downstream As with the typical form of GBS is characterized by protein-cell dissociation - tion to the CSF. Fisher syndrome is sometimes necessary to differentiate from the bot - ism, wherein in contrast to GBS occur bradycardia, typical reduction reaction the accommodation pupils.

From the syndrome of hemodynamic disturbance in the basilar artery, GBS is distinguished by the absence of tendon hyperreflexia and pyramidal signs. Lack of pupillary response in a conscious patient indicates a greater likelihood of GBS, rather than impaired circulation in the brain stem. It is also possible to differentiate GBS from myasthenia gravis, in which ptosis of the upper eyelids and weakness of the masticatory muscles are characteristic, which are rarely observed with GBS. The Somni -negative cases can be carried out prozerinovaya sample

usually gives a clear improvement in the function of the eye muscles in case miaste Research Institute **.**

T r e a t m e n t . With GBS, even in the case of severe movement disorders and respiratory disorders, there are still chances of satisfactory restoration of the impaired functions. At the same time, the prognosis is always serie zen, in this regard, the possibility of the necessary provision of resuscitation care to the patient is great.

Particular attention should be paid to the state of breathing. The first signs of respiratory failure characteristic of GBS are the inability to cough up, the weakening of the voice, the need for a pause during a conversation, difficulty in active inhalation, and a decrease in the vital capacity of the lungs (VC). In such cases, systematic monitoring of respiration and cardiac activity is shown ; with bulbar paralysis, it is often necessary to introduce a nasogastric tube, intubation, and connect a ventilator.

With severe movement disorders , plasmapheresis is indicated (with an exchange of 30-40 ml / kg in one session, up to 5 sessions in total, which are carried out every other day). The patient shows a passive and, if possible, and ac - tive physiotherapy, massage chest.

Launched in the first 2 weeks plasmapheresis it softens the severity organiches - Coy neurological disorders and helps to reduce the residual of the defect. The beneficial effect may be also on the course of treatment immu - noglobulinom (Sandoglobulin, etc.) at a dose 0.4 g / kg / day for 5 days. Until recently, treatment with methylprednisolone, dexazone or other corticosteroids was adopted, but the feasibility of such treatment is currently disputed.

Much attention should be paid to maintaining the purity and integrity of the skin, monitoring the functions of the pelvic organs (if necessary , catheterization is carried out, laxatives are prescribed). Needed regular and adequate food (in the case of bulbar syndrome - ma must be fed through a tube), the maintenance of water and salinity - Vågå balance and CBS. In the treatment of used hemodialysis overflow - Niya plasma.

We need total care, measures to prevent contractures, Trom - Bosa veins of the lower extremities, pneumonia. Particular attention should be paid to the rehabilitation of the patient. It should be a regular medi - naya physiotherapy, massage treatments; shows vitamin com - plexes, nootropic drugs, symptomatic agents.

Chronic demyelinating polyradiculoneuropathy

C l i n i c a l m a n i f e s t a t i o n s . The disease occurs underside - wills or subacute followed by progressive, recurrent or chronic monophasic current. It manifests itself in symmetric, mainly motor, to a lesser extent sensitive disorders, mainly in the distal extremities. WHO - can damage the respiratory muscles. With increasing severity of - bolevaniya possible cranial neuropathy: bulbar syndrome, bi - sided flaccid paralysis of muscles innervated III, V, VI, VII cranial nerves. Neri's radicular symptom, Lasegue's symptom , Tinel's symptom are positive. Vegetative-vascular and trophic disorders We mention - chayut mainly in the distal extremities, there may be occasional changes in heart rhythm, syncope, on - Rushen functions of the pelvic organs.

In the blood, desimmunoglobulinemia is detected, in the CSF, protein-cell dissociation. It marked slowing of nerve impulses predominantly in the distal peripheral ner - Islands. The disease occurs in people older than 40 years (more often men) and pro - be extended for many months and years.

F o r e c a s t . The prognosis is often poor. Described in 1982 as a chronic variant of Guillain-Bar - 're American neurologists Dyck, Lai and Ohta. However, identification with CT and MRI study of demyelinating lesions in the head the Ministry of Health - re allows us to consider the disease independent nosological odds mine.

Diphtheria polyneuropathies

With diphtheria, complications in the form of toxic polinsnropathy are often encountered . In this case, early and late complications of diphtheria are possible , which have a different clinical picture. Moreover, in recent years, there has been an increase in the frequency of severe forms of diphtheria polyneuropathy up to 55% (Piradov I.A., Popova L.M. et al., 1998).

C l i n i c a l m a n i f e s t a t i o n s . *Early diphtheria cranial polyneuropathy or diphtheria pharyngeal neuropathy* occurs on days 2-4 of the disease, toxic polyneuropathy of the cranial nerves manifests itself , usually III, IV, VII, IX, X. Disturbed accommodation , paresis of facial muscles, manifestations of bulbar syndrome (dysarthria, dysphagia), while breathing disorders are possible. In the same period, toxic diphtheria myocarditis often develops.

The pathogenetic basis of lesions of the nervous system in such cases in the first 2 weeks of clinical manifestations of diphtheria is toxic peripheral myelinopathy. The toxin suppresses the synthesis of myelin basic protein by 75% and myelin proteolipid by 50%. Demyelination is the pathophysiological basis of diphtheria polyneuropathy. In this case, segmental demyelination without significant involvement of the axons of the affected nerve fibers in the process is further replaced by remyelination, which determines a gradual regression of signs of polyneuropathy and usually the absence of residual effects.

Late diphtheria polyneuropathy ("fiftieth day" syndrome, Glanzmann - Zaland syndrome) manifests itself in 10% of patients with diphtheria after 2-8 weeks, sometimes 12 weeks after the onset of the underlying disease. Until recently, late neurological complications of diphtheria, usually presented in the form of diphtheria polyneuropathy with severe movement disorders, mainly in the legs, was regarded as a result of repeated dissemination of diphtheria toxin produced by diphtheria bacteria. However, as it turned out later, in most patients, the body is quickly cleared of the causative agent of diphtheria [Favorova L.A. et al., 1988]. Therefore, at the present time, late neurological complications of diphtheria are usually associated with the occurrence of an autoimmune process in the body [Mozolevsky Yu.V., Chernenko OA, 1991; Sorokina M.N. et al., 1996]. It is inappropriate to administer heterogeneous antidiphtheria serum in the later stages of the disease .

The occurrence of lesions of the nervous system in diphtheria can lead to violations of hemostasis caused by exposure to excite; diphtheria on the platelet link. The possibility of thrombohemorrhagic syndrome development in patients with diphtheria has been proved . This can explain the rare cases of development of

acute disorders of cerebral circulation in patients with diphtheria . The occurrence of a thromboembolic process is facilitated by cardiovascular pathology (myocarditis or endocarditis) that develops in 10-25% of cases .

Characterized by the *development of generalized polyradiculoneuropathy, which reaches its maximum severity by the 12th week of the disease due to (secondary myelinopathy*. In this case, movement disorders predominate , deep tetrapraesis is possible, changes in sensitivity of the polyneuritic type are less common, disorders of muscle- articular and vibration sensitivity are possible , causing sensitive ataxia, diphtheria psevdotabesu, and respiratory disorders in connection with paresis intercostal muscles Sometimes peripheral paresis propagation. - stranyayutsya bottom up, as in Guillain-Barre syndrome, involving a process of cranial nerves type Landry paralysis, which can lead to vital risks from respiratory disorders it is not characterized. - thorns of pain and muscle atrophy are possible hyperthermia to 38 "C, count. laptoidnoe state in the CSF is marked protein-cell dissociation, which usually develops from the 3rd week of illness Paresis to grow. - Stray changes on EMG, climax tion the severity of changes on EMG often appears only towards the stabilization of the clinical car - slime. Practical recovery at a favorable course of the disease - Niya occurs within 2-6 months.

The disease was described in 1935 by Swiss doctors: pediatrician E. Glanzmann (1887-1959) and therapist S. Saland.

Diabetic polyneuropathy

One of the most common forms of polyneuropathy - Diab - cally - manifested in 40-90% of patients, long-term (5-10 years of age and over) suffering from diabetes. Its options are: *distal symmetric polyneuropathy* (70% of all diabetic polyneuropathy), which can be mixed with motor - sensory, autonomic or similar advantages - but the motor, sensory (pain, paresthesia, hyperpathia, hypoesthesia, Sensi - tive ataxia) or autonomic (wasting of small muscles of the hand and feet, trophic ulcers, arthropathies of the Charcot joint type in the distal extremities, diabetic foot); *symmetrical proximal motor polyneuropathies* (diabetic neurogenic amyotrophy); not - symmetrical proximal motor neuropathy, multiple (usually the femoral, obturator, sciatic, median, ulnar nerve); *cranial nerve mononeuropathy,* the cup nerves, providing movement of the eyeballs (ophthalmopathy retaining pupillary rea - tions to the light); *mononeuropathy; tunnel mononeuropathies; asymptomatic - nye polyneuropathy* manifested only decline and loss of dry - veined, more heel and knee reflexes.

In diabetic polyneuropathy may vegetative Destroy - CTBA: bladder dysfunction, impotence, disorders potootdele - Nia, a disorder of the digestive tract function, orthostatic hypotension, painless myocardial infarction, rarely changes pupillary re - stocks.

P a t h o g e n e s i s . Are important chronic hyperglycemia, insufficient - current insulin accumulation in neurons sorbitopa disorders mikrotsir - kulyatsii in the nerves.

Axonal polyneuropathy Diabetic often, but when it WHO - are possible, and differ in the severity of symptoms of segmental demyelination. In the late stages

of diabetes mellitus, microvascular disorders often occur , leading to metabolic disorders in the perineurium.

With an expanded clinical picture of diabetes mellitus, it is usually possible to reveal a slowdown in the conduction of excitation along the motor nerves, signs of denervation of individual muscles, a slowdown in somatosensory conduction along the spinal cord.

C l i n i c a l m a n i f e s t a t i o n s . Diabetic polyneuropathy may be asymptomatic, while unable to identify a reduced heel and knee reflexes, and there may be heavy on the severity of the manifestations of forms, leading to disability of the patient's sensory disorders in diabetic polyneuropathy thickets are symmetric and appear in the distal extremities of the type ' ' socks and gloves ". They range from mild numbness to anesthesia. In this case , *arthropathies and trophic ulcers on the feet are possible.*

With diabetes mellitus, thick or those sensitive fibers of peripheral nerves can suffer to varying degrees , therefore, dissociation of the severity of disorders of various types of sensitivity is possible. With a predominant lesion of relatively thick myelinic sensory fibers, there is a predominance of deep sensitivity disorders in the legs, which can lead to clinical manifestations of the pseudotabetic form of diabetic polyneuropathy) The similarity with increases if the patient also has narrowed pupils and their response to light turns out to be sluggish. The involvement of thin sensory fibers in the pathological process can cause expressions of spontaneous deep, bursting, aching or superficial burning *pains, paresthesia and dysesthesia* or *hyperpathy,* usually in the diastal parts of the extremities, painful cramps like cramps are possible.

Vegetative disorders sometimes lead to the development of tachycardia, ort of static hypotension, intestinal hyperperistalsis, impaired discharge, impotence, and incomplete emptying of the bladder. Diabetic patients with autonomic neuropathy are more likely to die from cardiac arrest.

The incidence of all forms of diabetic neuropathy is in direct proportion to the duration of the disease in both type 1 and type 2 diabetes mellitus. At the same time, the importance of hypoinsulinemia and hyperglycemia is emphasized in their pathogenesis . *Diabetic proximal motor neuropathy is a* rare complication of diabetes mellitus. It is characterized by slowly progressive weakness of the muscles of the pelvic girdle of the thighs, primarily the iliopsoas and quadriceps femoris muscles, sometimes accompanied by aching pain in the thigh. It is assumed that this form of pathology is based on metabolic and vascular disorders.

Local and multiple diabetic mononeuropathies are usually characterized by *lesions of the femoral nerve* and *lumbar plexus* and develop rapidly within a few hours or several days. G is noted *severe pain in the front of the thigh,* reduction lennogo reflex, muscle loss, innervated *obturator ner tion,* and some other muscles. May be combined with distal sensory- motor polyneuropathy.

Mononeuropathies of the cranial nerves are *quite* common in diabetes mellitus . *Mononeuropathies of the optic nerve* are possible , but more often than other cranial nerves, the *oculomotor nerve is affected,* which manifests itself in

the form of ophthalmoplegia. Treatment: nootropics, B vitamins, thioctacid, symptomatic treatment.

Control questions on the topic: "Diseases of the peripheral nervous system"

1. What is the etiology and pathogenesis of vertebrogenic diseases of the peripheral nervous system?
2. What are the clinical manifestations of cervical, thoracic and lumbosacral radiculopathies?
3. What are the modern methods of diagnosis and differential diagnosis of vertebrogenic lesions of the peripheral nervous system?
4. What are the current approaches to the conservative and surgical treatment of vertebrogenic diseases of the peripheral nervous system?
5. What are the ways and methods of prevention of vertebral lesions Peripheral - tion of the nervous system?
6. What is the etiology of polyneuropathies?
7. What are the clinical manifestations of polyneuropathies?
8. What are the modern methods of diagnosis and differential diagnosis of polyneuropathies?
9. What are the current approaches to the treatment of polyneuropathies?
10. What are the ways and methods of prevention of polyneuropathies?
11 . What is the pathogenesis of facial nerve neuropathy?
12 . What are the approaches *to* treating facial nerve neuropathy?
13 . What is the pathogenesis of trigeminal neuralgia?
14. What is the clinical picture of trigeminal neuralgia?
15. What are the current approaches to the treatment of trigeminal neuralgia?
16. What is the pathogenesis of neuropathy of the radial, ulnar and median nerves?
17. What is the pathogenesis of neuropathy of the greater and peroneal nerves?
18. What is the pathogenesis of brachial plexitis?
19. What are the clinical manifestations of brachial plexitis?
20 . What are the etiology and pathogenesis of polyneuritis?
21. What are the clinical manifestations of polyneuritis?
22. What are the current approaches to the treatment of polyneuritis?
23. What are the etiology and pathogenesis of ganglionitis?
24. What are the current approaches to the treatment of ganglionitis?

Assimilation level tests (1st option)

a) Identification tests

I. Does hypothermia play a role in the development of radiculopathies?

II. Does degenerative lesion of mel- vertebral discs "

III. Are inflammatory changes in the peripheral blood characteristic of radiculopathy?

b) Tests for discrimination

I. Indicate which of the following symptoms are characteristic of radiculopathies of
the lumbosacral localization: 1) Antalgic posture. 2) Pain in the lumbar region. 3) Lower spastic paraparesis. 4) Neri's symptom. 5) Lack of Achilles reflex. 6) Lasegue symptom.

II. Note the signs of damage to the facial nerve: 1) Pain in the area behind the ear.

2) Decrease in superficial sensitivity on half of the face. 3) Paresis of facial muscles. 4) Lachrymation or dryness of the eye. 5) Divergent squint. 6) Decreased taste in the anterior two-thirds of the tongue. *7)* Hyperacusis. 8) Decreased brow reflex.

III. Specify the symptoms characteristic of polyneuropathy: 1) Hemiparesis. 2) Conductive disturbances of sensitivity. 3) Pain along the nerve trunks. 4) Flabby paresis of the hands and feet. 5) Muscular hypotension. 6) Decreased sensitivity in the distal extremities. 7) Symptoms of tension in the nerve trunks. 8) Vegetative-trophic disorders in the distal extremities. 9) Pathological foot reflexes. 10) Decreased tendon and periosteal reflexes

c) Classification test

Which of the following symptoms: a) pain and the proximal end; b) pain in the distal parts of the extremities c) sensory disorders of the root type, d) sensory disorders of the neuritic type; e) meningeal syndrome: f) protein-cell dissociation in the cerebrospinal fluid; g) systems of combined lesions of peripheral nerves that are part of a specific plexus; h) scoliosis with a bulge towards the lesion: i) ironed. lumbar lordosis; j) tension of long back mice are characteristic for: I) radiculopathy, 2) neuropathy, 3) plexitis.

Tests of the I level of assimilation (2nd option)

I. Is scoliosis in the lumbar spine typical for radiculopathy of the lumbosacral localization?

II. Is sensitive ataxia possible in alcoholic polyneuropathy?

III. Is the combination of motor, sensory and vegetative
trophic disorders in the distal extremities with neuropathy?

1 Tests for discrimination

I. Specify the symptoms characteristic of neuropathy of the femoral nerve: 1) Paresis of the iliopsoas. quadriceps and tailor muscles, 2) Paralysis of the calf of the leg muscle. 3) Lack of plantar reflex. 4) Lack of knee reflex. 5) Hypesthesia along the front of the thigh and the antero-inner surface of the lower leg, 6) Hypesthesia along the back of the thigh. 7) Symptoms of Wasserman and Matskevich. 8) Lasegue symptom.

II. Specify the symptoms typical for alcoholic polyneuropathies: 1) Proximal paresis of the extremities. 2) Conduction disorders of sensitivity 3,) Distal paresis

of the extremities. 4) Paresthesia and pain in the limbs. 5) Pain on palpation along the nerve trunks 6) Dysfunction of the vagus and phrenic nerves. 7) Decrease in superficial and deep sensitivity in the distal extremities. 8) Mental disorders

c) Classification tests.

Specify medicines used for: 1) Radiculopathy. 2) Neural gii . 3) Neuropathies:

a) Diclofenac. B) Novocaine blockade. C) Carbamazepine. D) Proserin. E) Vitamins of group B. E) Midocalm. G) Rumalon. 3) Aminazine. I) Indomethacin. K) Gangliosides. L) Suprastin.

Tests of the I level of assimilation (3rd **option)**

a) Identification tests

I. Can bulbar palsy develop with diphtheria polyneuritis?

II. Are the symptoms of loss of motor functions characteristic in neuropathy?

III. Is it possible to damage the oculomotor nerves in diabetic polyneuropathy ?

b) Tests for discrimination

I. Which of the following symptoms are characteristic of ganglionitis? 1) Radicular pain. 2) Tetraparesis. 3) Exudative eruptions. 4) Limited peripheral muscle paresis. 5) Sensory disorders

II . Which of the following symptoms are characteristic of the lower brachial plexi - ta? 1) Pain in the hand, in the supraclavicular region and along the nerve trunks. 2) Hypesthesia along the inner surface of the forearm and hand. 3) Increased carpo radial reflex. 4) Atrophy of the small muscles of the forearm. 5) Pain in the neck. 6) Paresis of the distal upper limb. 7) Decreased carporadial reflex. 8) Disorders of sensitivity on a conductive type. 9) Vasomotor disorders in the hand. 10) Horner's Syndrome.

III. Indicate which of the following syndromes can occur in osteochondrosis of the cervical spine: 1) Radicular. 2) Vertebrobasilar insufficiency. 3) The scalene muscle. 4) Hemiparesis. 5) Meningeal. 6) Hypertensive-hypokinetic. 7) Amyotrophic lateral sclerosis.

c) Classification tests

What research methods are most significant for diagnosis: 1) Radikulopa - TII and 2) Neuropathies:

a) Clinical blood test, b) Determination of blood and urine glucose levels, c) Study of cerebrospinal fluid. d) Spondylography. e) Myelography. f) Magnetic resonance imaging, g) Electromyography, h) X-ray of the spine,

Tests of the II **level of assimilation (1st variant)**

a) Staging tests

1) List the basic principles of neuropathy treatment (1-4)

2) List the basic principles of radiculopathy treatment (1-6)

b) Constructive tests

1) What are the indications for surgical treatment for neurological complications of osteochondrosis ?

2) List the additional research methods used for discogenic radiculopathies:

c) Problem.

A patient suffering from chronic alcoholism complains of pain and numbness in the feet , weakness in them. OBJECTIVE: flaccid paresis of the muscles of feet, muscle hypothetical rofii and hypotension. Absence of carporadial, Achilles and plantar reflexes on both sides. Hypesthesia in the area of the hands and feet.

1) Establish a diagnosis 2) Prescribe treatment

Tests of the II level of assimilation (2nd variant)

Staging tests.

1. List the main symptoms of polyneuropathy (1-5)
2. List the symptoms of an attack of trigeminal neuralgia. (1-4)

i) **Constructive tests**

What are the diseases of the peripheral nervous system?

c) Problem.

A 38-year-old patient after lifting the load felt a sharp pain in the lumbar region. The pain increased with movement. Objectively: tension of the long muscles from the pins, lumbar lordosis smoothed out. Right-sided scoliosis in the lumbar spine Restriction of movement in the lumbar spine in all directions. Pain of phi palpation in the **paravertebral** points in the lumbar spine. Lasegue's symptom on the right. Reduced Achilles and plantar reflexes on the right. Hypesthesia on the outer surface of the right leg. X-ray shows signs of spinal osteochondrosis. MRI revealed a paramedian hernia of 4 lumbar discs

1) Establish a diagnosis. 2) Prescribe treatment.

Tests of the II level of assimilation (3rd **option)**

a) Staging tests.

I. List the symptoms of facial nerve neuropathy. (1-7)

II. List the symptoms of ganglionitis (1-3)

b) Constructive tests

I. List additional methods for diagnosing neuropathy.

II. List the diseases of the peripheral nerves

c) Problem.

The patient 52 years after hypothermia were aching pain in the right lumbar region radiating to the front of the thigh and the inner surface of the right lower leg. Objectively: muscle tension in the lumbar region, flattening of the lumbar lordosis, limitation of movement in the lumbar region in the anteroposterior direction due to pain. Symptoms of Matskevich and Wasserman on the right. Reduced knee reflex on the right. Hypalgesia along the inner edge of the right lower leg. On radiographs of the lumbar spine: indications osteohon - Droz to lower the height of Lk-L4 disc. Flattening of the lumbar lordosis. 1) Establish a diagnosis. 2) Prescribe treatment.

HEREDITARY DISEASES OF THE NERVOUS SYSTEM.

The purpose of the lesson: to study hereditary diseases of the nervous system, their classification, pathogenesis, clinic, treatment of various nosological forms and methods of their prevention based on modern knowledge.

The student should know:

1. What does the concept of hereditary diseases include and what is their generality;
2. Classification and pathogenesis of hereditary diseases of the nervous system;
3. Clinic and treatment of certain nosological forms;
4. Modern methods of additional diagnostics and prevention of hereditary diseases of the nervous system.

The student should be able to:

1. Take anamnesis in order to establish hereditary burden with an emphasis on pedigree;
2. Identify the main clinical syndrome and, on this basis, establish a specific nosological form, determine the additional diagnostic methods necessary to confirm the diagnosis ;
3. Prescribe appropriate treatment.

Hereditary diseases are a wide range of diseases, the single etiological factor in which is the pathological heredity received by the patient's body through the germ cells of his parents. Depending on the mode of transmission, chromosomal and gene hereditary diseases are distinguished . It should be noted that the transmission of information to offspring is carried out through special structures of germ cells - the chromosomes of the cell nucleus, in which the functional units of heredity - genes - are concentrated. A change in the number of chromosomes or their structure (excess or shortage of genetic material in the chromosome) causes chromosomal diseases, while in true genetic diseases, the cytological picture of cells remains normal. The etiological factor of the disease is pathological mutant genes.

The pathogenesis of many hereditary diseases is still unclear. However, the study of hereditary phenomena at the molecular level using the methods of modern genetic research has shown that the main pathogenetic mechanism of hereditary diseases is metabolic disorders caused by gene mutation, while the synthesis of a certain polypeptide (structural protein or enzyme) is disrupted. As a result, a deficiency or inactivation of an enzyme (a group of enzymes) is found, which leads to insufficient or perverted synthesis of vital products, and this, in turn, to dystrophic or atrophic changes in tissues. In other cases, for the same reason, there is an accumulation of certain
substances that are deposited in organs and tissues violate them functioning.

General clinical features of hereditary nervous diseases

systems. United by the common pathogenesis, many hereditary diseases of the nervous system have other similarities:
a) the presence of repeated cases of the disease among relatives;
b) gradual progression of the disease without a visible connection with external factors (infection, trauma, etc.);
v) the systemic nature of the lesion and the symmetry of the pathology;
d) predominant damage to the nervous system.

The classification of hereditary diseases of the nervous system is still imperfect, due to the lack of a single principle, which in turn is associated with insufficient clarity of the pathogenesis of many diseases. Therefore, in addition to chromosomal and truly gene diseases , the following groups are currently distinguished:

1) Hereditary systemic degeneration of the nervous system:

1. Diseases with a predominant lesion of the cerebellum and its connections (familial ataxia of Friedreich, familial ataxia of Marie, olivopontocerebellar atrophy)
2. Combined degeneration of the cerebellar tract and peripheral nerves (Refsum disease, Russi-Levy disease, Dejerine-Sott's hypertrophic interstitial neuritis).
3. Diseases with a predominant lesion of the extrapyramidal system (hepatocerebral dystrophy, deforming muscular dystonia, Huntington's chorea, Minor 's family essential tremor, generalized tic).
4. Diseases with a predominant lesion of the pyramidal tract (familial spastic paralysis of Strumpel, amyotrophic lateral sclerosis).

2) Hereditary metabolic diseases occurring with damage to the nervous system.

1. Hereditary disorders of amino acid metabolism (phenylketonuria, histidinemia).
2. Hereditary disorders of lipid metabolism (amaurotic idiocy, Niemann-Pick disease, Gaucher disease, leukodystrophy).
3. Mucolipidosis (neurovisciral lipidosis, Derry's disease, fucosidosis, mannosidosis).
4. Hereditary disorders of carbohydrate metabolism (galactosemia, glycogenosis).

3) Hereditary connective tissue diseases.

1. Mucopolysaccharidoses.

2. Marfan's disease.
3. Chernogubov-Eles-Danlos syndrome.
4. Osteogenesis imperfecta.

4) **Phakomatoses** (Recklinghausen neurofibromatosis, Bourneville tuberous sclerosis, Sturge-Weber encephalotrigeminal angiomatosis , ataxia-telangiectasia, cerebroretinal angiomatosis Gipell-Lindau).

5) Hereditary neuromuscular diseases.

1. Progressive muscular dystrophies (lumbar-limb form of Erb-Roth, pseudohypertrophic form of Duchenne, late pseudohypertrophic form of Becker, distal forms, ophthalmoplegic myopathy).
2. Spinal and neural amyotrophies (Werdnig-Hoffmann spinal amyotrophy, Kugelberg- Welander spinal amyotrophy , neural amyotrophy).
3. Congenital non-progressive myopathies.
4. Hereditary neuromuscular diseases with myotonic syndrome (congenital Thomsen myotonia, Kurshmann-Batten-Steinert myotonic dystrophy).
5. Paroxysmal paralysis.
6. Myasthenia gravis.

Hereditary systemic degeneration of the nervous system.

This group includes diseases in which a hereditary degenerative process is localized mainly in certain and constant parts of the nervous system for each disease (cortico-muscular pathway, subcortical nuclei, etc.). The pathogenetic essence of many of these diseases remains unclear, but clear clinical differences allow in many cases to establish an accurate diagnosis. By the predominance of certain neurological symptoms, one can conditionally distinguish subgroups of systemic degeneration: with a predominant lesion of the cerebellum and its connections, combined damage to the cerebellar tract and peripheral nerves, with damage to the subcortical nuclei and cortico-muscular tract (pyramidal tract, anterior horn of the spinal cord).

Diseases with a predominant lesion of the cerebellum and its connections.

Friedreich's familial ataxia.

The mode of inheritance is autosomal recessive, although rare cases of autosomal dominant transmission have been described . Male persons get sick more often.

Pathomorphologically , degenerative changes are found in the posterior and lateral cords of the spinal cord: the pathways of deep sensitivity (especially the pathway of Gaulle), the spinal-cerebellar and, to a lesser extent, the pyramidal pathway are affected . Cerebellar atrophy is also detected.

Clinic . The disease begins most often at the age of 6-10 years. Ataxia appears, which has features of both sensitive and cerebellar ataxia. Initially, coordination disorders are found in the lower limbs: as the disease progresses, they spread to the trunk and upper limbs. Tendon reflexes are reduced. In the future, atrophy of the distal extremities occurs. Along with hypotension and hyporeflexia, pyramidal symptoms are detected. Nystagmus is a constant sign of the disease, chanted speech is often noted. In 15% of patients, intelligence is reduced.

Along with neurological disorders, deformities of the feet and spine are very often observed . The foot becomes "hollow", with a high arch, extension and main and flexion of the terminal phalanges ("foot Friedreich "). In the spine (especially in the thoracic region), kyphoscoliosis is found . From the side of the heart, signs of severe myocardial dystrophy are revealed .

Diagnosis. Muscle atrophy, foot deformities, decreased reflexes can resemble the symptoms of neural amyotrophy of Charcot-Marie .. However, in Charcot-Marie disease, such an expression does not suffer deep sensitivity , there is a significant decrease in muscle strength, and cerebellar and pyramidal symptoms are not typical. Friedreich's disease usually begins with the onset of ataxia, while Charcot-Marie's amyotrophy begins with muscle weakness and a disorder of superficial sensitivity. Friedreich's familial ataxia gradually progresses and, as a result, can lead to complete immobility of the patient.

Symptomatic **treatment** : massage, exercise therapy, orthopedic therapy, restorative agents.

Mari's familial ataxia.

The disease is inherited in an autosomal dominant manner. It begins at a later age (after 20 years). With Marie's ataxia, along with symptoms of cerebellar lesion, there are signs of pyramidal insufficiency, disorders of oculomotor functions, and often a decrease in vision due to pigmentary retinal degeneration and atrophy of the optic nerve. Decreased intelligence may develop.

Symptoms of sensitive ataxia are hardly detectable, which also distinguishes Marie's ataxia from Friedreich's disease. Pathomorphological examination reveals cerebellar atrophy and degenerative changes in the lateral cords of the spinal cord. Differentiate from other forms of hereditary ataxias.

Olivopontocerebellar atrophy.

Olivopontocerebellar atrophies are a group of diseases characterized by degeneration of neurons in the cerebellar cortex, nuclei of the pons and inferior olives. The cells of the spinal cord and basal ganglia are also affected. Diseases can manifest themselves at different ages. The main clinical symptom is cerebellar disorders, which are progressive in nature. Mental disorders are common and sometimes precede the onset of cerebellar disorders. They consist of changes in the emotional sphere (emotional dullness, lethargy, lack of initiative), a decrease in memory and a significant decrease in intelligence.

Diseases with a predominant lesion of the extrapyramidal system.

Hepatocerebral dystrophy (Wilson-Konovalov disease).

The type of inheritance is autosomal recessive. Males and females are affected with the same frequency.

Pathomorphologically , copper deposition is found in the subcortical nodes and in the liver. Copper deposits are also found in the cerebral cortex, cerebellum, spleen, kidneys, cornea, iris,
the lens of the eye. Disorders of copper metabolism are secondary and are primarily due to a deficiency of a protein substance - ceruloplasmin, which is involved in the transport of this element.

The clinical picture . The disease begins at the age of 10-15 years and is characterized by increasing muscle rigidity, various hyperkinesis (choreoform, athetoid, torsion), tremors of the limbs, head, trunk, dysarthria, mental changes. In some cases, epileptiform seizures are observed. Along with this , an increase and soreness of the liver, signs of liver failure are found. A specific symptom is a golden-green or greenish-brown ring on the iris — the Kaiser- Fleischer ring . In the blood, a low content of ceruloplasmin is determined, in the urine, an increased amount of copper. Hyperaminaciduria is also found.

According to the predominance of certain symptom complexes, 5 main forms of hepatocerebral dystrophy are distinguished : 1) abdominal, 2) rigid arrhythmic hyperkinetic, 3) trembling-rigid, 4) trembling, 5) extrapyramidal - cortical or pyramidal - hemiplegic.

The abdominal and rigid-arrhythmic-hyperkinetic form occurs mainly in children and is characterized by a rapid course and high mortality.

The tremulous-rigid form begins at the age of over 15 years, its course is slower. Basically, the trembling form occurs in adults and progresses very slowly, with remissions.

Hepatocerebral dystrophy should be differentiated from epidemic (lethargic) encephalitis, rheumatic chorea, deforming muscular dystonia. Wilson-Konovalov's disease differs from epidemic encephalitis by the presence of liver damage, changes in the iris of the eye, an increase in the level of copper in the urine and a decrease in the content of ceruloplasmin in the blood plasma. It is also important to take into account the family history - repeated cases of the disease among relatives. With rheumatic chorea , muscle rigidity, tremors are not noted, and signs of an ongoing rheumatic process are found.

Deforming muscular dystonia (torsion dystonia).

Torsion dystonia as a syndrome is observed in hepatocerebral dystrophy, epidemic encephalitis, and other lesions of the nuclei. She can
occur in the form of family cases, and both autosomal dominant and autosomal recessive type of disease inheritance are revealed.

Pathomorphologically , changes are found in the lenticular nuclei, red nuclei, subthalamic nuclei, and the dentate nucleus of the cerebellum.

Clinic. The disease usually begins at the age of 10-13, sometimes later. Typical symptoms are rotational spasms of the muscles of the trunk, proximal extremities, the head turns to the side and tilts back, the arms stretch out and go behind the back, the body turns around the vertical axis. The patient can freeze in such positions, and frequent hyperkinesis can lead to deformities.
spine. In sleep, hyperkinesis is significantly reduced or disappears altogether.

In addition to generalized hyperkinesis, there are forms of torsion dystonia with local spasms: spastic torticollis, writer's spasm.

Intellect does not suffer. Diagnosis is assisted by an analysis of the patient's pedigree.

The disease gradually progresses. For its treatment, drugs are prescribed that lower muscle tone and reduce hyperkinesis, vitamins of group B. Neurosurgical operations on the subcortical nuclei are successfully used - stereotaxic destruction of the globus pallidus,

ventrolateral nucleus of the thalamus.

Chorea of Huntington.

The type of inheritance is autosomal dominant, with a high penetrance of the mutant gene; family cases of the disease are very common. Males and females are affected with the same frequency.

Pathomorphologically , degenerative changes are found in the subcortical nodes, the cerebral cortex, and the expansion of the cerebral ventricles.

The pathogenesis of the disease is unclear. In the region of the substantia nigra, an increase in the iron content is found, in the erythrocytes the level of magnesium is increased. It is believed that the disease is based on gross violations of oxidative metabolism.

Clinic. The disease begins most often over the age of 25, but an earlier onset is possible. The clinical picture is dominated by two major signs: hyperkinetic syndrome and mental changes. Hyperkinesis are diverse, but they are mostly choreiform. Unlike rheumatic chorea, they are less rapid and patients can often arbitrarily delay individual violent movements.

Changes in the psyche consist in a gradually increasing weakening of attention, memory, and a decrease in intelligence, which gave rise to the appearance of another name for the disease - choreic dementia. Often, patients become irritable, agitated, but subsequently mental activity is inhibited.

Hyperkinesis and dementia build up gradually, patients can maintain the ability for self-care and intellectual activity for a long time . An analysis of the pedigree, the lack of data in favor of rheumatism, mental changes in patients, and a slower development of the disease help to differentiate the disease from rheumatic chorea .

For the treatment of Huntington's chorea, drugs are used that reduce muscle tone, sedatives and restorative agents. In recent years , attempts have been made for neurosurgical treatment.

Minor's familial essential tremor.

Inherited in an autosomal dominant manner The earliest sign is a small tremor of the hands, not always symmetrical, manifested only at the moment of awakening and voluntarily controlled

in a short time. Then a tremor of the arms, vocal cords, head, legs, trunk appears sequentially . Tremor increases with emotional stress. Unlike parkinsonism, the tremor is faster and with less amplitude; there is never a decrease in intelligence. The progression of the disease is expressed in the gradual generalization of the tremor and an increase in its amplitude.

The disease should be differentiated from hepatocerebral dystrophy, early forms of Huntington's chorea, the consequences of neuroinfections. The therapeutic

effect can be obtained from the use of vitamin B6, sedatives. In severe cases, adrenergic blockers are indicated.

Generalized tic (Tourette's syndrome).

The genetic aspects of the disease have not been clarified, since most of the cases described in the literature are sporadic.

The disease is manifested by multiple local tics. It usually begins at school age. First, twitching suddenly occurs in the muscles of the face, then in the muscles of the larynx and respiratory muscles. Patients suddenly make unusual sounds, words (sometimes of an obscene nature), cough, and have difficulty breathing. Later, when the muscles of the trunk and limbs are involved in the pathological process, one can observe impulsive lifting of the shoulders, squats, and stereotypical jumps. In patients with mental disorders may be in the form of weak will, lack of independence, the negative attitude to his condition. Haloperidol in combination with cyclodol has a therapeutic effect.

Diseases with a predominant lesion of the pyramidal ways.

Familial spastic paralysis of Strumpell.

Familial spastic palsy of Strumpell (familial spastic paraplegia) can be inherited both in an autosomal dominant and autosomal recessive manner. Family cases are quite common.

Pathomorphologically, degenerative changes in the pyramidal tract are found in the lateral and anterior cords of the spinal cord, primarily in the thoracic and lumbar regions. Gaul's bundles and cerebellar pathways can also be involved in the pathological process .

Clinic. The disease usually begins in early school age. Spastic paraplegia of the lower extremities gradually develops with a sharp increase in muscle tone, the presence of pathological reflexes and clonuses of the foot and patella. The feet acquire a varus (inwardly rotated) or equinovarus (drooping and inward rotation) setting . The lesion of the legs is symmetrical. In some cases, spastic phenomena extend to the upper limbs and even bulbar symptoms join. Intelligence usually does not suffer. Additional symptoms include ataxia, nystagmus, mild sensory disorders , and sometimes dementia. The disease progresses slowly.

Treatment . Prescribe drugs that reduce muscle tone, B vitamins , massage, exercise therapy, orthopedic measures.

Hereditary metabolic diseases occurring with damage to the nervous system.

Damage to the nervous system is observed in many metabolic diseases . The main pathogenetic mechanisms of neurological disorders are the toxic effect on the brain tissue of an excessive amount of abnormal metabolites formed as a result of an enzyme deficiency ; toxic effect of by-products of biochemical reactions; the absence of end products of the reaction due to a block at a certain stage of metabolism.

In about half of the cases, metabolic disease manifests itself at an early age and is characterized by delayed mental and motor development, seizures, impaired behavior in the form of apathy or excitement, and a decrease in height and body weight. With the later development of neurological disorders, children gradually lose

all acquired skills. Neurological disorders are progressively increasing. In many diseases there is a combined lesion of the nervous system, eyes, internal organs, and the musculoskeletal system.

Hereditary disorders of lipid metabolism.

Lipid metabolism is a multi-stage process in which dozens of enzymes are involved . Deficiency of any of them leads to severe disorders in the body. Conventionally, these diseases can be divided into 2 large groups: 1) storage diseases - intracellular lipoidosis, in which there is a predominant lesion of nerve cells and, for the second time, the pathological process involves pathways; 2) leukodystrophies, characterized by progressive decay of white matter due to impaired metabolism of lipids involved in the synthesis and metabolism of myelin.

Phakomatoses.

Phakomatoses are a group of diseases in which there is a combined lesion of the nervous system, skin, and very often internal organs. Phakoz means a spot. The characteristic symptoms of phakomatoses are pigmented, depigmented spots, angiomas of the skin vessels, fibromas, papillomas and a number of other skin changes. Neurological disorders are also very diverse: epileptiform seizures, hemiplegias, coordination disorders, extrapyramidal symptoms, autonomic disorders. Dementia is often observed, which can be either the result of mental retardation or a consequence of progressive dementia, and a combination of both is also possible.

The nervous system and the skin are formed from the same germ layer - ectodermal, therefore, in violation of embryonic development occur sochetannye neurocutaneous defeat .. phakomatoses referred to ektomezoderalnym dysplasia (blood vessels and internal organs derived from mesoderm), but in contrast to the vices of development observed in these diseases the progression of individual symptoms.

Recklinhausen's neurofibromatosis and Sturge-Weber's encephalotrigeminal angiomatosis are somewhat more common than other phakomatoses .

Recklinghausen neurofibromatosis.

The mode of inheritance is autosomal dominant with low penetrance. Men get sick more often, the disease occurs with a frequency of 1 in 5000 newborns.

Clinic. The first manifestations of the disease are usually observed in adolescence or adolescence and much less often from birth. Typical symptoms are tumors and age spots on the skin and along the nerve trunks. Tumors can be localized in the spinal, cranial nerves, roots of the spinal cord. There are also tumor growths in the brain (cerebral form). In some cases, there are neurofibromas located on the eyelids. Tumors are dense to the touch, usually painless, their number and size vary considerably.

Neurological symptoms depend on the location of the tumors. With the cerebral form, epileptiform seizures and mental changes are possible . With tumors of the optic and auditory nerves, visual and hearing impairments are noted . On the fundus, small nodules and plaques of pinkish-yellow and white color are often found , located in the retina and on the optic nerve head. The disease progresses slowly. Treatment - surgical removal of tumors.

Sturge-Weber encephalotrigeminal angiomatosis.

The type of inheritance is not precisely established; most likely, it is autosomal dominant with low penetrance. The incidence of the disease among the mentally retarded is 1 in 1000. In typical cases, there is a triad of symptoms: angioma of the skin, epileptiform seizures and glaucoma. Angioma is most often localized on the face, on the one hand, in the innervation zone of 1 or 2 branches of the trigeminal nerve. Bilateral angioma is less common.

Epileptiform seizures are caused by angiomatosis of the meninges , they are often focal, like motor Jackson. Often, after an attack, spastic hemiparesis develops on the side of the seizure contralateral to the angioma of the face. Glaucoma can be congenital or late. Usually it is unilateral and is localized on the side of the location of the angioma of the face.

Along with the classic forms of Sturge-Weber disease, there are so-called bisymptomatic forms, in which one of the symptoms of the triad absent. These options are observed more often than the classical ones, and are difficult to diagnose. The transition of bisymptomatic forms to trisymptomatic forms is possible, since not always all signs of the disease appear at the same time. Facial angioma is usually present from birth. Seizures occur most often in the 1st year of life, glaucoma appears at the age of 4-6 years. On the craniogram in patients older than 5 years, twisted double-contour shadows are found, more often in the occipital region. This symptom is very characteristic of Stulge-Weber disease .

Of other manifestations of the disease, dementia is very common, and its severity to some extent depends on the frequency and severity of epileptiform seizures.

The disease progresses gradually. Long non-convulsive intervals are possible. Angioma of the face increases in size, and sometimes decreases. For treatment, anticonvulsants are used, X-ray irradiation of the head in the area of localization of cerebral angioma. In some cases, the angioma of the meninges is surgically removed.

Hereditary neuromuscular diseases.

Hereditary neuromuscular diseases are a large group of diseases in which muscle tissue, peripheral nerves and often the anterior horns of the spinal cord are affected. The leading symptom in these diseases is muscle weakness, fatigue, often combined with hypotension and muscle atrophy, especially in the later stages of the development of the disease.

The most common neuromuscular diseases are progressive muscular dystrophies, spinal and neural amyotrophies; myotonia and periodic paralysis are less common. All of them are etiologically associated with gene mutations localized in the autosomes or the sex X chromosome; repeated cases of the disease in the family are not uncommon .

Progressive muscular dystrophies.

Progressive muscular dystrophies are a group of hereditary diseases in which the main pathomorphological changes are localized in muscle tissue.

Most forms of progressive muscular dystrophy begin in childhood. 35-40% of observations are of a family nature.

The lumbar-limb form of Erb-Roth. It is inherited in an autosomal recessive manner with some limited sex: boys get sick more often than girls.

Depending on the time of the onset of the first symptoms, 3 forms of the disease are distinguished: early, childhood and adolescent. The first symptom of the disease is weakness in the muscles of the pelvic girdle and proximal leg muscles . Difficulty running, climbing stairs. Later, the muscles of the trunk and arms are involved in the process ("ascending type"), less often the "descending type" is encountered. Muscle tone and strength decrease, and their atrophy develops. There may be pseudohypertrophies due to the growth of fat

and connective tissue. The gait of patients becomes waddling ("duck gait"), lumbar lordosis is expressed - the chest and abdomen protrude forward. The face is hypomimic ("sphinx face"), with protruding lips ("tapir lips"). The "wasp" waist is characteristic. When trying to get up from a lying position, the patient performs an action in several stages, connecting his hands to help (standing up with a "ladder"). The shoulder blades protrude, especially when the arms are abducted to the sides ("pterygoid" shoulder blades); when trying to lift the patient by the axillary areas, his shoulders freely rise up, and his head, as it were, falls between them (a symptom of "free shoulder girdles"). The disease progresses slowly, leading to complete immobility. Tendon reflexes are suppressed and disappear. Diffuse damage to muscle tissue extends to smooth muscles, heart muscle; revealed myocardial dystrophy, sluggish intestinal motility. With significant immobility, respiratory failure develops, also due to atrophy of the intercostal muscles. Congestion in the lungs is noted. In such cases, respiratory infections are very life-threatening.

The form of the disease, which begins with a lesion of the muscles of the pelvic girdle with an ascending type of spread of the pathological process, is also known in the literature under the name "progressive muscular dystrophy " Leiden-Moebius ".

Duchenne pseudohypertrophic form. Downstream, this is the most malignant form of muscular dystrophy. Its incidence is 27 cases per 100,000 newborns. The main type of inheritance is recessive, linked to the X chromosome. Boys are sick. The disease begins quite early (before the age of 3 years) and progresses rapidly. In addition to muscle weakness, decreased tone and reflexes, there are pseudohypertrophies, especially of the gastrocnemius muscles. In some cases, in the early stages of the disease, pseudohypertrophies are so pronounced that the patient has an athletic physique, combined with severe muscle weakness. Muscle atrophy leads to the formation of contractures. The muscle of the heart is often affected. In addition to damage to the neuromuscular system, diencephalic disorders can be observed - obesity like Itsenko-Cushing's syndrome, hyperhidrosis and other autonomic shifts. Often, patients lag behind in mental development. A biochemical study of blood plasma reveals a pronounced increase in the activity of muscle

enzymes and the level of amino acids. This distinguishes Duchenne muscular dystrophy from other forms of muscular dystrophy.

Shoulder-scapular-facial form of Landouzi-Dejerine. It is inherited in an autosomal dominant manner and is relatively benign, progressing slowly. The name of the disease indicates the predominant localization of the lesion: first of all, the muscles of the face and shoulder girdle suffer . Hypomimia develops, there is a weak severity of the nasolabial folds, the impossibility of tightly closing the eyes, as with bilateral damage to the facial nerve. Atrophy of the muscles of the shoulder girdle gradually increases , and in advanced cases, the muscles of the proximal parts of the lower extremities. Tendon reflexes persist for a long time.

Depending on the nature and sequence of the spread of the pathological process, the following forms of the disease are distinguished : face-shoulder - shoulder; face-scapularly - brachioperonial; face-shoulder-shoulder, buttock-femoral; face-to-shoulder - to shoulder
- femoral-peroneal; face-scapular - brachioperonial - gluteal - femoral.

Spinal and neural amyotrophies.

Spinal and neural amyotrophies represent a group of progressive neuromuscular diseases caused by primary damage to the spinal cord motor neurons and their analogs - the motor nuclei of the cranial nerves. If the body of the motor neuron is primarily affected, then they speak of spinal amyotrophies. Neural amyotrophies are the result of damage to axons (peripheral nerves). Muscle dysfunction is secondary due to their denervation.

Diseases with damage to the peripheral motor neuron lead to characteristic changes in the muscles. Muscle fibers are reduced in diameter, atrophied. Atrophied fibers are grouped and located next to intact, non-atrophied, or even compensatory hypertrophied. The transverse exhaustion of fibers persists for a long time. It disappears only at a late stage in the process. The nuclei in the atrophied fibers form clusters, so that in the later stages, only grouped nuclei are located in place of the atrophied fibers.

The pathogenesis of these diseases has not yet been studied.

Spinal styotrophy of Werdnig - Hoffmann.

It is inherited in an autosomal recessive manner. The disease is based on a steadily progressive degenerative process in the motor neurons of the spinal cord. The disease begins in the 1st year of life and in some cases is detected already at birth. Spontaneous motor activity of newborns is sharply weakened. Their posture resembles that of deeply premature babies: their legs are unbent, rotated outward and lie flat on the surface ("frog pose"). The arms are also extended, the shoulders are raised, there is no resistance to passive movements.

More often the disease manifests itself by the 5-8th month of life - the child begins to lag behind in motor development, loses the already acquired motor skills. In the affected muscles, tendon reflexes fade away, fibrillar and fascicular twitching in the fingers is observed - a small tremor. The face is hypomimic. The chest excursion is limited, resulting in frequent pneumonia.

Mental development is not affected. Atrophies reach a significant degree of severity, contractures are formed.

The disease progresses rapidly and is fatal within 1-3 years.

Spinal amyotrophy of Kugelberg - Welander.

It is inherited in an autosomal recessive manner. The first manifestations of the disease can be detected at an early age, but more often at the age of 8-10 years. Characterized by muscle atrophy of the proximal extremities and fascicular twitching. Muscle pseudohypertrophy is often noted,
lag in mental and physical development. The disease progresses slowly.

Neural amyotrophy (Charcot - Marie - Tooth - Hoffman disease).

The disease is inherited in an autosomal dominant manner; forms with an autosomal recessive and recessive type of inheritance linked to the X chromosome are less common . It begins more often at school age and later.

Pathomorphology. Degenerative changes occur initially in the anterior and posterior roots, motor neurons of the anterior horns, posterior cords of the spinal cord, peripheral nerves.

Clinic. Atrophy of the muscles of the distal extremities, most often the lower ones, develops . The extensors of the lower leg, small muscles of the foot, as well as the muscles that cause dorsiflexion of the foot are affected. As a result, the feet begin to sag, the patient walks, raising his legs high ("steppage"), a valgus position of the feet is formed (rotation of the feet outward). Tendon reflexes, primarily Achilles, quickly fade away . Some discrepancy between significant muscle atrophy and relatively satisfactory preservation of motor functions is characteristic .

Disorder of sensitivity is noted. These disorders are diverse: there is a decrease in superficial sensitivity in the distal parts of the type of "socks", "stockings", "gloves", paresthesias, spontaneous pain in the limb and tenderness on palpation along the nerve trunks can be observed . A disorder of deep sensitivity may also appear due to damage to the posterior cords of the spinal cord.

Deformation of the feet is often found: they become "hollow", with a high arch, extension of the main and flexion of the terminal phalanges of the fingers, especially the thumb (finger in the form of a trigger). The course of the disease is slowly progressive.

Treatment of progressive neuromuscular diseases is aimed at improving muscle trophism (glutamic acid, ATP, vit. E, glucose with insulin, cerebrolysin) and conduction of impulses along the nerve trunks (anticholinesterase drugs).

Shown means improving capillary beds and tissue oxygen supply (nicotinic acid), exercise therapy and massage.

Hereditary neuromuscular diseases with myotonic syndrome.

Myotonia refers to the inability of a muscle to relax quickly after a muscle contraction.

Diseases of this group include several genetically different forms of myotonia proper, myotonic dystrophy and some other nosological forms. In the pathogenesis of myotonic syndrome, a violation of myoneural conduction due to dysfunction of the presynaptic and postsynaptic membranes plays a role .

EMG when a long delay is determined myotonia relaxation muscles (potential consequences, myotonic delay), m. E. With okrativshayasya muscle relaxes.

Pathomorphological changes characteristic of myotonia are hypertrophy of muscle fibers, excessive branching of terminal nerve endings.

Congenital myotonia of Thomsen.

It is inherited in an autosomal dominant manner. It was first described by Thomsen on the basis of an analysis of his own disease and repeated cases of the disease among relatives (20 patients in four generations).

The disease can manifest itself as early as the first year of life. Patients have an athletic physique, they have well-contoured muscles of the shoulder girdle, especially the deltoid. Difficulty in relaxing the pharyngeal muscles causes impaired swallowing, which in young children is regarded as a neurogenic spasm. The myotonic reaction in the facial muscles increases with sucking and in the cold.

There are sudden muscle spasms, especially when trying to make a fast movement. Spasms intensify with cooling, internal tension. With sharp, quick movements, a kind of numbness develops , the patient is forced to stop. The gait becomes stumbling, the patient cannot immediately unclench the hand clenched against the fist. After repeating several movements, they are more free. The muscles are hypertrophied; when the muscles are tapped at the site of the impact, a "roller" or "fossa" is formed. The roller symptom can also be observed in the muscles of the tongue.

Congenital myotonia progresses extremely slowly. The psyche does not suffer.

Treatment is symptomatic. A potassium-restricted diet with a high calcium content is recommended . During the neonatal period, it is necessary to exclude cold food. Steroid hormones, ACTH, calcium preparations, quinine, ionogalvanization, faradization, massage, dosed physical exercises are used.

The autosomal recessive form, in addition to the nature of inheritance, is distinguished by a later onset (2-15 years), generalized muscle damage and a progressive course.

Kurshmann-Batten-Steinert myotonic dystrophy.

It is inherited in an autosomal dominant manner. The first symptoms that often arise in childhood are myotonic reactions, but later muscle atrophy is added to them, which is not typical for Thomsen's disease. Atrophy of the facial muscles is typical, which is found already in the early stages of the disease. Atrophy of the muscles of the trunk and limbs appears later. Vision is impaired due to the emerging cataract. Patients suffer from endocrine and vegetative - trophic disorders (sexual infantilism, baldness, seborrhea, early menopause).

Mental and emotional disorders are often noted - a feeling of fear, picky, irritability, progressive dementia.

The results of the study of the hormonal profile indicate the hypofunction of all endocrine glands, except for the pituitary gland.

The prognosis for myotonic dystrophy is poor.

Control questions:

1. What does the concept of hereditary diseases include?

2. What are the pathogenesis and classification of hereditary diseases from the point of view of modern knowledge?
3. What is the clinical commonality of hereditary diseases of the nervous system?

4. What are the clinical forms of myopathies?
5. What are the principles of myopathies treatment?
6. What are the clinical forms of hepatocerebral dystrophy?
7. What is the pathogenetic therapy for hepatocerebral dystrophy?
8. What are the clinical manifestations of Parkinson's disease?

9. Differential diagnosis of Parkinson's disease with post-encephalitic and atherosclerotic parkinsonism.

10. What are the treatments for parkinsonism?

11. What are the clinical manifestations and treatment of familial Friedreich's ataxia, Marie's ataxia and olivopontocerebellar atrophy?
12. What are the clinical manifestations and treatment of Wilson-Konovalov disease, torsion dystonia, Huntington's chorea, Minor family essential tremor, Tourette's syndrome?
13. What are the clinical manifestations and treatment of Schrumpell's familial spastic palsy?
14. What are the clinical manifestations and treatment of Recklinhausen neurofibromatosis , Sturge-Weber angiomatosis?
15. What are the clinical manifestations and treatment of progressive muscular dystrophies (lumbar-limb form of Erb-Roth, pseudohypertrophic form of Dyshenna, shoulder-scapular form of Landusi -Dejerine)?

16. What are the clinical manifestations and treatment of spinal and neural amyotrophies (Werdnig - Hoffmann, Kugelberg - Welander, Charcot - Marie - Tutta - Hoffmann)?
17. What are the clinical manifestations and treatment of congenital Thomsen myotonia and Kurshmann-Batten- Steinert myotonic dystrophy ?

Perinatal pathology.

The purpose of the lesson : to increase the student's level of knowledge about perinatal pathology and teach him how to use this knowledge in practice , namely, the ability to diagnose the disease and prescribe the appropriate treatment during the clinical analysis of patients and solving situational problems .

The student should know:

a) what does the concept of perinatal pathology mean;

b) its classification, including periods of perinatal pathology and clinical syndromes within each of these periods;

c) etiology, pathogenesis of diseases, treatment of clinical variants.

The student should be able to : independently, on the basis of complaints, anamnesis and examination of the neurological status, establish the diagnosis, the period of perinatal pathology, the main clinical syndrome, indicating the severity of the damage to the nervous system, and prescribe, if possible, etiopathogenetic treatment.

Perinatal lesions of the nervous system combine various pathological conditions caused by exposure of the fetus to harmful factors in the antenatal period, during childbirth and in the first days after birth.

Etiology

The cause of perinatal damage to the nervous system may be vnut - riutrobnaya hypoxia, which causes asphyxiation of the fetus and newborn, different maternal diseases, toxicosis of pregnancy, threatened abortion, immunological abnormalities in the system "mother - placenta-fetus" traumatic, toxic, radiological, metabolism, stress exposure , professional and household hazards, including taking medications during pregnancy, smoking, drinking alcohol.

The leading place in the perinatal pathology of the nervous system is occupied by asphyxia and intracranial birth trauma. This subdivision is arbitrary, since brain hypoxia, as a rule, is accompanied by punctate perivascular hemorrhages, and intracranial hemorrhages are often accompanied by oxygen deficiency of the brain. And yet, the pathogenesis and clinical manifestations of cerebral hypoxia and intracranial hemorrhage in newborns are different. Medical tactics and treatment-and-prophylactic measures for these conditions are also not identical, therefore, it is advisable to separate the presentation of cerebral hypoxia and intracranial hemorrhage in newborns.

Pathogenic factors can be diseases of the mother during pregnancy: toxicosis, infections, intoxication, metabolic disorders, immunopathological conditions and various obstetric pathology (narrow pelvis, prolonged or rapid labor, premature

discharge of water, premature placental abruption, entanglement with the umbilical cord, abnormal presentation of the fetus and etc.). Obstetric manipulations are also important, which can damage the nervous system of the fetus. In addition to these factors, some chemical substances and radioactive radiation play a certain role. The role of genetic factors has been established. There is a clear connection between the time the pathogenic effects on the developing organism and clinical manifestations: the earlier embryogenesis harmed - given the fetal brain, the more pronounced the effects of harmful influences. Various harmful influences in the perinatal period often lead to similar pathological conditions. In this regard, it is difficult to distinguish between the influence of individual factors.

It is important to keep in mind that intrapartum asphyxia and the so-called birth trauma often affect the nervous system of an abnormally developing fetus. In these cases, the harmful factors of the birth period are superimposed on dysembryogenesis. Even situations typical for normal labor activity may turn out to be "super strong **,** exceeding the adaptive capabilities of an abnormally developing fetus.

Pathogenesis . Oxygen deficiency leads to gross metabolic disturbances in the body of the fetus and newborn. There is an accumulation of acidic metabolic products, the electrolyte balance is disturbed , hypoglycemia develops, the activity of enzymes of aerobic and anaerobic respiration decreases . Under the influence of acidosis, the permeability of the vascular walls increases , cerebral circulation is impaired, ischemia, edema and swelling of the brain tissue develop. As a result of metabolic, hemodynamic and liquorodynamic disorders, the first cells are damaged. The degree of their damage depends on the severity and duration of intrauterine hypoxia and asphyxia at birth.

Classification of lesions of the nervous system in newborns and young children

The period of action of the pathological factor:

Prenatal:

a) embryonic;

b) early fetal (up to 28 weeks of gestation).

Perinatal:

a) antenatal (late fetal, after the 28th week); b) intranatal; c) neonatal.

Clinical form (by severity):

Mild form - in it is based on infringement gemolikvorodinamiki (distsirkulyatsii), reversible morphological and functional changes (adaptation).

Moderate form - it is based on edematous and hemorrhagic phenomena, congenital insufficiency of functional systems, dystrophic changes and focal gliosis of the brain.

Severe form - at the heart of cerebral edema, massive hemorrhages, profound metabolic disorders, a gross developmental defect, degenerative changes, atrophy, gliosis.

Disease periods:

Acute - from 7-10 days to 1 month.

Subacute: early **recovery** up to 4 months, late recovery - from 4 months to 12-24 months. **Defeat levels:**

The meninges and cerebrospinal fluid pathways, cerebral cortex, subcortical structures, brain stem, cerebellum, spinal cord, peripheral nerves, combined forms.

Clinical syndromes:

/. ***Acute period syndromes :***

1) comatose;

2) movement disorders;

3) general depression of the central nervous system (lethargy, weakness);

4) hypertensive-hydrocephalic (hypertensive, hydrocephalic);

5) convulsive;

6) syndrome of increased neuro-reflex excitability. **//. *Recovery period syndromes :***

1) cerebrasthenic;

2) vegetative-visceral;

3) movement disorders;

4) convulsive;

5) hydrocephalic;

6) delayed psychomotor, pre-speech development;

7) violation of predominantly static-motor functions;

eight) violation of the predominantly psyche.

Possible outcomes:

1. Recovery.
2. Delay in the pace of psychophysical and speech development.
3. Encephalopathy, manifested by mild symptoms:

a) scattered focal microsymptoms;

b) moderate intracranial hypertension;

c) astheno-neurotic syndrome, psychopathic and neurosis-like states.

4. Gross organic forms of lesions of the nervous system with severe motor, speech and mental disorders (oligophrenia), symptomatic epilepsy, progressive hydrocephalus, infantile cerebral palsy.

/. *The acute period of the disease is* characterized by the following syndromes: **Comatose syndrome is** considered as a manifestation of an extreme degree of depression with a complete lack of consciousness. It is dissociated with cerebral edema and rather extensive hemorrhages in the cranial cavity, which are usually the result of prolonged asphyxia and severe birth trauma against the background of pre- and perinatal complications. This is the most severe form of damage to the central nervous system. Muscle hypotension is clinically detected. Congenital reflexes, especially sucking and swallowing, are absent. The pupils are constricted, anisocoria is possible , the reaction of the pupils to light is weak or

absent. Often there are converging and diverging strabismus, failure of the facial nerve in the central type, nystagmus. Breathing is arrhythmic with frequent apnea. Cyanosis, bradycardia, deafness of heart sounds are noted. Arterial

the pressure is sharply reduced. Convulsions (repeated) with a predominance of the tonic component may occur .

Usually three (sometimes four) degrees of coma are distinguished. In degree I, there is no superficial (pain) sensitivity, skin reflexes are reduced. In the case of a II degree coma, there is a complete absence of sensitivity and skin reflexes, muscle atony, very low deep reflexes. A III degree coma is characterized by nystagmus, convergent strabismus, muscle atony, areflexia, dilated pupils with a complete lack of response to light. With edema of the brain stem , "stem" symptoms appear, which, with an increase in the pathological process, leads to a violation of vital functions and death.

It is possible to judge the level of damage to the brain stem in comatose syndrome by some clinical features. So, with damage to the upper sections of the trunk, vertical nystagmus, divergent strabismus, anisocoria, dilated pupils, decerebrational rigidity are observed .

In case of damage to the middle sections , gaze "floating" horizontally, horizontal spontaneous nystagmus, converging strabismus, miosis, decreased muscle tone can be observed .

Rotatory nystagmus, decreased blood pressure, respiratory arrhythmia, bulbar disorders are characteristic of lesions of the lower part of the brain stem.

Syndrome of general depression of the central nervous system is more often observed with moderate damage to the central nervous system . It manifests itself in hypodynamia, hyporeflexia, muscle hypotension.

The severity of the depression syndrome can range from mild lethargy to significant degrees of depression of consciousness. It is difficult to diagnose doubtfulness and sopor in newborns, so the pressure is sharply reduced for clinicians . Convulsions (repeated) with a predominance of the tonic component may occur .

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Hypertensive syndrome due to increased CSF PRESSURE - Niya. Clinical manifestations: with an upright position of the child, the large fontanelle is tense or even bulges, the saphenous veins of the head are dilated , frequent regurgitation, anxiety of the tongue, a sharp increase in muscle tone even with slight anxiety of the child, a spontaneous Moro reflex, “fin” installations of the hands and “heel” feet, increased deep reflexes, hyperesthesia, tremor of the chin and arms, violation of the sleep formula. The cerebrospinal fluid pressure exceeds 100 mm of water. Art.

Hydrocephalic, or hypertensive-hydrocephalic, syndrome

is a consequence of hypertensive syndrome and differs from the latter in the development of "dropsy of the brain", ie, the presence of an expansion of the cerebrospinal fluid pathways. The clinical picture in hydrocephalic syndrome, in comparison with hypertensive, is "enriched" with the following signs:

pathological increase in the size of the head and fontanelles, divergence of the seams of the skull, with percussion of the skull reveals the phenomenon of "cracked pot"; there are Graefe's syndrome, exophthalmia. With ophthalmoscopy, the phenomenon of stagnation in the fundus can be detected.

Convulsive syndrome is observed, as a rule, with moderate to severe degrees of perinatal encephalopathy.

In newborns, it can be provoked by hemorrhage into the cranial cavity, hypoglycemia, lack of vitamin B6, hypoxia, hypo- or hypernatremia, neuroinfection, hypomagnesemia, congenital lesion of the central nervous system. and metabolic disorders.

The syndrome of increased neuro-reflex excitability is characteristic of the acute period of perinatal encephalopathy and is clinically manifested in anxiety, tremors of the chin and limbs, violation of the formula, revitalization of deep reflexes and expansion of the zones of their evocation, sometimes flinching, in prolonged loud crying.

//. The recovery period of the perinatal pathology of the nervous system.

In the majority of children who have undergone mild and moderate forms of hypoxic traumatic damage to the central nervous system, a significant recovery of brain activity is observed .

In the recovery period, the following syndromes can be observed .

Cerebrastenic syndrome is observed mainly in children with a mild form of CNS lesion. Against the background of normal physical development, with insignificant environmental influences on the visual, auditory, and skin analyzers,

emotional lability and general motor restlessness are revealed . There is an increase in innate reflexes, anxious, superficial, insufficiently long sleep.

These manifestations can also be caused by somatic pathology: intestinal damage, ear pathology, violation of the diet, bathing, being in the fresh air.

The syndrome of vegetative-visceral **dysfunctions** indicates a violation of the diencephalic regulation of the vegetative-visceral reactions of the body. It is characterized by the appearance of vegetative-vascular spots, transient cyanosis, thermoregulation disorders, gastrointestinal dysfunctions with symptoms of pylorospasm, increased intestinal motility, rumbling, constipation, non-discharge of gases, regurgitation, and causeless vomiting. Lability of the cardiovascular and respiratory systems is also noted (arrhythmia, tachycardia, etc.).

The syndrome of motor disorders is manifested by an increase or decrease in motor activity, muscle hypo- or hypertension, mono- or hemiparesis, less often tetraparesis, and various hyperkinesis.

Muscle hypotension occurs when the cerebellum, anterior horns of the spinal cord, plexuses, or peripheral nerves are affected. At the same time, spontaneous activity, muscle tone, and deep reflexes are reduced . Trophic disorders can also be observed.

Muscular hypertension is observed when the pyramidal pathway in the brain and spinal cord is damaged, accompanied by an increase in deep reflexes, an expansion of the zones of their induction, the presence of a contralateral effect, clonus of the feet, and spontaneous Babinsky reflexes.

Congenital reflexes have a paradoxical dynamics: on the one hand, there is a suppression of the reflexes of sucking, swallowing, support, automated gait, crawling, and on the other hand, an increase and later delay in the reverse development of reflexes of oral automatism, Robinson, Babkin, labyrinthine tonic and cervical reflexes. tonic reflexes. Crossing of the legs in the lower third of the lower leg, disturbances in tone when raising the thighs, and support on toes are often observed.

Extrapyramidal insufficiency manifested dystonic disorders of muscle tone with a tendency to hypertension or hypotension, increased hyperkineses Athetoid or torsion character and frequent groundless protruding tongue, autonomic disorders.

Cerebellar lesions can be identified when making re - Benko purposeful movements, for example, when he picks up a toy or reaches for her. In this case, mimic hits, misses are observed . Trunk ataxia can be detected by observing how the child sits down, and at a later age - when the child gets up and moves around the playpen. Less often, speech disorders can be noted in the form of chanted pronunciation of words. Muscle hypotension and nystagmus are often noted .

Symptomatic epilepsy is characterized by seizures of - polymorphic nature (partial, generalized). In some cases, they stop as the disappearance of hemodynamic races - stroystv, cerebral edema phenomena mozga- intracranial hypertension. In the presence of ischemia, foci of necrosis, areas of hemorrhage, atrophic

processes in the brain, expansion of the ventricular system, convulsions become progressive in nature. Often, having stopped in the acute period, under the influence of various factors, seizures resume after 1.5-2-3 months. In some children, seizures

occur in the 2nd month of life. Their appearance is facilitated by the layering of additional exogenous factors.

Availability EEG individual and group sharp waves, a series of high - amplitude slow wave complexes "peak - slow wave", as well as paroxysmal outbreaks gipersinhronnyh biopotentials indicates **an epileptic** nature of the attack. In addition to convulsive seizures of various forms , children often have a lag in psychomotor and pre- speech development, paresis, paralysis of a spastic nature are often noted, and babbling is absent.

Hydrocephalic syndrome may manifest as communicating (outer) hydrocephalus divergence skull sutures, bulging fontanel change percussion skull sound, increasing head, the presence of continuous or intermittent symptoms Graefe, extension - Niemi cutaneous veins of the forehead and the nose bridge, the predominance of the cranium over the obverse, some prolapse eyeballs. There are also growing symptoms in the form of strabismus (converging and diverging), nystagmus, the appearance of pyramidal signs.

With internal hydrocephalus, often accompanied by microcephalus, hypertensive syndrome may be absent, additional research methods must be used - neurosonography, fundus examination , CT, MRI. Patients diagnosed with perinatal encephalopathy, as a rule, are observed by a neurologist and pediatrician at the age of up to one year, then this diagnosis must be specified.

Exit syndromes from perinatal encephalopathy. With a mild degree of damage to the central nervous system, as a result of correct treatment , recovery usually occurs. In the future, when attaching (layering)

exogenous factors, certain neurological manifestations can be noted , which must be regarded as violations of perinatal genesis, complicated by somatic or other pathology.

Asthenoneurotic syndrome is a consequence of the syndrome of increased neuro-reflex excitability and can manifest itself as affective-respiratory seizures and a number of behavioral disorders: increased irritability, moodiness, an increased level of claims in the form of excessive demands, speed of motor speech ("hesitation"), self-centered manifestation, excessively loud emotional crying, violation of the sleep formula (both falling asleep and inversion of sleep), negativism, autonomic disorders, some increase in the reflex background in a functional type, refusal to eat. It is important to differentiate the perinatal genesis of these manifestations from possible defects in upbringing, environmental influences.

Delay in the pace of psychophysical and speech development. Static-motor functions, analytical functions, pre- speech and speech development of the child may suffer . It is recommended to evaluate the child's psychomotor development according to the table proposed by Zhurbuy and Mastyukova

In violation of static-motor functions, the child begins to hold his head much later, roll over, sit, walk, stand. With a delay in mental development, the patient later fixes his gaze, does not immediately take the toy in his hands, worsens in the environment and performs the proposed tasks

A delay in pre-speech and speech development can manifest itself as a violation of the stages and rates of speech development (delay in the formation of

articulation, alalia, dysarthria, general underdevelopment of speech at various levels). There may be a combined delay in the rate of development with a predominance of impairment of any function.

The most unfavorable prognostic factor is mental retardation. exogenous factors, certain neurological manifestations can be noted , which must be regarded as violations of perinatal genesis, complicated by somatic or other pathology.

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The most unfavorable prognostic factor is mental retardation. learning difficulties that are not associated with an intellectual disability or conduct disorder (school maladjustment syndrome). Counting , writing, reading, perception of spatial relationships may be impaired . With age, abnormalities in abstract thinking appear and grow . Speech disorders are especially frequent, manifested by a delay in the development of speech, articulatory defects, slowness or, conversely, its explosiveness.

Additional research methods, for example, a skull X-ray , neurosonography, EEG, CT, NMR, can detect signs of compensated hydrocephalus, sometimes with signs of intracranial hypertension, manifested in the form of headache, fatigue and sleep disturbances.

The most unfavorable outcome of perinatal encephalopathy is infantile cerebral palsy.

CHILDREN'S CEREBRAL PARALYSIS

Cerebral palsy (CP) is characterized by pronounced persistent nutritional, paroxysmal, speech, behavioral disorders, mental retardation

(oligophrenia), and not only motor, but also postural mechanisms that are responsible for maintaining the posture are upset . A pathological postural stereotype is formed, which causes an increase in muscle tone and the appearance of pathological innervation.

Symptoms of the "risk" of cerebral palsy in the first half of life are high or low muscle tone, a delay in the development of statics and motor skills, prolonged retention of reflexes of spinal automatism, the presence of reflex-inhibiting positions.

Currently, there is no single generally accepted classification of cerebral palsy. Sometimes the classification is based on the topographic principle, according to which tetraparesis, hemiparesis, paraparesis or

the nature of the violation of muscle tone - spasticity, rigidity, dystonia. Practically more convenient is the classification that distinguishes spastic, hyperkinetic, atonic-astatic and mixed forms of cerebral palsy.

The spastic form is clinically expressed by a variety of syndromes. It includes spastic diplegia (Little's disease). With this form, tetraparesis takes place, however, paretic phenomena are most significantly presented in the legs , tone prevails in the flexors of the arms, extensors and adductor muscles of the legs. Due to spasticity , the proximal parts of the limbs are most affected. In the hands, movements in the wrist joints and fingers are possible , while movements in the shoulder and elbow joints are difficult. The patient's hands are usually in the position of abduction in the shoulder joints, in the position of flexion in the elbows and pronation in the hands. In the "lying" position, the patient's legs are usually extended. When trying to place the child, his legs are crossed with an emphasis on the socks. Deep reflexes are sharply increased, their zone is expanded, a persistent contralateral effect is noted . Clonuses of the feet and, less often, of the patella are observed . Pathological foot marks of flexor and extensor muscle groups are widely represented .

With age, stiffness in large joints gradually develops, and then limitation of movement due to contractures. Constant tension of the flexors of the feet leads to significant tension in the Achilles tendon. In the "sitting" position, the patient's back has the shape of an arc. Some children suffer from epileptic seizures, which significantly impair their speech and mental development.

Double hemiplegia (tetraplegia) is the most severe form of cerebral palsy. This severity is due to significant damage to psychomotor development and the presence of symptomatic epilepsy. Children, as a rule, are immobilized; in the “supine” position, their arms are brought to the body and bent at the elbow joints, or, conversely, extended and extended. It is noted

crossing the legs in the lower or upper third of the lower leg. Breeding of the thighs is severely impaired . Muscle tone is increased, contractures often develop in many joints, deep reflexes are high, with an extended reflexogenic zone, pathological foot signs are caused . Pseudobulbar symptoms are almost always present, making it difficult to chew and swallow pints.

Many children have symptomatic epilepsy, characterized by the presence of generalized and partial seizures.

The hemiplegic form is one-sided paralysis or paresis, in which the hand is predominantly affected. In most patients, hemiparesis manifests itself in the first

months of life. Muscle tone is increased on the affected side, and the patient's arm is bent at the elbow joint, brought to the body, and the leg is extended. Deep reflexes are high on both sides, but on the side of the affected limbs they are usually higher, pathological symptoms and clonuses are caused, and sometimes insufficiency of the VII and XII pairs of cranial nerves is revealed. Abdominal reflexes may be reduced on the affected side. In some patients , generalized seizures are observed, but more often the seizures are of the Jacksonian type. The gait of the patients is of a circumferential nature. Intellect, as a rule, suffers to a lesser extent than in other forms of cerebral palsy.

The hyperkinetic form is usually manifested by a change in muscle tone and the presence of involuntary movements in the form of athetosis, choreoathetosis, torsion dystonia and other hyperkinesis.

Involuntary movements can be detected as early as the middle of the first year of a child's life. Prior to this, the patient has anxiety, delayed motor skills, changes in muscle tone of a dystonic nature. By the end of the first year of life , various hyperkinesis begin to dominate in the clinical picture , which are not clearly expressed at rest . With any actions, emotional stress, excessive movements become distinct, capturing, mainly, the distal parts of the limbs, facial muscles. In the musculature of the trunk and neck, movements such as torsion dystonia are observed. Muscular tone changes, most often dystonia with a tendency to hypertension, less often hypotension can be noted . Deep reflexes often remain normal or elevated. The patient's movements are awkward, impulsive.

Hyperkinesis significantly inhibits the development of general motor skills in children. They either cannot sit and sit, or they start very late. The speech of the patients is significantly impaired, its general underdevelopment, dysarthria, tongue impediment are noted . The decline in intelligence can be minor to gross. Significant disorders of autonomic functions in the form of spontaneous hyperthermia, hyperhidrosis, tachycardia, and blood pressure lability are characteristic .

The atonic-astatic form is sometimes called the cerebellar, although the connections of the cerebellum with other parts of the central nervous system are disrupted, and the cerebellum itself, as a rule, does not suffer. The muscle tone of patients is reduced, there is some "looseness" in the joints, hyperextension in the elbows and knee joints. During movements, ataxia, dysmetria, and asynergy are clearly expressed. Sometimes there are tremors of the trunk and head, horizontal nystagmus. Deep reflexes can be increased. In some patients, cerebellar disorders are combined with pyramidal and extrapyramidal.

With a combined lesion of the pyramidal and extrapyramidal systems , a mixed form of infantile cerebral palsy is isolated. In patients in this case, spastic paralysis and hyperkinesis are observed.

Treatment of children with perinatal CNS injuries .

Treatment should be comprehensive, if possible, etiopathogenetic, gentle and continuous from the first days of life.

In the *acute period, the* treatment of newborns is carried out in the maternity hospital and is aimed at restoring the disturbed vital functions and stimulating the normal development of the nervous system. The main importance is attached to

the stimulation of the sequential development of motor and mental functions.

In the early recovery period , ***anticonvulsant, dehydration therapy is*** carried out **,** aimed at eliminating the neurological syndrome, normalizing muscle tone, myelination of the pyramidal and extrapyramidal pathways, and restoring the functional maturity of nerve cells.

When carrying out ***protoconvulsant therapy*** , drugs are used that minimally depress the respiratory center (seduxen, sodium oxybutyrate). And only if they do not help, they turn to anticonvulsants that strongly depress respiration (barbiturates, magnesium sulfate).

Generalized seizures are more common in young children than in adults. This is due to a number of morphological and functional features of their brain: increased permeability and the blood-brain barrier, instability of metabolic processes, a tendency of the brain to edema, insufficient synthesis of an inhibitory mediator - gamma-aminobutyric acid, etc.

Pharmacodynamics and pharmacological effects.

Anticonvulsants inhibit the increased activity of neurons involved in the formation of a convulsive reaction, and suppress the irradiation of excitation by disrupting synaptic transmission. First of all, there is a suppression of the cerebral cortex, reticular formation and hippocampus.

The mechanism of action of different drugs is different.

Seduxen activates specific benzodiazepine receptors located on the cell membrane of neurons in the limbic structures of the brain, reticular formation of the brain stem, hypothalamus, thalamic nuclei , spinal cord, i.e., brain structures related to the regulation of the human emotional sphere, the level of excitability of the central nervous system, the threshold of convulsive reactions and the flow of nerve impulses that maintain normal muscle tone. Excitation of these receptors leads to sensitization of functionally and morphologically related GABA receptors. The increased effect of GABA on its receptors is associated with the effects of benzodiazepines. It should be noted that benzodiazepines have an antihypoxic effect, which contributes to the survival of neurons in conditions of a low oxygen content in tissues.

Sodium oxybutyrate (GHB) is a derivative of gamma-aminobutyric acid (GABA), a mediator of inhibition in the central nervous system. Sodium oxybutyrate inhibits the release of excitatory mediators from presynaptic terminals (by affecting GABA-B receptors) and causes postsynaptic inhibition (by affecting GABA-A receptors). Sodium oxybutyrate is an antihypoxant, that is, it has the ability to maintain the function of the brain and other organs and tissues under conditions of hypoxia. In addition, the drug improves cerebral circulation by affecting the GABA-B presynaptic receptors.

Barbiturates (phenobarbital, barbamil), like benzodiazepines, have an allotopic effect on the GABA receptor complex, which includes both benzodiazepine and special barbiturate receptors. They increase the sensitivity of GABA receptors to the corresponding mediator. In addition, barbiturates protect the brain from circulatory hypoxia, since the drugs dilate the vessels in the ischemic and, conversely, narrow in the non-ischemic areas of the brain and

thereby also improve the hemoperfusion of the former; and also limit the activity of free oxygen radicals in ischemic areas. As a result of barbiturates prevent - stated failures cell membrane Ca^{+}-, K^{+}- pump development intracellular edema and increased intracranial pressure.

Magnesium sulfate. Magnesium ions in competition with calcium ions, by submitting - lyayut release of neurotransmitters, especially acetylcholine from presynaptic terminals in the central nervous system, and in neuromuscular synapses. This leads to the central and Peripheral - tion muscle relaxation.

The effect of all anticonvulsants is dose dependent. It can be of varying severity from sedative, analgesic, hypnotic to anticonvulsant and even narcotic. When using anticonvulsants in large doses, it is possible to suppress the subcortical structures of the patient's brain, which leads to disruption of the respiratory, cardiovascular and other body systems. Therefore, an individual selection of doses for patients is required.

Pharmacokinetics of anticonvulsants

Seduxen is administered intravenously, binding to blood plasma proteins is more than 90%. Due to its high affinity for plasma proteins, the drug is practically not dialyzed, and even mild hypoalbuminemia leads to a significant increase in the free fraction of medicinal substances in the crop, which, on the one hand, accelerates the elimination, and on the other, enhances its effects. The free fraction of the drug easily penetrates various tissues and liquids, including the cerebrospinal fluid. Biotransformation of the drug occurs in the liver. Excretion in the form of inactive metabolites (glucuronides) is carried out by the kidneys. In patients with liver damage, a mandatory dose adjustment of seduxen is required .

Sodium oxybutyrate is prescribed intravenously slowly, preferably by drip, intramuscular injection or by mouth. The drug easily penetrates the blood-brain barrier. The effect of intravenous administration develops in 10-15 minutes and lasts about 3 hours.

Magnesium sulfate, as an anticonvulsant, is administered intramuscularly (less often intravenously slowly, since there is a great danger of respiratory depression). The drug is excreted by the kidneys; in the process of excretion, diuresis increases.

Adverse Effects of Anticonvulsants

1. Respiratory depression, dysfunction of the cardiovascular system, inhibition of reflexes can occur when anticonvulsants are prescribed to eliminate seizures.
2. When using anticonvulsants for the prevention of convulsive syndrome, sedation, drowsiness, ataxia, nystagmus, dysarthria, hand tremors, skin rash, shifts in the blood count may appear , and an addictive phenomenon may develop when the same dose of the drug is prescribed. decrease in its effectiveness.

Dehydration therapy is usually performed using glycerin and diacarb. *Glycerin* has a moderate diuretic effect, its dose is 0.5-2.0 g / kg. *Diacarb,* inhibiting carbonic anhydrase in the kidneys and in other tissues, reduces the secretion of cerebrospinal fluid and increases diuresis. It is used at a dose of 30-60 mg / kg. The duration of treatment is individual and depends on the severity of the hypertensive syndrome. In between taking diacarb, you can prescribe a mixture of citral 5 ml per day, herbal cocktails (field horsetail, cornflower flowers, birch leaf, rose hips).

With the *syndrome of motor disorders with a predominance of muscle hypertension,* midocalm, skutamil-c, amizil and metamizil are prescribed. *Mydocalm* mainly has an inhibitory effect on the caudal part of the reticular formation, suppresses muscle rigidity, and has some anticonvulsant effect. It is prescribed orally at 0.0025-0.005 g once a day 30 minutes before the massage. *Skutamil-c* is a combined preparation consisting of an analgesic - paracetamol and isoprotane, capable of inhibiting tonic reflexes and eliminating spastic contractions of skeletal muscles, it is prescribed in a dose of 0.25 mg per day.

Amisil and metamizil central M-anticholinergics have moderate spasmolytic activity, normalize the activity of the parasympathetic nervous system.

With the syndrome of muscle hypotension, drugs are used that increase the release of acetylcholine and inhibit the activity of acetylcholinesterase, thereby enhancing the conduction of even a weak impulse. *Galantamine* (Nivalin) dissolves well in lipids and therefore easily penetrates into all tissues. 0.25% galantamine solution is prescribed at 0.1 ml per year of life, once a day subcutaneously or intramuscularly. *Neurin* and *oxazil* have a predominant effect in the neuromuscular synapses, having little effect on the central nervous system. Proserin is prescribed in the form of a 0.05% solution of 0.1 ml per year of life (but not more than 0.75 ml), subcutaneously or intramuscularly once a day. Proserin and oxazil are used by mouth, in a daily dose of 0.001 g per year of life, the frequency of administration is from 1 to 3 times a day.

Dibazol has the ability to restore the conduction of nerve impulses and has a vasodilating effect. Its dose ranges from 0.001 to 0.005 g once a day, depending on age. When *vegeto visceral, dysfunctions* administered chlorpromazine, neuro - leptik with pronounced sedation and Cerucalum which submitting - wish to set up predominantly pathological activity emetic center without affecting the other functions of the CNS. 0.5% solution *Cerucalum* appointed chayut intramuscularly at a daily dose of 0.5-1 mg / kg. *Aminazine* also has an antiemetic, hypothermic, antihistamine effect, it is prescribed at a dose of 0.5 mg / kg per day.

To improve metabolic processes in the brain, nootropic drugs are used that help to normalize cell metabolism and improve intellectual and mental processes. *Piracetam* (nootropil) at a dose of 40-200 mg / kg per day.

Solcoseril contains a wide range of natural nizkomoleku - -molecular peptides and amino acid derivatives. Numerous ex - experimentally and clinically proved that preparation as a universal activator of cell metabolism increases the proportion of aerobic glycolysis and oxidative phosphorylation in cells exposed

to hypoxia, enhances glucose transport across biological membranes, promotes recycling acidic metabolic products (lactate) in the conditions of acidosis causes blockade peroxidation lipid oxidation and activates antioxidant protection, that is, it has membrane and cytoprotective effects, has an angioprotective effect, increases the deformability of erythrocytes in conditions of impaired microcirculation, stimulates the proliferation of fibroblasts and the activity of microphages, increases collagen synthesis, has a systemic anti-ischemic effect. Solcoseryl is prescribed to children at a dose of 40-200 mg / kg per day orally for 30 days or parenterally at age-specific dosages. In order to prevent or eliminate adhesions, absorbable therapy is used, for example, the drug *lidase* , 8-32 units intramuscularly every other day. *Physiotherapeutic treatment is* prescribed in the acute period in the form of electrophoresis on the cervical spine with aminophylline, novocaine, nicotinic acid, magnesium. Subsequently, treatment is carried out with lidase, iodine, and with flaccid paralysis - with proserin or galantamine. High-frequency current, paraffinic pi ozokerite applications are also used .

Massage and physiotherapy exercises in the acute period are limited to the use of therapeutic and corrective measures with the help of orthopedic styling, and from 3-4 weeks acupressure is used. In patients with spastic paralysis, hydrokinetic therapy with the addition of pine extract or herbal sedative cocktails can be used . In the treatment of organic damage to the central nervous system, surgical orthopedic intervention is used. Sanatorium-resort treatment is shown .

Cerebral Palsy Treatment

Medical treatment of infantile cerebral palsy begins from the moment of diagnosis and continues up to 2-3 years, sometimes up to 5 years. At this stage, therapy is of a *compensatory, restorative nature* and is carried out in a comprehensive *manner* . Special attention is paid to physiotherapy exercises and massage, stimulation of mental, pre-speech and speech development, physiotherapy procedures. They use drugs that have a stimulating, relaxing, anticonvulsant, sedative effect.

In the treatment of cerebral palsy, great importance is attached to therapeutic exercises and massage. *Therapeutic gymnastics* aims to restore congenital motor reflexes, suppress pathological tonic and labyrinth reflexes, pathological synergies. Active exercises begin only after muscle relaxation, which is achieved by acupressure or relaxing massage. A number of exercises are carried out on a large ball, placing the child on it with his stomach or back (training of the vestibular apparatus). Patients develop the ability to crawl, the ability to move from the "lying" position to the "sitting" position, gripping a toy, getting up, walking. When performing the exercises, verbal guidance must be used. During the 2-3rd years of life, the child is brought up elements of stereognosis, praxis, perception of the spatial arrangement of surrounding objects.

From *physiotherapeutic procedures* used electrophoresis aminophylline and nicotinic acid, calcium, magnesium (in the form of a collar on Scherbakov, superimposed on the neck or chest parts of the spine), and the effect of pulsed current for the conventional circuit, a sinusoidal modulated current in the parietal-

temporal region of the head. Coniferous and "sea" baths with hydrokinesis therapy, application of ozokerite and paraffin have a beneficial effect on children .

To correct pathological settings, prevent and eliminate contractures, orthopedic treatment is used by applying permanent or removable plaster braces, special "mittens" and "boots". In some cases, they resort to operative elimination of pathology (achillo- and adductomy).

Drug treatment for cerebral palsy includes the use of drugs that provide a variety of effects on an already completed pathological process in the nervous system.

Stimulating drugs are piracetam (nootropil), pyriditol (encephabol), vitamins of group B. The course of treatment with them is from 1 to 1.5-2 months, 3-4 courses are carried out a year.

In spastic cerebral palsy, in order to suppress the pathological activity of the structures of the reticular formation of the brain stem and reduce muscle tone, midocalm, skutamil-c, amisil, metamizil are used.

Treatment is carried out in courses of 1-2 months 3-4 times a year. The drug is used for 20-30 minutes before the massage, when there is a maximum re - relaxation

In hyperkinetic forms of cerebral palsy, amidine, cyclodol, ridinol, L-DOPA are used, gradually increasing the daily dose by 2-3 times during the course of treatment.

When cerebellar form ICP accompanied usually myshech - hydrochloric hypotension, apply galanthamine (Nivalin), neostigmine methylsulfate, oksazil, dibasol, ATP. Treatment also held courses for 1-2 months *3* -4 times a year. If necessary, use anticonvulsants: valproate, phenobarbital, finlepsin. The indications for their appointment are the presence of changes in the EEG or clinically pronounced seizures.

To improve the hemodynamics of the nervous system is recommended reception cavinton being oxygenator and activator of cerebral meta - bolizma. The dose should be 1 / 4-1 / 2 tablets 3 times a day (1 tablet contains 5 mg of the drug). Actovegin is used to relieve the effects of hypoxia, stabilize cerebral circulation in a dose of 1 to 2-5 ml 1 time per day intramuscularly. Sermion increases vascular permeability, oxygen and glucose consumption, and motor impulse meditation. It is used in a dose of 10 to 30 mg per day.

The prognosis depends on the degree of brain damage. Gross brain defects lead to severe clinical manifestations of cerebral palsy, accompanied by hydro- or microcephaly, symptomatic epilepsy, a significant lag in psycho-speech development.

In the absence of severe pathology, long-term staged complex therapy can lead to a significant improvement in the patient's motor skills, independent movement, self-service, and in some patients - to the possibility of training in a secondary school program.

Control questions.

1. What is included in the concept of "perinatal pathology" and what are its causes?
2. What are the periods of perinatal pathology and syndromes within these periods?
3. What is the clinic of hypertensive- hydrocephalic syndrome, as a syndrome of the
acute period of perinatal pathology?
4. What is the clinic of the neuroreflex excitability syndrome as a syndrome of the
acute period of perinatal pathology?
5. What does the concept of D.TS.P. include?
6. What are the clinical options for D. Ts. P.?
7. Describe the clinical variants of D. Ts. P., which are a consequence of the defeat of the
pyramidal pathways?

8. What symptoms are characterized atonically - astatic form of D. Ts. P.?
9. What are the symptoms of the hyperkinetic form of D.TS.P.?
10. What does the concept of "obstetric paralysis" include , and what are its causes?
11. What are the symptoms of clinical variants of obstetric paralysis?

General principles of treatment and diagnosis of neurosurgical diseases.

Purpose of the lesson : Familiarization of students with the organization of work of the neurosurgical department using the example of a hospital, an operating unit and an outpatient appointment. Joint examination of patients.

The student should know:

1. General principles of conservative treatment.
2. General principles of neurosurgical operations.
3. Lumbar, suboccipital puncture.
4. Puncture of the brain and ventricles.
5. Drainage of the spinal subarachnoid space.
6. Concept of trephination and craniotomy (resection and osteoplastic). Laminectomy. General understanding of microsurgical, stereotaxic, endoscopic and endovascular technologies in neurosurgery.
7. Methods for stopping bleeding.

The student should be able to:

1. To carry out surgical treatment of wounds of the soft tissues of the head.
2. Provide transport immobilization for spinal cord injury.

H. Interpret the results of X-ray examination.
4. Perform lumbar puncture.

1. General principles of conservative treatment

The nature of the disease of the nervous system, the characteristics of its course and the severity of the patient's condition determine the therapeutic tactics for each patient. In this regard, as quickly as possible, it is necessary to resolve the issue of the advisability of treatment in a specialized hospital or the possibility of outpatient treatment. Patients with acute craniocerebral and spinal injuries, acute disorders of cerebral circulation, acute infectious lesions of the nervous system, status epilepticus require emergency hospitalization. If possible, they should be referred to intensive care units or neuroresuscitation units and treated until the condition stabilizes. Patients with impaired vital functions need urgent hospitalization. Patients with primary and secondary meningitis, encephalitis and other acute infections of the nervous system should be hospitalized in an infectious diseases hospital, with the exception of patients with tuberculous meningitis who need a room in the phthisiatric department, as well as patients with secondary purulent meningitis that have arisen against the background of purulent lesions of facial tissues, inner ear, oral cavity, sinuses. Such patients should be referred to the appropriate department (dental, otorhinolaryngology), where there is a possibility of operative sanitation of the primary focus of infection.
Patients with acute craniocerebral or spinal trauma, as well as suspected sub- or epidural chronic hematoma, should be referred to the neurosurgical department for follow-up and, if necessary , neurosurgical treatment. The neurosurgical department hospitalizes patients with spontaneous subarachnoid hemorrhages, intracranial hematomas and cerebellar hemorrhages. Considering that at the prehospital stage, as well as upon admission to the hospital, it is not always possible to accurately diagnose these conditions, the indicated contingent of patients can be hospitalized in neurological or intensive care units of

a multidisciplinary hospital with subsequent transfer to a neurosurgical hospital. ... Patients are sent to the hospital, who, in order to clarify the diagnosis, must be constantly monitored and for a comprehensive laboratory and instrumental examination. In some cases, hospitalization may be due to social reasons , difficulties in carrying out treatment at home, the inability to provide full care.

Indications for hospitalization in terminal states of patients with deep depression of consciousness and vital functions, the presence of severe incurable diseases (late stages of oncological diseases with multiple metastases, cancer intoxication; severe dementia) are limited.

Treatment of a patient in a hospital should be carried out on the basis of a full and comprehensive instrumental and laboratory examination, the volume of which is determined by the nature of the disease and the severity of the patient's condition. The presence of concomitant diseases, complications of the main pathological process requires the participation of doctors of related specialties.

It is necessary to take into account the high risk of developing bedsores, pneumonia, urinary tract infections in patients who are in a supine position for a long time . In this regard, a regular toilet of the skin, the use of anti-decubitus mattresses, passive and active breathing exercises, control of urination and defecation are necessary . It is important to prevent the development of deep vein thrombosis of the legs in long-term patients, for this purpose pneumomassage of the lower extremities can be used, and subsequently, when the patient is transferred to an upright position, tight bandaging of the legs with an elastic bandage. The most important aspect of caring for a seriously unconscious patient with swallowing disorders is proper feeding. Nutrient solutions can be administered parenterally; in addition, there are complexes for administration through a nazagastric tube, including nutrients, multivitamins, enzyme supplements.

Rehabilitation measures aimed at restoring impaired motor functions should be started as early as possible . So, starting from the first days of the acute period of stroke, it is necessary to start treatment with a position, giving the affected limbs a position that prevents the formation of contractures. As the condition stabilizes, with the normalization of the central hemodynamic parameters, therapeutic massage, passive and then active gymnastics are included in the complex of therapeutic measures. Electrical stimulation of paretic muscles, therapeutic measures based on the use of biofeedback can be used . Physiotherapy (electrophoresis of proserin, calcium preparations) and reflexology have a certain effect, mainly with flaccid paresis.

After the end of the course of treatment in a neurosurgical hospital, many patients need to continue treatment in the conditions of rehabilitation and rehabilitation departments. First of all, this applies to persons with movement disorders and speech disorders. Treatment of such patients should include (in addition to medication) the widespread use of remedial gymnastics, including the use of exercise equipment, massage, physiotherapy. Subsequently, in the presence of persistent motor deficits, orthopedic correction may be required.
existing violations. Treatment of speech disorders is carried out in conjunction with a speech therapist. Treatment can also be carried out in a

spa setting. Sanatoriums in central Russia and the foothills of the Caucasus are preferred . It is undesirable to refer patients to places with an excessively hot and humid climate, especially during the hot season.

Carrying out drug therapy requires detailed consideration of the characteristics of the pharmacodynamics of the drug, on the one hand, and the sensitivity of the patient's body to drugs, on the other. It should be remembered that the simultaneous use of drugs from different pharmacological groups can enhance their effect (for example, the use of sedatives and clonidine can lead to a significant decrease in blood pressure, the use of chlorpromazine increases the analgesic effect of analgin), which makes it possible to achieve an optimal therapeutic effect when using low doses of drugs. At the same time , undesirable side effects may develop with the simultaneous administration of a number of drugs, for example, the occurrence of confusion with the simultaneous use of carbamazepine and amitriptyline. It should be borne in mind that in patients with impaired

functions of the liver, kidneys, in elderly people, the catabolism of xenobiotics slows down , their excretion from the body is impaired. The consequence of this is an increase in the concentration of drugs in the blood and an increase in the risk of overdose, which requires their use in lower doses.

In the drug treatment of neurological patients, the problem of mono- or polytherapy is very important . In most cases, the simultaneous use of drugs of the same or similar pharmacological groups (for example, two or more antiplatelet agents, analgesics, vasodilators) is not justified . In this case , the load on the liver and kidneys increases, there is a threat of allergization of the body, while the therapeutic effectiveness increases to a lesser extent than if the therapy was carried out with one of the drugs, but in large doses or in combination with drugs from other groups that potentiate its effect. In addition, in such cases, control over the effectiveness of the treatment is significantly complicated. At the same time, in a number of cases, in particular in epilepsy, if there is no effect from monotherapy, clinical efficacy increases with the combined use of two or more drugs of different action.

The quality and effectiveness of the drug treatment carried out largely depends on the form of administration of the drug into the body. In acute situations (stroke, traumatic brain injury, infectious damage to the brain and its membranes), the maximum effect is achieved with parenteral, namely intravenous, high doses of drugs. This contributes to the rapid achievement of the maximum concentration of the drug in the blood, cerebrospinal fluid, brain tissue. The feasibility of endolumbar administration of drugs in most cases is questionable. In acute stages of diseases (migraine attack, pain syndromes associated with degenerative lesions of the spine), oral administration of liquid forms of drugs or their rectal (after a cleansing enema) administration is possible.

The latter method has a significant advantage in patients with swallowing disorders, repeated vomiting, and gastric ulcer. In the treatment of chronic diseases, if it is necessary to take drugs for a long time, over several months or years, it is

advisable to use prolonged dosage forms, for example, slow-release tablet forms of trental, nifedipine.

Considering that the development of many neurological diseases is accompanied by the formation of asthenoneurotic, asthenodepressive reactions, sleep disorders, emotional disorders, a complex of therapeutic measures requires include sedatives or small tranquilizers (valerian, bromides, motherwort, phenazepam, other benzodiazepine derivatives), antidepressants (amitriptyline, anafranil), biogenic stimulants (ginseng, eleutherococcus, lemongrass). The use of hypnotics (nitrazepam, barbiturates) is often necessary . At the same time, in addition to pharmacological therapy, the role of a doctor acquires exceptional importance in these situations, whose task is to conduct explanatory conversations with the patient, maintain in him a sense of optimism, self-confidence, and form the correct attitude towards his condition. The existence of a strong psychological contact between the doctor and the patient is an important part of the treatment process.

Patients who have undergone a severe neurosurgical disease need rational employment, many of them are forced to stop working , need outside care. It is often necessary to revise the household stereotypes that developed before the disease (features of the daily regimen, the nature of the diet, the presence of bad habits), which, in combination with preventive drug therapy, underlies the secondary prevention of neurological diseases.

2. General principles of surgical treatment

2.1. Operations on the skull and brain

Indications for brain surgery can be various diseases: tumors, cerebral aneurysms, intracerebral hematomas, traumatic injuries of the skull and brain, deformities, some parasitic and inflammatory diseases, and a number of others.

Operations on the skull and brain differ depending on the nature of the access and the degree of radicality of the surgical intervention. In addition, they can be diagnostic and therapeutic.

2.1.1. Surgical approaches

Milling holes. Small holes in the skull, usually 1.5-2 cm in diameter, are made mainly for diagnostic studies: detection of intracranial hematoma in traumatic brain injury, for puncture of the brain in order to obtain a fragment of pathological tissue for histological examination or for puncture of the ventricles of the brain.

Milling holes are placed at typical locations through small skin incisions. To perform this operation, various trepans are used, the most common are mechanical, electrical and pneumatic trepans. The cutters used to create holes in the skull differ in their design and size. In some cases, the so-called crown cutters are used, with which a circle is cut out in the bones of the skull, which, after the completion of the operation, can be put in place.

Craniotomy (craniotomy). Distinguish resection of bone and plastic craniotomy.

Resection trepanation - consists in removing a section of the skull. For this purpose, a milling hole is placed, which is then expanded with the help of bone nippers to the desired size. Resection trepanation is usually performed for the purpose of decompression of the brain in traumatic brain injury, if intracranial

pressure is sharply increased, or in case of a multiple fracture that does not allow maintaining the integrity of the bone. In addition, resection trepanation is used in operations on the posterior cranial fossa. Bone resection in this area is technically easier than
osteoplastic trepanation. At the same time, a powerful layer of the occipital muscles reliably protects the structures of the posterior cranial fossa from possible damage, and the preservation of bone in these cases is not as important as in operations on the cerebral hemispheres during supratentorial processes.

Osteoplastic trepanation consists in the formation of a bone flap of the desired configuration and size, which, after the completion of the operation, is placed in place and fixed with sutures. The place of craniotomy is determined by the localization of the pathological process. When performing trepanation, the surgeon must be well oriented in the relationship between the skull and the main anatomical structures of the brain, primarily such as the lateral (Sylvian) groove separating the temporal lobe from the frontal, central (Roland) groove, central gyri, etc.

There are various methods and schemes for transferring the projection of these formations to the skull. One of the schemes used to date was proposed by Krenlein. To determine the projection of the Sylvian groove and the Roland groove, he offers the following method (Fig. 1). Initially, a baseline is drawn through the internal auditory canal and the lower edge of the orbit, then a second line is drawn through the upper edge of the orbit , parallel to the first. From the middle of the zygomatic bone, a perpendicular is restored, the point of intersection of which with the upper horizontal line is the lower point of the Roland groove, to determine the direction of which its upper point is determined . It corresponds to the intersection of the perpendicular passing through the mastoid process with the convexital surface of the skull. The bisector of the angle formed by the projection of the Roland groove and the upper horizontal line defines the position of the Sylvian groove.

Depending on the localization of the process (tumor, hematoma, abscess, etc.), in connection with which trepanation is carried out, skin incisions are made in the corresponding area. The most commonly used are horseshoe-shaped incisions facing the base of the skull. Straight cuts are also used. In neurosurgical operations for cosmetic purposes, mainly incisions located within the scalp are used.

With incisions in the frontotemporal region, it is desirable to preserve the main trunks of the superficial temporal artery, located anterior to the ear.

With the help of a trephine, several milling holes (usually 4-5) are placed around the perimeter of the bone graft being formed . It is important that the cutter holes are located at some distance from the skin incision to prevent the formation of rough Cicatricial adhesions. With the help of a special guide , a wire saw (Jigli) is passed under the bone between adjacent milling holes and the bone is sawn around the entire perimeter. To avoid the failure of the bone flap, outwards, the bone is cut at an angle by bevel

In the area of the periosteal-muscular "leg" of the flap, the bone is only sawn off and then broken when the bone is lifted with the help of special bone lifters.

Recently, special pneumatic and electric traps are increasingly being used, which allow cutting out bone grafts of any size and configuration from one milling hole. A special paw at the end of the craniotome detaches the dura mater from the bone as it moves. The bone is cut with a thin, fast rotating cutter.

The dura mater incisions can be of different configurations, depending on the size and size of the pathological process to which access is planned. Horseshoe, cruciform and patchwork incisions are used.

At the end of the operation, if the state of the brain permits, it is necessary to suture the dura mater tightly with interrupted or continuous sutures , if possible .

In cases where after the operation there is a defect in the dura mater, it must be closed. For this purpose, a specially processed cadaveric dura mater, fascia lata of the thigh, aponeurosis or periosteum can be used.

In order to stop bleeding from the bone, the cut site and the inner surface of the bone flap are treated with surgical wax.

To prevent epidural postoperative hematomas, the membrane is sutured to the periosteum in several places along the perimeter of the bone opening.

To reduce the risk of postoperative blood accumulation in the surgical wound, the bone flap is separated from the periosteum and muscles throughout the entire length and during the operation it is stored in isotonic sodium chloride solution.At the end of the operation, the bone flap is placed in place and fixed with bone sutures. For this purpose, a thin bur is used to put holes in the bone on both sides of the cut, through which a special wire or strong ligatures are passed .

In modern neurosurgery, **extensive basal approaches** with resection of the bones of the skull base are increasingly used . Such approaches are necessary to remove tumors located near the median, most distant from the surface of the brain structures (tumors of the parastem localization, tumors of the clivus and cavernous sinus, basal aneurysms, etc.). Wide resection of bone structures of the base of the skull, including the roof and lateral wall of the orbit, the wings of the sphenoid bone, the pyramid of the temporal bone and other bone formations, allows you to approach the most deeply located pathological foci with minimal traction of the brain.

For resection of bone structures near large vessels and cranial nerves, high-speed drills and special diamond-coated cutters are used.

In some cases, to approach deep, medially located tumors, **facial approaches** are used **, approaches through the paranasal sinuses:** wedge-shaped, maxillary (maxillary) and through the mouth.

Transnasal -transphenoidal access to tumors developing in the Turkish saddle cavity, primarily to pituitary tumors, is especially widespread .

2.1.2. Brain surgery technique

The exceptional functional significance of the entire brain and its individual structures makes it necessary to use such a surgical technique that would make it possible to perform operations with minimal risk to the patient. This task becomes feasible using microsurgical techniques.

The position of the patient. To perform operations on individual structures of the brain, various positions of the patient on the operating table are used: on the

back, sometimes with the head turned to the side, on the side, in some cases, the patient is operated in a prone position with the head lowered and bent, in operations on the posterior cranial fossa widely the patient's sitting position is used.

In each individual case, the surgeon determines the optimal position of the patient to expose certain areas of the brain. When choosing the position of the patient, it is necessary to take into account the possible change in hemodynamics (primarily venous circulation). If the patient is in a sitting position during the operation, then the pressure in the venous sinuses of the head decreases sharply and can be negative. This phenomenon explains the possible development of air embolism - the ingress of air into damaged large venous collectors and its accumulation in the chambers of the heart, while there is a danger of cessation of cardiac activity. This complication must be remembered when the patient is operated on in a sitting position, and a number of preventive measures must be taken. The easiest method to recognize a wound
large veins, is compression of the jugular veins in the neck. If during the operation the patient's head is lowered down or, due to its sharp bending, the veins are compressed, the venous pressure can increase sharply, which leads to an increase in the volume of the brain, its bulging into the wound, and excessive bleeding. Continuation of the operation in this case is fraught with serious complications, and the patient's position must be changed.

Microsurgical technique. The main components of microsurgery are the use of special binocular loupes and operating microscopes. At the present time in neurosurgical operations used operating microscopes, which have the following features: mobility, allowing free peremetat microscope in different required the surgeon to directions; wide-ranging magnification, good illumination of the operating field, the presence of additional eyepieces for the assistant. A miniature television camera, which can be equipped with a microscope, allows an assistant, an operating nurse and other persons involved in the operation (anesthesiologist, neurophysiologist, etc.) to see the operating field on the screen. Television and photo attachments are required to obtain the transaction documentation.

The use of a microscope makes it possible to carry out an operation in a narrow deep wound with minimal displacement of the brain. Additional opportunities when examining deeply located parts of the brain appear when moving the operating table and giving the patient's head different positions. For this purpose, special tables and head restraints are used to fix the patient's head.

To perform the operation under magnification, a variety of microsurgical instruments are used: tweezers, scissors, dissectors, miniature clamps for clamping blood vessels, suture material.

Brain retractors. Operations on the brain, especially on its deep structures, require displacement of the brain (lifting, moving away), often for a long period of time. To achieve this, special automatic retractors are used, which can hold the brain in various positions necessary for the surgeon. These retractors are attached either to the edge of the trepanation hole, or to special frames that are fixed to the table and the patient's head. When using spatulas, the

surgeon should always remember that a sharp displacement and compression of the brain leads to the so-called retraction ischemia, damage to the brain tissue and its vessels (especially veins) and the risk of intracerebral bleeding in the postoperative period. Traction of the brain should be minimal, the position of the spatulas must be constantly changed during the operation.

Protecting the brain from drying out. For this purpose, the exposed surface of the brain is covered with quilted jackets moistened with isotonic sodium chloride solution. During long-term operations, quilted jackets must be changed and moistened so that they do not dry out to the cerebral cortex.

Methods for stopping bleeding. The brain, one of the most vascularized organs, is permeated with a mass of vessels. Stopping bleeding from the brain tissue is distinguished by significant specificity, since in a narrow and deep wound, vascular ligation, which is widely accepted in general surgery, is practically impossible. In some cases, special miniature clips-clips are used to stop bleeding from large vessels of the brain . However, the most common way to stop bleeding is coagulation (mono- and bipolar). Point bipolar coagulation is of particular importance , in which the current circulates only between the tips of the forceps and there is no heating of adjacent structures, which is extremely important in operations on the brain, especially on its deeply located structures.

To stop parenchymal bleeding from brain tissue, a special hemostatic fibrin sponge, hemostatic gauze, biological glue (tissucol) and a number of other drugs that cause blood coagulation are widely used
and the formation of a strong blood clot. Along with these means , wound washing with isotonic sodium chloride solution and tampons moistened with hydrogen peroxide are widely used.

Bleeding from damaged large vessels and venous sinuses can also be stopped by tamponade with a piece of flexed muscle.

Intraoperative diagnostic methods. For orientation in the operating wound and the detection of formations located deep in the brain, it is often necessary to use special techniques and additional diagnostic methods.

Puncture of the brain. The most commonly used method that allows the surgeon to detect a pathological formation located deep in the brain (tumor, abscess, hematoma) is puncture. For this, special cerebral cannulas with a blunt end and a lateral opening are used. By the change in resistance that the surgeon experiences when immersing the cannula into the brain, he can determine the edge of the tumor, the wall of the abscess, and cysts. The flow of cystic fluid, blood, and pus through the cannula gives the surgeon additional information and allows him to determine the further plan of the operation.

To detect deeply located tumors during the operation , special *radiosttillation probes* can be used to determine the areas of accumulation of the radioactive isotope. For this purpose, before the operation, the patient is injected intravenously with an isotope preparation (radioactive phosphorus, mercury), which selectively accumulates in the tumor. A change in the readings of the radio scintillation counter and the corresponding sound indication indicate that the probe has entered the tumor tissue .

Ultrasound location of the brain. Recently, ultrasound location has been used to detect formations located deep in the brain. After craniotomy , an ultrasound sensor is installed on the unopened dura mater or the exposed surface of the brain , changing the position of which it is possible to obtain on the screen an image of deep structures (ventricles, cerebral crescent) and neoplasms located in the thickness of the brain (tumor, hematoma, abscess).

Surgical aspirators. One of the characteristic features of brain surgery is that the surgeon must constantly remove cerebrospinal fluid, which flows in large quantities from the cerebral ventricles and subarachnoid spaces. The use of special aspirators greatly simplifies this task. The suction devices used by the neurosurgeon are at the same time an important tool with which tissue dissection can be performed. In order not to cause damage to the brain, not to injure blood vessels, it is necessary that the tip of the suction is rounded, without sharp edges. Depending on the situation, suction units of different diameters and configurations are used.

Ultrasonic suction. An important invention of recent years is ultrasonic suction, with the help of which it is possible to simultaneously destroy pathological tissue (tumor) and aspirate it, as well as remove cerebrospinal fluid.

For dissection of brain tissue, stopping bleeding, evaporation of pathological tissue during neurosurgical operations, laser installations are used, combined with an operating microscope (argon, neodymium), etc.

2.1.3. *Types of neurosurgical operations*

Depending on the purpose, brain surgery can be conditionally divided into **radical and palliative interventions** . The goal of radical operations
consists in the removal of pathological formations (hematoma, abscess, tumor), restoration of normal anatomical relationships (reconstruction) with traumatic skull fractures, deformities, etc. The concept of "radical intervention" is used with some caveat. It determines the purpose of the operation, but its result does not always correspond to the task at hand (for example, in case of a brain tumor, it is often not possible to achieve its radical removal).

Palliative operations are not aimed at relieving the patient of the disease itself, but are aimed at alleviating the patient's condition. An example of palliative surgery is the creation of new pathways for the outflow of cerebrospinal fluid from the ventricles of the brain in inoperable tumors leading to occlusion of the cerebrospinal fluid pathways and impaired CSF circulation (ventriculo-atrial or ventriculo-peritoneal shunting).

Depending on the urgency of the operation, neurosurgical interventions are divided into **planned and emergency (urgent)** . Emergency operations are usually done for health reasons. The need for urgent operations arises with traumatic hematomas, with acute occlusion of the cerebrospinal fluid, when the patient develops symptoms of dislocation of the brain and compression of its stem sections in the large occipital or tentorial foramen.

Stereotaxic operations. Along with open operations in the brain, which require execution craniotomy skull used and so -called stereotactic (from the Greek. Stegeos - volume, spatial and Greek taxis. Location) interventions performed through a small burr hole.

The essence of stereotaxic operations is that various instruments are inserted into precisely defined parts of the brain (usually deeply located): electrodes for destruction and stimulation of brain structures, cannulas for cryodestruction, instruments for biopsy or destruction of deeply located tumors.

These instruments are introduced into the brain using special stereotaxic devices fixed on the patient's head. These devices have devices that allow you to spatially orient the instrument introduced into the brain and determine the depth of its immersion.

To determine the coordinates of targets (subcortical ganglia, nuclei of the thalamus, midbrain and other deeply located structures of the brain, as well as deeply located tumors, hematomas, abscesses, etc.), special stereotaxic atlases and data from computed tomography and magnetic resonance studies are used.

Modern stereotaxic devices make it possible to insert the necessary instruments into the brain structures with an accuracy of 1 mm.

Stereotactic operations have found a particularly widespread use in functional neurosurgery (treatment of hyperkinesis, pain syndromes, epilepsy, etc.).

Method of spatial orientation in time operations on the brain the brain in recently it has become possible without the use of stereotaxic devices.

In this case, on the display screen, the surgeon can reproduce any sections of the brain obtained earlier using computed and magnetic resonance imaging, and determine the position of the instruments he uses (tweezers, suction, etc.) on them, which is achieved by locating these instruments using infrared or other rays.

Endoscopic operations. As in other branches of surgery, endoscopic interventions have been widely used in neurosurgery in recent years. In general, these operations are performed on the ventricles of the brain. Rigid and flexible endoscopes are used, equipped with instruments for tissue sampling, destruction and stopping bleeding (using coagulation or laser exposure).

The introduction of endoscopes can be carried out using stereotaxic devices.

Radiosurgical interventions. The principle of spatial orientation, which is the basis of stereotaxic operations, is also used for strictly focused radiation exposure to the brain.

For this purpose, special radiosurgical installations are used, the best of which is the gamma knife, developed by the famous Swedish neurosurgeon A. Leksell. The gamma knife looks like a huge helmet, into which about 200 point sources of gamma rays are mounted . The radiation of all sources is focused at one point. The position of the patient's head in relation to the helmet and the collimation of the radiation make it possible to obtain a zone of influence of a strict geometric shape, which makes it possible to purposefully destroy deeply located tumors, practically avoiding dangerous irradiation of nearby tissues.

In terms of accuracy, this effect is equivalent to surgical intervention, which justifies the name of such radiation treatment - "radiosurgery". Similar

results can be obtained using a strictly focused beam of protons, electrons and some other types of high energy.

Endovasal interventions. For a number of vascular diseases of the brain , the so-called endovasal method of treatment is used. It consists in the fact that under X-ray control, special catheters are introduced into the vascular bed, which allow special occluding devices to be delivered to the affected vessel : miniature balloons filled with latex, spirals that cause thrombus formation in the lumen of the vessel, and some others.

Typically, these operations are performed under local anesthesia. The femoral or carotid artery is catheterized . Operations are tolerated by patients more easily than open operations on the vessels of the brain, carried out under anesthesia and requiring complex surgical approaches. Endovasal surgeries are used to "turn off" some types of aneurysms and cerebral aneurysms.

2.2. Spine and spinal cord surgeries

Spinal cord surgeries, as a rule, are performed in an open way, only in rare cases stereotaxic or puncture interventions are performed. The most common operation is a laminectomy, which exposes the posterior surface of the spinal cord. It consists in resection of the arches and spinous processes in accordance with the location of the pathological process.

If it is necessary to expose the spinal cord over a large extent , osteoplastic laminotomy can be used: cutting out with the help of special osteotomes a block consisting of arches, spinous processes of the vertebrae, including their ligamentous apparatus. After the completion of the spinal cord surgery, the integrity of the spinal canal is restored by fixing the edges of the vertebral arches with wire sutures.

For operations on the ventral surface of the spinal cord at the cervical level, anterior approaches with resection of the vertebral bodies are used. When indicated , lateral approaches to the vertebrae are also used. To close defects in the vertebral bodies and stabilize them, bone grafts are used (for this purpose , fragments of the ilium and tibia, cadaveric bone are used).

If it is necessary to stabilize the spine in case of damage, various fixing metal structures are used (plates, fixed with screws to the vertebral bodies, ties, wires, etc.).
Operations on peripheral nerves are carried out according to the principles adopted in general surgery. It is important to use a surgical microscope to suture the damaged nerves.

In modern neurosurgery, prosthetics of functionally important nerve trunks are widely used when they are damaged over a considerable length with the help of nerve fragments that are not of great functional importance (for example, the superficial cutaneous nerves of the extremities).

Anesthesia. In most cases neurosurgical open operations are performed under anesthesia with controlled breathing. Stereotaxic and endovasal interventions are more often performed under local anesthesia, which makes it possible to assess the physiological effect of the operation.

An important feature of anesthesia in neurosurgical patients is such techniques and medications that do not significantly affect intracranial pressure. In many patients, especially with intracranial volumetric processes, an increase in intracranial pressure can be critical and lead to serious complications. This must be remembered during patient intubation, in which an increase in intracranial pressure

is possible. When carrying out anesthesia, you should avoid the use of drugs that can cause intracranial hypertension due to an increase in blood circulation in the brain.

An important task facing the anesthesiologist is to prevent an increase in blood pressure to a critical level. Anesthesiologists should have the means to operate under conditions of controlled arterial hypotension. Such a need usually arises during operations for ruptured aneurysms.

An important problem is the prevention and timely recognition of air embolism, which is especially dangerous during operations in a sitting position. For this purpose, a ventilation mode is selected that maintains positive pressure in the veins of the head and neck. If necessary, compression of the cervical veins is performed, which makes it possible to identify damage to large venous collectors. To register the initial signs of air embolism, the level of CO2 in the blood is constantly determined during the operation (capnography).

Depending on the nature and duration of the operation, various types of combined anesthesia are used: inhalation anesthesia using fluorothane, nitrous oxide and other gaseous anesthetics, neuroleptanalgesics, and electron anesthesia.

2.3. Features of neurosurgical operations in childhood

The skull of a newborn has a number of features. The bones of the skull are thin, elastic, the skull has fontanelles, the seams between the bones of the cranial vault are not formed. The formation of the skull is completed by the age of 2, when the cranial sutures are closed. The closing of the fontanelles is completed earlier. At an early age, in some cases, during operations on the brain, access through open fontanelles can be used; the presence of open sutures allows them to be used for trepanation. A child's thin bones can be dissected with scissors.

When planning operations, one should take into account the peculiarity of the topographic relationship in connection with the incompleteness of the formation of the skull, the flattening of its base and the weak expression of the so-called cranial fossa.

In young children under 2 years of age, due to weakness of the cervical muscles and insufficient development of the articular-ligamentous apparatus in the cervical spine, operations in a sitting position are impossible. There are also significant features in the anesthetic support of neurosurgical operations.

Neuroradiological research methods

No matter how perfect the topical diagnosis of diseases of the nervous system, no matter how vast the experience of the clinician, anatomical verification of the diagnosis is desirable and often necessary. To choose a treatment, especially when it comes to neurosurgical surgery, clear ideas about the nature, exact location and size of the pathological process, its relation to the surrounding brain structures, etc. are needed . Answers to these questions are given by X-ray contrast research methods that provide visualization of the pathological process. Some of these research methods, such as pneumoencephalography and ventriculography with air, which appeared at the beginning of the 20th century, are now practically not used, giving way to more informative and safer methods, such as computed tomography and MRI of the brain and spinal cord.

Craniography. The skull has a complex anatomical structure, therefore, in addition to general images in frontal and lateral projections, special sighting images are taken. Craniography makes it possible to recognize congenital and acquired deformities of the skull, traumatic bone injuries, primary and secondary tumor processes, some inflammatory changes, fibrous dysplasia, manifestations of a number of endocrine diseases and other lesions. Craniography reveals intracranial physiological and pathological calcifications, which make it possible to determine the side of the location of the hemispheric volumetric process by their displacement.

For topical diagnosis, it is important to identify local bone changes on radiographs caused by the effect of an intracranial pathological process (hyperostosis, usuria, increased development of vascular grooves, etc.). Typical are local changes in the sella turcica in pituitary tumors, expansion of the internal auditory canal in neuromas of the VIII cranial nerve, expansion and change in the edges of the optic nerve opening in gliomas, etc.

X-ray examination can reveal general signs of hydrocephalus: a change in the shape of the skull, an increase in its size, a flattening of the base, an increase in the vascular pattern of the bones of the vault. General changes in the skull caused by a prolonged increase in intracranial pressure are revealed : secondary changes in the sella turcica, shortening and porosity of its back, porosity of the anterior and posterior inclined processes, widening of the entrance to the saddle and deepening of the bottom, changes in the structure of the bones of the arch in the form of so-called digital depressions, discrepancies of the open cranial sutures.

Spondylography. X-ray examination of the spine is usually performed in lateral and direct projections. If necessary, sighting radiographs and photographs in special projections are made. Spondylography reveals pathological curvatures of the spine (kyphosis, scoliosis, rotation along the axis), anomalies in the development of the vertebrae. It is the main method for diagnosing traumatic injuries of the spine, nonspecific and specific (tuberculosis) lesions.

X-ray examination reveals various manifestations of vertebral osteochondrosis: narrowing of the intervertebral spaces, changes in the vertebral bodies, posterolateral osteophytes, uncovertebral arthrosis, etc. In this case, it is important to establish the size of the spinal canal, especially its sagittal diameter. Possible detection of instability of the spinal segment, displacement of the vertebrae (spondylolisthesis).

Spondylography reveals changes in tumors of the spinal cord and its roots: expansion of the intervertebral foramen in neuromas of the spinal roots, destruction of the vertebral arches in extramedullary tumors, local expansion of the spinal canal. The destruction of the vertebral bodies in metastatic tumors is also revealed.

X-ray contrast study of the cerebrospinal fluid pathways. The contrast agents used in X-ray examination of the cerebrospinal fluid spaces of the brain and spinal cord may be different. Water-soluble substances (konpey, dimer-x, amipak), easily mixing with cerebrospinal fluid, give good contrast (like casts of the ventricles of the brain and subarachnoid space), however , the level of occlusion can not always be clearly identified. For these purposes, it is better to

use heavy contrast agents, the relative density of which is greater than 1.0 (mayodil, iodinefendylate).

For contrasting liquor pathways, gases such as air, oxygen, helium can be used .

Ventriculography. Radiopaque examination of the ventricles of the brain is mainly used in the differential diagnosis of occlusive and open (communicating) hydrocephalus. The study begins with a puncture of the anterior or posterior horn of the lateral ventricle. In ventriculography with mayodil, the puncture of the anterior horn of the lateral ventricle is performed while the patient is lying down, and a contrast agent (1.5-2.0 ml) is injected while sitting; the head is slightly tilted forward and in the opposite direction. In the absence of blockage of the cerebrospinal fluid, a heavy contrast agent penetrates through the interventricular openings into the third ventricle, midbrain aqueduct, IV ventricle, cisterna magna and spinal canal. The presence of an obstacle creates a contrast agent delay, which is determined on radiographs of the skull, performed in two projections. Ventriculography should not be performed for tumors, hematomas, abscesses, parasitic cysts of the cerebral hemispheres, since it significantly worsens the condition of patients, giving scant information.

Pneumoencephalography. X-ray examination of the ventricles of the brain and the subarachnoid space by introducing air into the subarachnoid space in the sitting position of the patient through a lumbar puncture. This procedure can be performed in two ways, which are significantly different from each other: with and without excretion of cerebrospinal fluid. In pneumoencephalography, in the first way, trying to achieve good filling of the ventricles of the brain and subarachnoid space, a large amount of air (up to 60-80 ml or more) is injected and, in order not to cause a significant increase in intracranial pressure, cerebrospinal fluid is withdrawn in parallel . With pneumoencephalography without excretion of cerebrospinal fluid, air is introduced in a small amount (not exceeding 20-25 ml) slowly and strictly directed into the area of the alleged localization of the pathological process. If it becomes necessary to introduce air into the subarachnoid spaces of the base of the brain (into the cisterns), then during the manipulation the patient's head is thrown back as much as possible. Plain radiography and tomography of the skull in two projections are performed in the sitting position of the patient. This method is referred to as pneumocisternography, respectively. Although ventriculo- and pneumoencephalography makes it possible to clarify the nature and localization of a number of pathological processes (tumors, the consequences of traumatic brain injury, vascular and inflammatory diseases), recently they are practically not used, since they are invasive and inferior in their informational content to computed and magnetic resonance imaging ...

Myelography. The introduction of a contrast agent into the subarachnoid space of the spinal cord, followed by X-ray of the spine, makes it possible to clarify the nature and localization of the pathological process. Myelography is indicated for tumors of the spinal cord, hernias of intervertebral discs, chronic spinal arachnoiditis and other pathological processes that limit the lumen of the spinal canal.

Distinguish between ascending and descending myelography , depending on the type and
the relative density of the contrast agent. With descending myelography with the introduction of mayodil into the greater cistern, a suboccipital puncture is performed, 2-3 ml of cerebrospinal fluid is removed and an equal amount of mayodil is injected. X-ray examination is performed in a sitting position or lying on a table with a raised head end. When the subarachnoid space of the spinal cord is blocked, the contrast stops over the pathological focus ("rider" symptom).

In ascending myelography, the contrast agent is injected through a lumbar puncture. X-ray examination of the spine is performed with the head end of the table lowered . In this case, the lower boundary of the obstacle to the cerebrospinal fluid can be detected .

Air (pneumomyelography) and radioactive inert gas - xenon (isotopic myelography) can be used as a contrast agent . In the latter case, the propagation of xenon in the subarachnoid space is determined using a highly sensitive radio scintillation counter.

In the presence of magnetic resonance imaging, the indications for myelography are limited.

X-ray contrast study of blood vessels. Cerebral angiography. The contrast agent is injected into the great vessels of the head and a rapid serial X-ray is taken using special devices. Angiographic methods can be conditionally subdivided into straight ones, in which the carotid or vertebral artery is punctured, and catheterization, when a contrast agent is injected into the great vessels of the head by catheterization through the femoral, axillary or brachial arteries. Cerebral angiography makes it possible to clarify the nature and localization of the pathological process and is used in the diagnosis of brain tumors, malformations of the vascular system (arterial and arteriovenous aneurysms, arteriovenous fistulas), some forms of stroke to clarify the indications for surgery, as well as to monitor the results of a number of surgical interventions.

Angiography is important for examining collateral blood supply and determining the rate of cerebral blood flow. The arterial, capillary and venous phases of the passage of the contrast medium through the vessels of the brain are distinguished. Normally, the contrast leaves the vascular bed of the brain in 8-9 s, however, with a sharp increase in intracranial pressure caused by a tumor, hematoma, hydrocephalus, cerebral edema , the time of cerebral circulation can lengthen up to 15-20 s. With an extreme degree of intracranial hypertension and brain death, there is a cessation of cerebral circulation, the contrast agent does not enter the vessels of the brain. Acceleration of cerebral blood flow is noted with arteriovenous aneurysms and anastomosis.

Spinal angiography. Spinal angiography is also performed by catheterizing the arteries that supply the spinal cord at different levels. The need for this complex and time-consuming study arises when arteriovenous malformation of the spinal cord is suspected and in some spinal tumors.

Gammaencephalography

Gammaencephalography is a method for examining the brain using isotopes that have gamma radiation, a short half-life and the ability to be quickly eliminated

from the body. Such an isotope is currently technetium, which is administered intravenously or (in childhood) orally. Normally, the blood-brain barrier does not allow the isotope to penetrate into the brain tissue, while the soft integument of the head and especially the mucous membranes, muscles and glands accumulate it intensively. For some pathological processes, including tumors, barrier functions are impaired, therefore, when scintigraphy on special gamma devices, foci of excessive accumulation of the isotope are found.

CT scan

The method was proposed in 1972 by G. Housfild and Y. Ambrose, who were awarded the Nobel Prize for this development. The method is based on the measurement and complex computer processing of the difference in the absorption of X-ray radiation by tissues of different density . In a CT scan of the head, these are integumentary tissues, bones of the skull, white and gray matter of the brain, cerebrospinal fluid spaces.

Modern computed tomographs allow one to differentiate tissues with minimal structural differences and obtain images that are very close to the usual brain slices presented in anatomical atlases.

Particularly informative images can be obtained using the so-called spiral computed tomography.

For additional information, computed tomography uses radiopaque substances administered intravenously before the study. Computed tomography can provide comprehensive information on vascular diseases, traumatic injuries, brain tumors, abscesses, malformations and many other diseases of the brain and spinal cord. Numerous examples indicating the informative value of this method are given in the corresponding sections of the textbook.

It should also be noted that with the help of modern computed tomographs, it is possible to obtain an image of the vessels of the brain, to recreate a volumetric image of the skull, brain and spine. These data can be indispensable when it comes to clarifying the topographic relationship between the brain and the skull, planning reconstructive surgeries, etc.

Magnetic resonance imaging

The method is based on the registration of electromagnetic radiation emitted by protons after their excitation by radio frequency pulses in a constant magnetic field. The emission of energy by protons in the form of electromagnetic oscillations of different frequencies occurs in parallel with the relaxation process - the return of protons to their original state to a lower energy level.The contrast of the image of tissues on tomograms depends on the time required for the relaxation of protons, or rather on its two components: T1 - the longitudinal time and T2 - transverse relaxation time. The researcher, choosing the scan parameters that will be obtained by changing the delivery of radio frequency pulses ("pulse train"), can influence the contrast of the image.

Examination in the T1 mode gives a more accurate idea of the anatomical structures of the brain (white, gray matter), while the image obtained in the examination in T2 mode more reflects the state of water (free, bound) in the tissues.

Additional information can be obtained with the introduction of contrast agents. For MRI, such contrasts are paramagnets - magnevist, omniscan, etc.

In addition to capturing anatomical images, MRI can measure the concentration of individual metabolites in the brain (called MR spectroscopy).

It should also be noted that an important advantage of MRI is its safety for the patient. However, there are certain restrictions on the use of this method: it cannot be used in patients with pacemakers, implanted metal
(non-magnetic) structures.

With the help of MRI, three-dimensional images of the head, skull, brain, spine can be obtained.

Magnetic resonance imaging, performed in the so-called vascular mode, allows you to get an image of the vessels that supply the brain.

MRI can detect changes in the brain associated with its physiological activity. So, with the help of MRI, the position of the patient's motor, visual or speech centers of the brain, their relation to the pathological focus - tumor, hematoma (the so-called functional MRI) can be determined .

Positron emission tomography

The method of positron emission tomography is associated with the use of short-lived isotopes, which are used to mark substances introduced into the body (glucose, ATP, etc.) that participate in the metabolic processes of the brain. The method makes it possible to judge the state of metabolism of these substances in various areas of the brain and to reveal not only changes in the structure, but also the characteristics of metabolism in the brain.

Diagnostic operations Lumbar puncture

A lumbar puncture is performed for various purposes: obtaining cerebrospinal fluid for its analysis, determining intracranial pressure and patency of subarachnoid spaces, performing myelography, for therapeutic purposes (to extract cerebrospinal fluid and thus reduce intracranial pressure; to administer medications).

The puncture is usually performed with a special needle between the spinous processes of the L3-L4-L5 vertebrae. The patient is placed on his side with legs bent and brought to the stomach. The patient's head is also somewhat bent and located in the same horizontal plane with the body. The gap between the spinous processes of the L4-L5 vertebrae is located at the level of the line connecting the iliac crests. After processing the skin at the puncture site with a disinfectant solution (alcohol, hebitan) , anesthesia of the skin and soft tissues is performed (2-3 ml of a 0.5% solution of novocaine).

For puncture, a special needle with a diameter of 0.5-1 mm and a length of 9-12 cm is used. The needle with a mandrel inserted into it is advanced strictly in the sagittal plane and slightly upward, respectively, the gap between the spinous processes. The surgeon determines the moment of puncture of the dura mater by the feeling of "falling through" of the needle. The needle is advanced a few millimeters deeper, then the mandrel is removed, and cerebrospinal fluid flows out of the needle. When the needle is advanced into the subarachnoid space, severe pain may occur if the needle touches the cauda equina. In this case, you must carefully change the position of the needle. A pressure tube is connected to the needle . Normally, in the supine position, it ranges from 100 to 180 mm of water column. In the presence of indications, liquorodynamic tests are carried out . After that, 2-3 ml of

cerebrospinal fluid is extracted for laboratory research (determination of the amount of protein, cellular composition, Wasserman reaction, etc.).

For therapeutic purposes, especially after neurosurgical operations, different amounts of liquid (up to 10-15 ml) can be extracted.

Liquorodynamic tests are performed to determine the patency of the subarachnoid space of the spinal cord.

Kwekenstedt 's *test* consists in compression of the veins of the neck, as a result of which

intracranial pressure rises. In the absence of a block of CSF spaces above the puncture level, an increase in pressure is simultaneously noted in the manometric tube connected to the puncture needle (negative Kvekenstedt test).

If there is an obstruction in the circulation of cerebrospinal fluid, there is a slow, insignificant rise in pressure at the lumbar level. With a complete block of the subarachnoid space, there is no change in pressure in the manometric tube in response to compression of the neck veins (positive Kvekenstedt test). A similar result can be obtained when the patient's head is bent, which also leads to the obstruction of the cerebrospinal fluid outflow from the cranial cavity and an increase in intracranial pressure.

Additional information on the patency of the subarachnoid space of the spinal cord can be obtained by pressing on the patient's abdominal wall - Stuckey's test, which also leads to an increase in cerebrospinal fluid pressure due to obstruction of outflow from the veins of the abdominal cavity and spinal canal. When the CSF space is blocked at the cervical or thoracic level during the Stuckey test, the CSF pressure at the lumbar level will increase, while when the veins of the neck are compressed (Quekenstedt's test) it will be unchanged.

Suboccipital puncture

Suboccipital puncture (puncture of the cisterna magna) is performed for diagnostic purposes (analysis of cerebrospinal fluid), for the administration of drugs and for performing myelography. It can be performed both in the supine and seated position of the patient. Suboccipital puncture is performed as follows. The hair in the cervico-occipital region is shaved, the skin is disinfected. With a sharply bent head of the patient, the external occipital tubercle and the spinous process of the II cervical vertebra are determined . In the middle of the distance between them, the skin is anesthetized. The needle is inserted strictly in the sagittal plane until its end abuts against the occipital bone. As the needle is immersed, soft tissues are anesthetized with a solution of novocaine. After the needle rests on the bone, it must be removed somewhat and the end of it must be shifted down in the direction of the occipital cistern. This movement of the needle is performed until its end falls below the edge of the occipital bone. When the needle moves inward, the surgeon experiences elastic resistance at the moment of puncture of the atlantooccipital membrane. When the end of the needle enters the cisterna magna after removing the mandrel from the needle, cerebrospinal fluid begins to flow.

Performing a suboccipital puncture requires great care and skill. With the wrong technique, serious complications are possible, primarily such as injury to the posterior inferior cerebellar artery and damage to the medulla oblongata.

Ventricular puncture

Puncture of the lateral ventricles of the brain is carried out for diagnostic purposes (obtaining cerebrospinal fluid for research, measuring intracranial pressure); to perform ventriculography (contrasting the ventricles of the brain using radiopaque substances); performing some operations on the ventricular system using a ventriculoscopy.

Sometimes it is necessary to resort to ventricular puncture with a therapeutic purpose in order to reduce intracranial pressure by extracting cerebrospinal fluid in case of impaired outflow of cerebrospinal fluid from the ventricles of the brain. Ventricular puncture is also performed when installing an external drainage system for the ventricles of the brain or
performing other shunting operations on the cerebrospinal fluid system.
More often, puncture of the anterior or posterior horn of the lateral ventricle is performed.

During the *puncture of the anterior horn of the lateral ventricle* , a linear incision of the soft tissues with a length of about 4 cm is made. The edges of the skin are parted using a Jansen retractor .

A milling hole is applied, which should be 2 cm anterior to the coronary suture and 2 cm lateral to the midline (sagittal suture). The dura mater is opened crosswise and a cannula is inserted into the brain for ventriculopuncture.

The cannula is advanced parallel to the sagittal plane in the direction of the internal auditory canal. Normally, in adults, the anterior horn is located at a depth of 5-5.5 cm. With hydrocephalus, this distance can be significantly reduced.

For *puncture of the posterior horn, the* milling hole is placed 3 cm laterally and 3 cm above the external occipital protuberance. The cannulas are immersed in the brain towards the upper outer edge of the orbit. Normally, the posterior horn is located at a depth of 6-7 cm.

Test questions on the topic: General principles of treatment, diagnosis and treatment of neurosurgical diseases.

1. Pathology of the nervous system in emergency hospitalization in a neurosurgery hospital.
2. Types of surgical operations, depending on the nature of the access and the degree of radicalism.
3. Methods for stopping bleeding.
4. Types of neurosurgical operations. The purpose of their application, depending on the type.
5. Operations on the spine and spinal cord: posterior and anterior
approaches. Spinal fusion methods.
6. Craniography - informative research capabilities, indications.

7. Spondylography as a diagnostic method, what pathologies (list).

8. R-contrast methods of investigation of cerebrospinal fluid pathways. Types, technique, indications for use.
9. R-contrast methods for studying blood vessels. Types, general principles of their implementation.
10. Computer tomography. Types, possibilities, contraindications.
11. Diagnostic operations on the cerebrospinal fluid system. Types, indications, contraindications.

1

Traumatic damage to the nervous system

Purpose of the lesson: familiarization with the frequency and structure of craniocerebral injuries, the organization of neurotraumatological care, the problems of disability and rehabilitation of patients who have undergone severe TBI, the classification of TBI Pathogenesis of craniocerebral injuries: ideas about primary and secondary, diffuse and focal injuries. Traumatic brain disease concept.

Jelly should know :

1. Clinic and diagnosis of concussion and brain contusions.
2. Features of the clinic of diffuse axonal brain damage.
3. Clinical picture and diagnosis of the main forms of brain compression : epidural, subdural and intracerebral hematomas, depressed fractures of the skull bones.
4. Compression of the head.
5. Features of craniocerebral trauma in children, the elderly and injuries associated with alcohol intoxication.
6. Complications and consequences of traumatic brain injury.
7. Diagnosis formulation.

The student should be able to:

1. Examine victims with craniocerebral trauma.
2. Draw up a plan for conservative treatment of patients with craniocerebral trauma.
3. Determine the tactics of surgical treatment

1. Traumatic brain injury. Surgery.

Traumatic brain injury (TBI) is one of the most common causes of disability and death in the population. In the United States, about 50 thousand people die annually as a result of TBI . The incidence of TBI in Russia is

approximately 4: 1000 of the population, or 400 thousand injured annually, while about 10% of them die and the same number become disabled.

In peacetime, the main causes of TBI are road traffic accidents and domestic injuries.

The term "traumatic brain injury" means combined damage to the skull and brain. However, severe brain injury is often possible without concomitant damage to the bones of the skull. The opposite situation occurs when fractures of the bones of the skull are accompanied by minimal trauma to the brain.

Biomechanics of traumatic brain injury. The mechanisms of damage to the bones of the skull are more or less obvious. Under local impact (hit by a heavy object, falling on the asphalt, etc.), the bones of the cranial vault are deformed and deflected. Due to the low elasticity of the bones of the skull (especially in adults and the elderly), cracking occurs first of the inner bone plate, then the bones of the fornix throughout its entire thickness, cracks form. With high-force impacts, bone fragments are formed, which can be displaced into the cranial cavity, often damaging the brain and its membranes. From the place where the force is applied, cracks can spread over a considerable distance, including to the base of the skull.
Fractures of the base of the skull are a common component of severe traumatic brain injury. Despite the massiveness of the bone structures of the base, they do not differ in strength, since they are extremely heterogeneous: powerful bone formations - the pyramid of the temporal bone, the crest of the wings of the sphenoid bone alternate with areas where the bone sharply becomes thinner or in its thickness there are holes and cracks through which the vessels pass and cranial nerves (upper and lower orbital fissures, oval, round openings, canals and cavities in the pyramid of the temporal bone, etc.). With various types of injury (falling on the back of the head, falling from a height to the legs, etc.), mechanical influences are transmitted to the bones of the base, causing them to crack in many areas. Fissures can pass through the roof of the orbit, the optic nerve canal, the paranasal sinuses , the pyramid of the temporal bone, the foramen magnum. In this case, along the crack, defects can occur in the dura mater and the mucous membrane of the paranasal sinuses, i.e. the integrity of the structures that separate the brain from the external environment is violated .

Mechanisms of brain damage in traumatic brain injury. The mechanisms of action on the brain in traumatic brain injury are varied and have not yet been fully understood. Let's dwell on the most obvious ones.

When a damaging force is *directly applied* to the brain, for example, when struck by a heavy object, the impact is only partially absorbed by the bones of the skull, therefore, local damage to the brain may occur at the place of application of the force. These injuries are more significant if bone fragments are formed that penetrate the brain, if a wounding instrument or projectile penetrates the brain, causing the destruction of its structures.

Acceleration and deceleration that occur with all types of mechanical influences leading to a rapid movement of the head or a rapid cessation of its movement can cause severe and multiple brain damage. But even with a fixed, motionless head, the traumatic effect of these forces is important, since the brain, due to a certain mobility, can shift in the cranial cavity.

Consider a case when, under the influence of a traumatic force, a rapid movement of the patient's head occurs, followed by rapid braking (hit by a heavy object, falling onto a stone floor, asphalt, etc.). Directly under the influence of the traumatic force, damage (contusion) of the brain occurs on the side of the impact. At the moment of collision with an obstacle, acquiring a certain inertia, the brain hits the inner surface of the vault, as a result of which a focus of brain contusion is formed on the opposite side (contre coup). It should be noted that damage to the brain on the side opposite to where the force is applied is one of the most frequent manifestations of traumatic brain injury. This must be constantly remembered. So, in a victim who has fallen on the back of the head, in addition to damage to the posterior parts of the brain, one should expect combined damage to the frontal lobes.

The movement of the brain in the cranial cavity resulting from trauma, in itself, can cause multiple injuries to its various parts, primarily the trunk and intermediate mole.

So, possible bruises of the brain stem on the edges of the large occipital and tentorial foramen. An obstacle to the displacement of the brain is the crescent of the large brain, along its edge a rupture of brain structures, for example, fibers of the corpus callosum, is possible. Severe damage can occur in the hypothalamus, which is fixed by the pituitary leg to the Turkish saddle, where the pituitary gland itself is located. The bark of the lower surface of the frontal and especially the temporal lobes can be seriously damaged due to bruising on the multiple bony protrusions of the base of the skull: the crest of the wings of the sphenoid bone, the pyramid of the temporal bone, the wall of the sella turcica.

Due to the inhomogeneity of the internal structure of the brain, the forces of acceleration and inhibition act on it unevenly, and therefore internal damage to the structures of the brain, rupture of the axons of cells that cannot withstand the deformation arising from trauma are possible . Such damage to the pathways passing in the brain is multiple and can become the most significant link in a number of other brain damage (diffuse axonal damage).

The mechanisms of brain
damage during trauma deserve special attention .
arising in connection with the *rapid movement of the head in the anteroposterior direction* , for example, with a sudden throwing back of the unfixed head of a person in the car when hitting the car from behind.In this case, the movement of the brain in the anteroposterior direction can lead to a sharp tension and breakage of the veins flowing into the sagittal sinus.

Among the mechanisms affecting the brain in traumatic brain injury, the *role of uneven pressure distribution in its various structures is* undoubted . Moving the brain in a closed cavity of the dura mater filled with cerebrospinal fluid leads to the appearance of zones of a sharp decrease in pressure with the phenomenon of cavitation (similar to what happens in a pump when its piston moves). Along with this, there are zones where the pressure is sharply increased. As a result of these physical processes, pressure gradient waves appear in the cranial cavity, leading to structural changes in the brain.

Mechanical impact in traumatic brain injury is transmitted to the cerebrospinal fluid-filled ventricles of the brain, resulting in "liquor waves" that can injure the structures of the brain adjacent to the ventricles (*hydrodynamic shock* mechanism).

In severe craniocerebral trauma, the brain usually experiences the combined effect of the above factors, which ultimately determines the picture of its multiple damage.

Pathomorphological manifestations of traumatic brain injury. The pathological manifestations of the effects of trauma on the brain can be very diverse. In case of mild trauma (concussion), changes occur at the level of cells and synapses and are detected only with special research methods (electron microscopy). With a more intense local effect on the brain of injury, pronounced changes in the structure of the brain occur with the death of cellular elements, damage to blood vessels and hemorrhages in the area of injury. These changes are greatest when the brain is crushed.

In some types of traumatic effects, structural changes occur in the medulla itself, leading to rupture of axons (diffuse axonal damage). In the place of rupture, the contents of the cell - axoplasm is poured out and accumulates in the form of small bubbles (the so-called axonal containers).

Traumatic brain injury often results in damage to the vessels of the brain itself , its membranes and the skull. These vascular changes can be extremely variable in nature and severity.

With diffuse brain damage, multiple petechial *hemorrhages* are observed , localized in the white matter of the hemispheres, often paraventricularly. Such hemorrhages can be in the brain stem, which poses a threat to the patient's life.

Due to crushing of the brain, rupture of its vessels, the outflowing blood can enter the subarachnoid space, and so-called *subarachnoid hemorrhages* occur .

The same mechanisms underlie the more rare *intracerebral* and *ventricular hemorrhages.* Of particular importance in traumatic brain injury are meningeal hematomas, which are divided into 2 main groups: epidural and subdural hematomas.

Epidural hematomas localized between the bone and the dura sheath

Subdural hematomas are located in the space between the dura mater and the brain.

Classification of traumatic brain injury. Craniocerebral injuries are divided into open and closed.

With *open* traumatic brain injury, there is damage to soft tissues to multiple, affecting vital structures. Morphological changes in the area of injury are also extremely variable: from punctate hemorrhages, death of individual cell groups, local edema to gross extensive imputation with complete destruction of brain tissue (crush injury). rupture of blood vessels, hemorrhages in the destroyed tissue, pronounced phenomena of edema, spreading to large areas of the brain, sometimes to the entire brain. Changes in volumetric intracranial relationships often lead to dislocation of the brain, wedging and entrapment of the brain stem in the tentorial and foramen magnum.

Morphological changes are accompanied by a variety of functional disorders, such as damage to the mechanisms of self-regulation of cerebral circulation, violation of metabolic processes (the processes of anaerobic glycolysis begin to prevail over aerobic oxidation typical for the function of a normal brain), and intracranial pressure can sharply increase. With bruises of the hypothalamic region and the trunk, the central mechanisms of regulation of water-salt, protein, carbohydrate and other types of metabolism are damaged ; central respiratory and cardiovascular disorders develop , which can lead to the death of the patient. There is a violation of the functions of other organs: lungs, nights, liver , etc.

To the same extent, neurological symptoms are also polymorphic, which can be observed with brain contusions. This is primarily a disturbance of consciousness lasting from several minutes to prolonged coma.

With mild and moderate hemispheric injuries, weakness in the opposite limbs, impaired sensitivity, aphatic disorders, and epileptic seizures can be detected .

With basal bruises, often accompanying a fracture of the base of the skull, symptoms of damage to the cranial nerves are noted: visual with fractures passing through the optic nerve canal, With a fracture of the pyramid, deafness and paralysis of the VII pair of cranial nerves may develop .

The most dangerous are bruises of the trunk and subcortical structures, which can manifest as paralysis of the limbs, hormonal convulsions, decerebral rigidity in combination with life-threatening autonomic disorders.

The picture revealed with the help of computed and magnetic resonance imaging is also variable : from small local areas of a decrease in the density of brain tissue to multiple foci with signs of contusion, with concomitant changes characteristic of brain compression.

Depending on the severity of the injury, bruises are of mild, moderate and severe severity.

Brain contusion of mild severity is clinically characterized by switching off consciousness after trauma from several minutes to tens of minutes. After its recovery, complaints of headache, dizziness, nausea, etc. are typical. As a rule, there are retro-, con-, anterograde amnesia, vomiting, sometimes repeated. Vital functions usually without significant disturbances. Moderate bradycardia or tachycardia, sometimes arterial hypertension, may occur. Respiration and body temperature without significant deviations. Neurological symptoms are usually insignificant (nystagmus, anisocoria, signs of pyramidal insufficiency, meningeal symptoms, etc.) and regress at 2-3 weeks. Unlike concussion, fractures of the bones of the cranial vault and subarachnoid hemorrhage are possible.

Brain contusion of moderate severity is clinically characterized by switching off consciousness after trauma lasting up to several tens of minutes - hours. Expressed con-, retro-, anterograde amnesia. The headache is often severe. Repeated vomiting may occur. There are mental disorders. Possible transient disorders of vital functions, bradycardia or tachycardia, increased blood pressure, tachypnea without disturbances in the rhythm of breathing and patency of the tracheobronchial tree; subfebrile

condition. Meningeal symptoms are common. Stem symptoms are also noted: nystagmus, dissociation of meningeal symptoms, muscle tone and tendon reflexes along the body axis, bilateral pathological reflexes. Focal symptoms are clearly manifested, determined by the localization of brain contusion: pupillary and oculomotor disorders, paresis of the extremities, disorders of sensitivity, speech, etc. These focal symptoms are gradually (within 2-5 weeks) smoothed out, but they can persist for a longer time. Fractures of the bones of the vault and base of the skull, as well as significant subarachnoid hemorrhage, are often observed .

Severe brain contusion is clinically characterized by a loss of consciousness after trauma lasting from several hours to several weeks. Motor excitement is often expressed. Severe threatening violations of vital functions are observed; often dominate stem neurological symptoms (floating eye movements, gaze paresis, multiple nystagmus, swallowing disorders , bilateral mydriasis or miosis, divergence of the eyes along the vertical or horizontal axis, changing muscle tone, hormonegonia, bilateral pathological foot reflexes, etc.), which the first hours or days overlap focal hemispheric symptoms. Paresis of the extremities (up to paralysis), subcortical disorders of muscle tone, reflexes of oral automatism, etc. can be detected . Generalized or focal seizures are sometimes noted . Focal symptoms regress slowly: gross residual phenomena are frequent , a violation, first of all, of the motor and mental spheres. Severe brain contusion is often accompanied by fractures of the cranial vault and base, as well as massive subarachnoid hemorrhage.

Subarachnoid hemorrhages occur as a result of rupture of the vessels of the pia mater, veins flowing into the sinuses, and intracortical vessels, especially with brain bruises, less often due to rupture of the vessels and sinuses of the dura mater. Their symptomatology is varied. The early period is characterized by symptoms of irritation of the cerebral cortex (epileptic seizures, psychomotor agitation: patients scream, try to get up, wave their arms), meningeal and radicular symptoms. The clinical picture develops sharply or gradually. In the latter case, patients complain of headache, back pain. Their localization depends on the location of the lesion of the membranes: most often pain in the occipital or parietal region prevails, less often in the cervico-occipital region with irradiation into the eyes: often there are radicular pain in the spine. Dizziness, tinnitus, flickering of dots in front of the eyes are noted. More often, subarachnoid hemorrhage manifests itself acutely, without precursors, immediately after injury: a sharp headache suddenly occurs , meningeal symptoms appear early , psychomotor agitation, delirium, disorientation in time and space, euphoria. Excitement is replaced by stunnedness. The reaction to irritation in an unconscious patient persists. With subarachnoid hemorrhage, localized at the base of the brain, ptosis, strabismus, double vision appear; pupil response to light is often diminished. Tendon reflexes are revived at first, later reduced. The pulse is slowed down. Hyperthermia is observed. The pressure of the cerebrospinal fluid is usually increased, and an admixture of blood is found in it. Acute meningeal phenomena are expressed within a few days and gradually decrease. The course is favorable if it is possible to stop the bleeding.

Diffuse axonal injury. It is usually characterized by prolonged loss of consciousness, various symptoms of severe brain damage, paresis of the extremities, impaired tone, symptoms of decerebration, oculomotor disorders, respiratory and cardiovascular disorders. When the computer-tomographic study revealed

diffuse changes characteristic increase in brain volume, - compression of the ventricles, subarachnoid tanks. Against this background, small focal hemorrhages in the white matter of the brain can be detected .

Compression of the brain. It is noted in 3-5% of victims with traumatic brain injury. It is characterized by a rapid increase in the symptoms of brain damage, primarily of its stem sections, and poses an immediate threat to the patient's life. Most often, compression of the brain is due to the formation of intracranial hematomas: meningeal (epi- and subdural) and intracerebral. Other causes of brain compression may be cerebral edema, acute disturbance of the outflow of cerebrospinal fluid from the ventricles of the brain, subdural hygromas, depressed fracture, and some others.

With the development of a syndrome of compression of the brain, early recognition and emergency, as a rule, surgical intervention are necessary .

In this regard, the main types of brain compression will be discussed in the section on surgical treatment.

Assessment of the condition of a patient who has suffered a traumatic brain injury is of great importance for determining the outcome and possible consequences.

The most significant integral symptom reflecting the severity of brain damage is impaired consciousness. It can be clear in patients who have suffered minor trauma. For more severe injuries, stunning (moderate or deep) is observed ; stupor (the patient reacts only to strong painful irritations) and coma (complete loss of consciousness), which in turn can be moderate, deep and terminal (all signs of reflex activity are absent).

The Glasgow Coma Scale is widely used to assess the severity of a patient's condition. In it assesses points in some of the most significant symptoms. The greater the sum of points, the better the patient's condition: 15 points corresponds to a clear consciousness and good orientation of the patient in space and time, 7 points and less - a severe form of traumatic brain injury.

Diagnostics. To recognize the nature of the lesion in traumatic brain injury, it is necessary to use a set of methods. In this case, the most important thing is to adhere to the principle of dynamic observation of the patient. The condition of a patient who has suffered a traumatic brain injury, especially a severe one, can change rapidly, primarily with the development of symptoms of brain compression. A constant neurological assessment of the patient's condition in these cases is of decisive importance.

Computed tomography and magnetic resonance imaging have undeniable advantages among modern research methods. These methods allow obtaining complete information about the state of the brain (presence of contusion foci, intracranial hemorrhages, signs of brain dislocation, the state of the ventricular system, etc.).

Craniography has not lost its diagnostic value, which allows detecting fractures of the skull bones, metallic foreign bodies.

Under certain conditions, especially when it is not possible to carry out computed tomography, such methods as echoencephalography (determination

of the mixing of the middle echo) and the imposition of exploratory trepanation holes are of great importance .

A lumbar puncture is of some importance, which makes it possible to recognize subarachnoid hemorrhages and to judge intracranial hypertension. It should be noted , however, that lumbar puncture is contraindicated in patients with intracranial volumetric processes causing compression and dislocation of the brain. In severe traumatic brain injury, it is important to control intracranial pressure for targeted appropriate therapy and prevention of the most dangerous complications. For this purpose, special sensors are used to measure pressure, which are installed in the epidural space. by overlaying milling holes. For the same purpose, catheterization of the lateral ventricles of the brain is performed.

Before a doctor examining a patient with traumatic brain injury, the task is to determine the type of injury (closed, open, penetrating) and the nature of the brain damage (concussion, contusion, compression, diffuse axonal damage), to clarify the cause of compression (hematoma, depressed fracture), to determine the severity of the patient's condition; assess the nature of bone damage.

Treatment. The first measures in providing first aid to patients with traumatic brain injury at the scene of the accident should be aimed at normalizing breathing and preventing the aspiration of vomit and blood, which usually occurs in unconscious patients. To do this, put the victim on his side or with the linden down. The task of the ambulance service is to clear the respiratory tract of mucus, blood, vomit, if necessary, perform intubation, and in case of insufficient breathing, ensure adequate ventilation of the lungs. At the same time, measures are taken to stop bleeding (if any) and maintain cardiovascular activity. A patient with severe traumatic brain injury with appropriate immobilization should be urgently taken to a specialized hospital.

Principles of conservative treatment of traumatic brain injury. The volume and nature of therapeutic measures is determined by the clinical form and severity of the patient's condition with TBI. the severity of cerebral edema and intracranial hypertension, disorders of cerebral circulation and cerebrospinal fluid circulation, as well as accompanying complications and vegetative-visceral reactions, the age of the victim, premorbid and other factors.

With a *concussion* , conservative treatment is carried out, which includes analgesics, sedatives and hypnotics; bed rest is recommended for 2-5 days . For mild and moderate brain bruises, along with this, moderate dehydration therapy (furosemide, lasix, diacarb), antihistamines (suprastin, tavegil) are prescribed. With subarachnoid hemorrhage, hemostatic therapy is performed (gluconate or calcium chloride, dicinone, askorutin). Lumbar puncture for therapeutic purposes (for sanitation of cerebrospinal fluid, they are used only when there are no signs of compression and dislocation of the brain.

The duration of bed rest with a mild brain injury is 5-7 days, with a moderate injury - up to 2 weeks. depending on the clinical course and the results of instrumental studies.

With open traumatic brain injury and the development of purulent-inflammatory complications, antibiotics that penetrate the blood-brain barrier are

used (semi-synthetic analogs of penicillin, cephalosporins, fluoroquinolones, aminoglycosides, lincomycin, etc.). In case of lacerated wounds of the soft tissues of the head , primary chemical treatment and mandatory prophylaxis of tetanus are necessary (tetanus toxoid, anti-tetanus serum are injected). Compression of the brain with epidural, subdural or intracerebral hematoma, subdural hygroma, as well as depressed fractures of the skull bones are indications for surgical intervention of osteoplastic or decompressive craniotomy and removal of the substrate compressing the brain.

Resuscitation measures for severe traumatic brain injury, accompanied by a violation of vital functions, begin at the prehospital stage and continue in a hospital setting. In order to normalize breathing, they provide free patency of the upper respiratory tract (freeing them from blood, mucus, vomit, introduction of an air duct, intubation of the trachea, tracheostomy), inhalation of an oxygen-air mixture is used, and, if necessary, artificial ventilation of the lungs.

With psychomotor agitation, convulsive reactions, sedatives and anticonvulsants (seduxen, barbiturates, etc.) are used. In case of shock, it is necessary to eliminate pain reactions, fill the deficit in the volume of circulating blood, etc. Carrying out medical and diagnostic manipulations, including in patients in a coma, should be carried out in conditions of blockade of pain (nociceptive) reactions, since they cause an increase in cerebral blood flow and intracranial pressure.

When *cerebral edema and intracranial hypertension* use saluretics osmotic and colloid-osmotic agents, artificial ventilation in hyperventilation etc. Saluretics mode (Lasix in a dose of 0.5 - 1 mg / kg per day). Administered on the first day after injury (simultaneously panangin, potassium chloride is administered to prevent hypokalemia). With the development of a clinical picture of increasing intracranial hypertension, dislocation and compression of the brain due to its edema, osmotic diuretics (beckoning, glycerin) are used at a dose of 0.25-1 g / kg. Repeated or prolonged use of saluretics and osmotic diuretics requires careful monitoring and normalization of water and electrolyte balance. The attitude towards the use of corticosteroids as decongestant therapy is very restrained, including in connection with the threat of internal bleeding and other complications during their use. The reduction of intracranial pressure is facilitated by artificial ventilation of the lungs in the mode of hyperventilation with an oxygen-air mixture, which also provides. prevention and treatment of brain hypoxia and its consequences. To improve venous outflow from the cranial cavity and reduce intracranial pressure, it is advisable to put the patient in a position with the head up. In cases where these methods do not eliminate intracranial hypertension, persistent convulsive and severe vegetative-visceral reactions, and the results of clinical and instrumental studies make it possible to exclude the presence of intracranial hematomas, barbiturates or sodium oxybutyrate are used in intensive care units of specialized hospitals against the background of mechanical ventilation with careful monitoring of intracranial and arterial pressure.

For severe bruises and crush injuries of the brain with its pronounced edema, antienzyme drugs, protease inhibitors (contrical, gordox, etc.) are used. It is also advisable to use antioxidants - inhibitors of lipid peroxidation

(alpha-tocopherol, emoxipin, etc.). In case of severe and moderate traumatic brain injury, according to indications, vasoactive drugs are prescribed (aminophylline, cavinton, sermion, etc.). Intensive therapy also includes the maintenance of metabolic processes using enteral (tube) and parenteral nutrition, correction of acid-base and water-electrolyte balance disorders, normalization of osmotic and colloidal pressure, hemostasis system, microcirculation, thermoregulation, prevention and treatment of inflammatory and trophic complications.

In order to normalize and restore the functional activity of the brain, psychotropic drugs are prescribed, including nootropics and GABAergic substances (piracetam, gammalon, pyriditol, pantogam, etc.), as well as cerebrolysin and drugs that normalize the exchange of neurotransmitters (galantamine, levodopa, nakom, madopar, etc.).).

Measures for the care of patients with traumatic brain injury include the prevention of pressure ulcers, hypostatic pneumonia (systematic turning of the patient, cupping, massage, skin toilet, etc.), passive gymnastics to prevent the formation of contractures in the joints of the paretic limbs. In patients with depression of consciousness to stupor or coma, impaired swallowing, decreased cough reflex

it is necessary to carefully monitor the patency of the airways, using suction to free the oral cavity from saliva or mucus, and during tracheal intubation or tracheostomy - sanitize the lumen of the tracheobronchial tree, carefully monitor the physiological functions, take the necessary measures to protect the cornea from drying out in comatose patients (instill petroleum jelly in the eyes, close the eyelids with an adhesive plaster, etc.). It is important to regularly cleanse the oral cavity.

Patients with traumatic brain injury are subject to long-term dispensary observation. Rehabilitation treatment is carried out according to indications. Along with the methods of physiotherapy exercises, physiotherapy and occupational therapy, metabolic (piracetam, gammalon, pyriditol, cerebrolysin, etc.), vasoactive (cavinton, sermion, stugeron, etc.) drugs, biostimulants (aloe, vitreous body. FiBS), lidase, vitamins (B1, B6, B15, C, E, etc.).

For the treatment of epileptic seizures resulting from TBI. therapy is selected individually, taking into account the nature and frequency of epileptic seizures, their dynamics, age, premorbid and general condition of the patient. In traumatic brain injury (taking into account its severity, characteristics of brain damage and EEG data) , prophylactic antiepileptic drugs may be indicated.

Surgery. Surgical treatment of patients with traumatic brain injury includes primary surgical treatment for open injuries, stopping bleeding, eliminating brain compression, and eliminating liquorrhea. Surgical intervention is also used for the consequences of traumatic brain injury: suppuration of cerebral wounds and abscesses, traumatic hydrocephalus, epileptic syndrome, extensive bone defects, vascular complications (carotid-cavernous anastomosis) and a number of others.

1.1. Traumatic intracranial hemorrhage

Epidural hematomas. The cause of epidural hematomas is most often the rupture of the branches of the middle meningeal artery, which, after exiting the spinous foramen, is located in a deep groove or canal in the thickness of the

temporal bone. With cracks passing through this canal, an artery rupture occurs. The blood flowing out of the artery exfoliates the dura mater from the bone and forms a hematoma, which can lead to dislocation of the brain and its insertion into the tentorial foramen within the next few hours after the injury .

Epidural hematomas can be caused by bleeding from the sinuses of the dura mater if its outer wall is damaged.

It is also possible the formation of epidural hematomas due to bleeding from diploic vessels with extensive damage to the bones of the skull. Most epidural hematomas are located in the temporal region.

Clinical manifestations. It is important to note that in a significant percentage of cases, epidural hematomas arise from impacts of relatively low force. In this regard, many patients do not lose consciousness at all, or they note a relatively short loss of consciousness for several minutes, usually less than an hour (in about 40% of cases). After the return of consciousness, a light interval sets in, and only after a while the patient's condition begins to deteriorate again . Stunnedness, drowsiness appear, alternating with stupor and coma. Signs of wedging of the brain into the tentorial foramen are revealed, one of the first signs of which is the expansion of the pupil, usually on the side of the lesion; paresis of the opposite limbs may develop. Later, signs of decerebration appear . There are violations of cardiovascular activity - bradycardia, increased blood pressure. If the victims are not provided with emergency assistance, they die with increasing symptoms of compression of the brain stem and increased intracranial pressure.

When evaluating clinical symptoms, it should be borne in mind that due to the dislocation of the brain, compression of the brain stem against the opposite edge of the tentorial foramen is possible , as a result of which hemiparesis may occur on the side of the location of the hematoma.

With primary severe brain injury (brain contusion with prolonged loss of consciousness), the light gap is absent in the patient, there is a steadily progressive deterioration of the condition with increasing signs of compression of the brain stem.

Diagnostics. The characteristic sequence of the development of symptoms, the presence of a light gap allow, with a significant degree of probability, to assume the development of an epidural hematoma in the patient.

To clarify the diagnosis, craniographic examination is of great importance: the detection of cracks in the temporal bone corresponding to the projection of the middle meningeal artery and its things confirms the assumption of an epidural hematoma.

Computed tomography and MRI studies reveal a typical picture of an epidural hematoma, which has a characteristic lenticular shape. At the same time, these studies make it possible to reveal the degree of brain dislocation and signs of tentorial implantation. If it is impossible to use computed tomography for diagnostics, valuable information can be obtained by ultrasound examination of the brain: mixing the M-echo allows us to determine the side of the lesion.

Treatment. Since help to a patient who is suspected of having an epidural hematoma should be provided in any conditions, the imposition of

exploratory cutter holes, primarily in the basal parts of the temporomandibular frontal region, in accordance with the projections of the middle meningeal artery , has not yet lost its significance.

Technique for removing epidural hematomas. To perform the operation , a direct incision of soft tissues in the anterior parts of the temporal region and resection of the temporal bone scales by expanding the milling hole can be used . If prior to surgery the location and size of the hematoma is determined using computed tomography or magnetic resonance imaging, osteoplastic trepanation with a horseshoe-shaped incision of soft tissues is preferable . Removal of the hematoma itself is not difficult: the clots are aspirated by suction, removed with tweezers, washed with isotonic sodium chloride solution. It is important to find the source of the bleeding. The damaged meningeal artery is coagulated or ligated by suturing the dura mater where the artery passes. Sinus bleeding is stopped as described earlier. When bleeding from diploic veins, the edges of bone fragments are coated with wax. After removing the hematoma, the volume of which often reaches 70-100 ml, the brain straightens, its pulsation appears. In osteoplastic trepanation, after stopping the bleeding, the bone is put in place and the wound is sutured in layers.

Subdural hematomas. Subdural hematomas are located between the dura mater and the surface of the brain. The source of their formation can be veins, more often in the parasagittal region, damaged as a result of trauma, bleeding from the sinuses and cerebral vessels during contusion and softening.

Distinguish between acute, subacute and chronic subdural hematomas.

Acute subdural hematoma. It usually occurs with severe traumatic brain injury, accompanied by contusion and crush injury to the brain. Acute subdural hematoma manifests itself clinically within the first three days. Bleeding occurs from damaged cerebral vessels in the area of injury and from broken veins. Most often, hematomas are located on the convex surface of the brain. In 10-20% of cases, they can be bilateral.

Most significantly, acute subdural hematoma is one of the manifestations of severe brain injury. It develops against the background of loss of consciousness and other symptoms of massive brain damage. In this regard, the light gap, so characteristic of epidural hematomas, is often not detected. Clinically, the formation of an acute subdural hematoma can be suspected on the basis of an increase in symptoms of dislocation and compression of the brain.

As with epidural hematomas, computerized X-ray or magnetic resonance imaging of the brain is decisive for the diagnosis of subdural hematomas .

Important information can be obtained using carotid angiography, which reveals a large lenticular avascular area and a sharp displacement of the cerebral vessels.

Depending on the situation, echoencephalography and the imposition of exploratory trepanation holes can be used to recognize hematomas.

The detection of a subdural hematoma justifies the indications for surgical intervention, since the removal of the hematoma is a necessary condition for eliminating vital dislocation and compression of the brain. At the same time, it is always necessary to take into account the concomitant brain damage, the severity of which can

be decisive for the prognosis, which in acute subdural hematomas is often unfavorable, the mortality rate reaches 40-50%.

Immediately after injury, when the content of the hematoma consists mainly of liquid blood, it can be emptied through the cutter holes. Osteoplastic trepanation provides a great opportunity to remove both the liquid and the organized part of the hematoma, as well as revision of the concomitant brain damage. When it is crushed, aspiration of dead brain tissue and stopping bleeding are advisable .

Despite the removal of the hematoma, the pressure in the cranial cavity can remain high, the brain begins to prolapse into the wound, and therefore it is not possible to put the bone flap in place. In this case, it is important to perform plastic surgery of the membrane and carefully close the wound.

Subacute subdural hematoma develops within 4-14 days after injury, is caused by less intense bleeding and is more often accompanied by lesions of lesser severity. For a subacute subdural hematoma, symptoms of increasing compression of the brain are characteristic already in the period when the acute manifestations of traumatic brain injury begin to subside, the patient's consciousness clears up and focal symptoms begin to disappear. The prognosis for subacute subdural hematomas is more favorable and the mortality rate is 15-20%. When recognizing them using computed tomography, it must be remembered that the density of the hematoma may not differ from the density of the brain, and only the displacement of the midline structures indirectly indicates the presence of a hematoma.

Chronic subdural hematomas differ from acute and subacute ***hematomas*** by the presence of a restrictive capsule, which determines the features of their clinical course. They are diagnosed weeks, months, or (less often) years after the injury. They often occur after minor injuries that pass unnoticed by the patient. This is a kind of pathology. In the pathogenesis of chronic subdural hematomas, age-related changes, concomitant vascular pathology, alcoholism, and diabetes mellitus are of great importance . More often, chronic hematomas occur in elderly people (60 years and older).

Chronic subdural hematomas are manifested by headaches, mental disorders, manifested by a change in character, memory impairment, and inadequate behavior. The appearance of these symptoms is often the reason for hospitalization of patients with chronic subdural hematomas in psychiatric institutions. Symptoms of local brain damage can be detected: hemiparesis, aphatic disorders. A wavy course of the disease is characteristic.

Chronic subdural hematomas usually have a well-formed capsule with its own vasculature. Pathological vessels of the capsule can be a source of repeated bleeding into the hematoma cavity and lead to an exacerbation of the disease. The volume of the hematoma can be varied by filtering fluid through the semipermeable wall of the hematoma.

Chronic hematomas often reach enormous sizes, covering most of the convexital surface from the forehead to the occiput. Their thickness can reach several centimeters, and their total volume exceeds 200 ml. An increase in the volume of a hematoma can lead to dislocation of the brain and its wedging into the tentorial foramen.

In 10-20% of cases, bilateral chronic subdural hematomas are observed .

Computed tomography and magnetic resonance imaging are the best methods used to recognize chronic subdural hematomas.

Surgery. Since most chronic subdural hematomas contain liquid lysed blood, it is advisable to empty them through milling holes. The sparing technique is also justified by the large volume of hematoma and the elderly age of patients. Radical removal of the hematoma together with the capsule is more dangerous.

To empty the hematoma, it is advisable to use catheters with hermetically connected containers, where the contents of the hematoma are collected.

The catheter should be inserted into the hematoma through a small incision in the capsule to avoid air entering the hematoma cavity. The contents of the hematoma should itself flow into the drainage system as the brain expands. Forced emptying of a hematoma can provoke a retraction of the brain, rupture of blood vessels and the development of intracranial hemorrhages.

In some cases, it is advisable to wash the contents of the hematoma. It is better to do this through two milling holes, using one catheter to introduce the solution into the hematoma cavity , the other to empty it.

Special precautions are needed to avoid infection of the hematoma.

With bilateral hematomas, drainage must be carried out simultaneously so as not to cause abrupt dislocation of the brain.

With the observance of the above precautions, the emptying of hematomas in most cases leads to the recovery of patients.

Subdural hematomas of newborns. More often associated with head trauma during childbirth, especially when the fetus is extracted with forceps. They are manifested by anxiety in the child, vomiting, and a rapid increase in the size of the head. The fontanelle is tense. Emptying the hematoma is carried out either by puncture through the fontanelle, or by craniotomy with radical removal of the hematoma together with the capsule.

Intracerebral hematomas. With severe bruises of the brain, causing arrosion of blood vessels, the formation of hematomas in the thickness of the brain is possible. Their occurrence aggravates focal and cerebral symptoms caused by injury. Their recognition is possible mainly with the help of computed and magnetic resonance imaging.

Their removal is carried out by craniotomy, which allows not only to remove the blood accumulated in the thickness of the brain, but also to revise the place of brain contusion and to find the source of bleeding.

Subdural hygromas. The cause of brain compression may be an acute accumulation of cerebrospinal fluid over the cerebral hemisphere due to rupture of the arachnoid membrane. The manifestations of such hygromas differ little from the signs of subdural hematomas.

Treatment consists in opening the capsule of the hygroma (thickened arachnoid membrane) and emptying it.

In some cases, the syndrome of compression of the brain may have been caused by the accumulation of air in the subarachnoid space (pneumocephalus).

1.2. Skull fractures

It is advisable to subdivide fractures of the skull bones into convexital and basal, while it should be remembered that in severe traumatic brain injury, cracks starting in the region of the cranial vault can spread to its base.

Depending on the nature of the fracture, there are cracks, comminuted fractures, fractures with a bone defect - perforated fractures.

With a traumatic brain injury, suture divergence is possible, which is essentially not a fracture. With cracks in the cranial vault, no special treatment is required. Within a few weeks, defects in the area of the crack are filled with connective tissue, and later with bone tissue.

In case of comminuted bone injuries, indications for surgery arise if there is a deformation of the skull with displacement of fragments into its cavity - a depressed fracture.

With depressed fractures, there is often concomitant damage to the dura mater and brain. The operation is indicated in almost all cases, even if there are no neurological symptoms. To eliminate a depressed fracture, a skin incision is made in such a way as to widely expose the fracture site and maintain a good blood supply to the bone flap. If the fragments are free, they can be lifted using an elevator. In some cases, a milling hole is placed near the fracture site, through which a lift can be inserted to mobilize depressed bone fragments.

With a rupture of the dura mater and concomitant damage to the brain, the defect in the membrane expands to a size that allows revision of the brain. Blood clots, brain detritus are removed. A thorough hemostasis is performed. If the brain does not protrude into the wound, the dura mater should be sutured tightly (defects in it can be closed using an aponeurosis). Bone fragments are placed in place and fixed to each other and to the edges of the bone defect with wire (or strong ligature) sutures.

If, due to high intracranial pressure, the brain begins to prolapse into the wound, it is not possible to sew up the dura mater. In these cases, it is advisable to perform plastic surgery using a periosteal-aponeurotic flap, fascia lata of the thigh, or artificial dura mater substitutes . Bone fragments are removed. To prevent possible liquorrhea, soft tissues must be carefully sutured in layers.

In case of contamination of the wound, it is advisable to remove bone fragments due to the danger of osteomyelitis and perform cranioplasty after a few months.

With old depressed fractures, it is impossible to eliminate the deformation of the skull in the described way due to the strong fusion of fragments between themselves and with the edges of the bone defect. In these cases, it is advisable to perform osteoplastic trepanation along the edge of the fracture, to separate the fragments, give them a normal position and then rigidly fix them with bone sutures.

Fractures of the bones of the base of the skull. Fractures of the bones of the base of the skull, as noted earlier, are usually accompanied by symptoms of contusion of the basal parts of the brain, trunk, symptoms of damage to the cranial nerves.

Fractures of the base of the skull usually look like cracks, often passing through the paranasal sinuses, the Turkish saddle, the pyramid of the temporal bone. If simultaneously with the bone the membrane and the mucous membrane of

the paranasal sinuses are damaged, then there is a risk of brain infection, since there is a communication between the cerebrospinal fluid spaces and the paranasal airways (such damage is regarded as penetrating)

Clinical manifestations. The picture of a fracture of the bones of the base of the skull includes cerebral symptoms, signs of brainstem disorders, lesions of cranial nerves, bleeding and liquorrhea from the ears, nose, mouth, nasopharynx, as well as shell symptoms. Bleeding from the external auditory canal is often observed (with a fracture of the temporal bone pyramid in combination with a ruptured eardrum), nose (with a fracture of the ethmoid bone), mouth and nasopharynx (with a fracture of the sphenoid bone). Liquorrhea or the outflow of blood containing cerebrospinal fluid indicates the presence, in addition to ruptures of the mucous membranes and a fracture of the bones of the base of the skull, damage to the dura mater. Bleeding from the nose and ears acquires diagnostic value only when it is combined with neurological symptoms and if it is possible to exclude as a causal factor rupture of the mucous membranes during a bruise or eardrum under the influence of a blast wave. Such bleeding is minor and stops easily. Heavy and prolonged bleeding usually indicates a fracture.

Fractures in the anterior cranial fossa often cause bruising in the eyelids and periorbital tissue ("glasses"). It can also be a bruise with local soft tissue injury. Typical for a fracture of the bones of the base of the skull is a pronounced and symmetrical nature of bruises in the form of "glasses", sometimes with their late development and exophthalmos. With fractures in the region of the middle cranial fossa , a hematoma may form under the temporal muscle, which is determined by palpation in the form of a testate tumor. Bruising in the area of the mastoid process can occur with fractures in the region of the posterior cranial fossa.

A feature of the clinical manifestations of fractures of the base of the skull is the defeat of the cranial nerves. More often there is a lesion of the facial and auditory nerves, less often - the oculomotor, abducens and block, as well as the olfactory, visual and trigeminal. In rare cases, with fractures in the region of the posterior cranial fossa , damage to the roots of the glossopharyngeal, vagus and hypoglossal nerves is observed . The most common combination is damage to the facial and auditory nerves.

Course and outcome. Fractures of the base of the skull, if they are accompanied by gross damage to the basal parts of the brain, can immediately after injury or in the near future lead to death. Some patients are in a serious condition for a long time (respiratory and cardiac disorder, confusion), often restless, anxious. A dangerous complication of the early period in violation of the integrity of the dura mater is purulent meningitis. Persistent headaches (due to hydrocephalus, cicatricial changes in the membranes), damage to the cranial nerves, and pyramidal symptoms persist as persistent consequences .

The main complications of such fractures of the bones of the base of the skull are the outflow of cerebrospinal fluid (liquorrhea) and pneumocephalus.

Distinguish between nasal and ear liquorrhea. Nasal liquorrhea develops as a result of damage to the frontal sinus, the upper wall of the ethmoid labyrinth (in the region of the perforated plate), with cracks passing through the sella turcica and sphenoid sinus.

If the pyramid of the temporal bone is damaged, cerebrospinal fluid may leak through the external auditory canal or through the auditory (Eustachian) tube into the nasopharynx (cerebrospinal fluid rheum).

In the acute stage of traumatic brain injury, cerebrospinal fluid may leak with a large admixture of blood, and therefore liquorrhea may not be immediately detected.

Treatment. In the acute stage, treatment is usually conservative. It consists in repeated lumbar punctures (or lumbar drainage), dehydration therapy, and prophylactic antibiotics. In a significant number of cases, in this way it is possible to cope with liquorrhea.

However, in some patients, the flow of cerebrospinal fluid continues weeks and months after the injury and can cause repeated meningitis. In these cases, there are indications for the surgical removal of cerebrospinal fluid fistulas. Before the operation, the location of the fistula must be precisely determined. This can be done by means of a radioisotope study with the introduction of radioactive preparations into the cerebrospinal fluid or using computed and magnetic resonance imaging, especially if these studies are combined with the introduction of special contrast agents into the cerebrospinal fluid.

For nasal liquorrhea, trepanation of the frontal region is usually used. The approach to the location of the CSF fistula can be carried out both extra- and intradurally. It is necessary to carefully close the defect of the dura mater by suturing or plasty using an aponeurosis or fascia.

The bone defect is usually closed by a piece of muscle.

If the source of liquorrhea is damage to the wall of the sphenoid sinus, a transnasal approach with sinus muscle tamponade and a hemostatic sponge is usually used .

With cracks in the bones of the base of the skull passing through the air cavities, in addition to the outflow of cerebrospinal fluid, it is possible for air to enter the cranial cavity. This phenomenon is called pneumocephalus. The reason is the emergence of a kind of valve mechanism: with each inhalation, a certain amount of air enters the cranial cavity from the paranasal sinuses, it cannot come back , because when you exhale, the leaves of the torn mucous membrane or dura mater stick together. As a result, a huge amount of air can accumulate in the skull above the cerebral hemispheres , symptoms of increased intracranial pressure and dislocation of the brain appear, with a rapid deterioration in the patient's condition. Trapped air in the skull can be removed by puncture through the milling hole. In rare cases, there is a need for surgical closure of the fistula in the same way as it is done for liquorrhea.

With fractures of the skull base passing through the optic nerve canal, blindness may occur due to injury or compression of the nerve by a hematoma. In these cases, intracranial intervention with opening of the canal and decompression of the optic nerve is justified .

Cranioplasty. The consequences of traumatic brain injury can be varied, often extensive skull defects. They result from comminuted fractures; if it is impossible to preserve the bone flap due to high intracranial pressure and prolapse

of the brain into the surgical wound. The cause of bone defects can be in the case of osteomyelitis wound infection.

Patients with large bone defects react to changes in atmospheric pressure. The development of a cicatricial adhesive process along the edges of a bone defect can cause pain syndromes. In addition, there is always a risk of damage to areas of the brain that are not protected by bone. Cosmetic factors are also important, especially with frontal-basal defects.

These reasons support the indication for cranioplasty.

Defects in the convexital parts of the skull can be closed with prostheses made of fast-hardening plastic - styracryl, galaosta. While this polymer is in a semi-liquid state, a lamina corresponding to a skull defect is formed from it . To avoid the accumulation of blood and exudate between the dura mater and the plastic plate, several holes are made in the latter . The graft is firmly fixed with sutures to the edges of the defect. Tantalum plates and mesh are also used to close bone defects.

Recently, the bone of the patient himself has been used for cranioplasty. For this purpose, a symmetrical section of the skull is exposed and a bone fragment is cut out, corresponding in size to the bone defect. With the help of special oscillating saws, the bone flap is stratified into two plates. One of them is put in place, the other is used to close the bone defect.

A good cosmetic effect can be obtained when specially treated cadaveric bone is used for cranioplasty, but recently this method has been refrained from using due to the risk of infection with the virus of slow infections.

The most difficult cranioplasty for parabasal injuries, including the frontal sinuses, orbital walls. In these cases, a complex operation to reconstruct the skull is required . The extent and configuration of the bone lesion should be carefully examined prior to surgery . Volumetric reconstruction of the skull and soft tissues of the head using computed tomography and magnetic resonance imaging can be of great help. In these cases, the skull's own bones and plastic materials are used to restore the normal configuration of the skull .

1.3. Open head injury.

In open traumatic brain injury, the same damaging factors act on the brain as in closed trauma. The difference lies in the risk of infection, especially with penetrating wounds.

Treatment. The tactics of treating patients with open injuries is primarily determined by the task of preventing wound infection.

Primary surgical treatment. After determining the nature of the damage using the previously mentioned diagnostic tests, the patient's head is shaved and the skin is thoroughly disinfected. Crushed, non-viable areas of soft tissues are excised . The cutaneous wound is expanded if necessary in order to expose the damaged areas of the skull. Loose bone fragments and foreign bodies are carefully removed . Crushed bone areas are resected with nippers. If the dura mater is intact and there are no signs of an intracranial hematoma, it is better not to open it. The wound is sutured tightly in layers. If the membrane is damaged, its edges are excised for 1-2 mm. It is opened with additional incisions to expose the brain. Bone fragments, hair, foreign bodies are carefully removed along with

brain detritus and blood clots, the wound is thoroughly washed with isotonic sodium chloride solution and disinfectant solutions (furacilin, dioxidine). A hemostatic fibrin sponge containing an antibiotic can be used to stop bleeding.

If conditions permit (there is no brain prolapse), it is necessary to hermetically sew up the membrane. Soft tissues are carefully sutured in layers. For significant bone defects, primary cranioplasty can be performed.

When the medulla is swollen, it is necessary to perform plastic surgery of the dura mater, using the aponeurosis or periosteum for this. Closing the membrane defect in these cases prevents further bulging of the brain and its infringement in the bone hole. Local and parenteral administration of broad- spectrum antibiotics is shown . Primary surgical debridement of the wound is performed within the first three days (early surgical debridement).

If, for some reason, assistance to the victims was not provided within these terms, the so-called delayed primary treatment (3-6 days after the injury) is justified.

Consequences and complications of traumatic brain injury. Many patients who have suffered severe traumatic brain injury remain severely disabled due to mental disorders, memory loss, impaired movement, speech, post-traumatic epilepsy and other reasons.

Complications in the form of amnesia, decreased performance, persistent headaches, autonomic and endocrine disorders can be observed in a large number of patients who have suffered traumatic brain injury of mild to moderate severity.

These symptoms may be based on atrophic processes in the brain, inflammatory changes in its membranes, impaired cerebrospinal fluid circulation and blood circulation, and a number of others.

Some consequences of craniocerebral injuries require surgical treatment: post-traumatic purulent complications (abscesses, empyema), resorptive hydrocephalus, severe epileptic syndrome, carotid-cavernous fistulas and a number of others,

A brain abscess is punctured through a milling hole, then with the help of a catheter inserted into the abscess, pus is removed, its cavity is washed, antibiotics are injected. Drainage of the abscess is carried out for several days under the control of repeated CT examinations until the flow of discharge from its cavity stops. Enclosed abscesses can be removed as a whole, together with the capsule.

The cause of *hydrocephalus* in patients with traumatic brain injury is often a violation of the resorption of cerebrospinal fluid. If severe ventriculomegaly is accompanied by periventricular edema, there may be indications for bypass surgery to drain cerebrospinal fluid into the abdominal cavity (lumbar or ventriculoperitoneal drainage) or into the atrium (ventriculoatriostomy).

One of the dangerous complications of a fracture of the bones of the base of the skull can be *injury to the carotid artery.*

With cracks passing through the wall of the sphenoid sinus, in case of rupture. carotid artery, extremely dangerous *recurrent nosebleeds* can occur

If the patient is not given urgent help, he may die from acute blood loss. Treatment consists of occluding the carotid artery at the rupture site with an occlusive balloon.

If a rupture of the carotid artery occurs in the place where it passes through the cavernous sinus, characteristic *symptoms of the carotid-cavernous anastomosis* appear . Surgical treatment - endovasal occlusion of the cavernous sinus or carotid artery.

Ability to work. The clinical and labor prognosis for traumatic brain injury depends to a certain extent on the correct solution to the issue of examination of temporary disability. With a concussion, inpatient treatment lasts on average 5-7 days, temporary disability - within 2-3 weeks; with mild brain contusion - 10-14 days and 4-5 weeks, respectively; with moderate brain contusion - 2-3 weeks and 1.5-2 months; with severe brain contusion, often with a fracture of the skull bones, compression of the brain, massive subarachnoid hemorrhage, the duration of inpatient treatment can be 1-2 months, and sometimes much more.

With a likely favorable clinical prognosis, follow-up treatment of patients with an extension of the period of temporary disability is practiced.

The injured are subject to referral to a medical and labor examination for registration of disability , for whom, despite the complex of medical rehabilitation and social-preventive measures, clinical and labor forecasts remain unfavorable: persistent pronounced dysfunctions , remitting or progressive course of traumatic illness.

The criteria for establishing the III group of disability are moderately expressed vegetative-vascular, vestibular, liquorodynamic, epileptic, diencephalic, somatic disorders, mental disorders, movement and speech disorders with stationary or slowly progressive and remitting course with rare exacerbations and long periods with stable compensation social factors in each case.

The criterion for establishing the II group of disability is a progressive or remitting course of traumatic brain disease with frequent and long periods of decompensation, pronounced organic changes, disorders psyche, vestibular, liquorodynamic,
vegetative-vascular, metabolic-endocrine disorders, impaired motor function and speech, vision, with parkinsonism and other severe clinical manifestations

The criteria for establishing group I disability are persistent severe disorders of the motor function of the limbs (hemiplegia, gross hemiparesis), speech (total, sensory, motor aphasia), psyche (traumatic dementia), coordination disorders that impede movement, epileptic convulsive syndrome with frequent seizures, prolonged twilight states of consciousness, psychoorganic syndrome and sharp pronounced
intellectual and mnestic disorders, pronounced manifestations of parkinsonism, depriving patients of the possibility of self-care.

One of the important links in the comprehensive system of rehabilitation of disabled people who have suffered a traumatic brain injury is professional rehabilitation, which consists of the psychological focus of the disabled person on work activities, shown to him for health reasons, labor recommendations for a rational work arrangement, vocational training and retraining.

2. Injury of the spine and spinal cord. Surgery

Damage to the spinal cord and its roots is the most dangerous complication of spinal cord injury. It is observed in 10-15% of those who have suffered spinal trauma: 30-50% of victims die from complications caused by spinal cord injury. Most of the survivors become disabled with serious movement disorders, dysfunction of the pelvic organs, pain syndromes that persist for many years, often for life. Injuries of the spine and spinal cord are divided into *open* , in which the integrity of the skin and underlying soft tissues is disturbed, and *closed* , in which these injuries are absent. In peacetime, closed injury is the predominant type of injury to the spine and spinal cord.

Spinal injuries accompanied by damage to the spinal cord and its roots are called *complicated.*

2.1. Closed injuries of the spine and spinal cord

Spinal injury. Closed injuries of the spine arise under the influence of flexion, rotation, extension and compression along the axis. In some cases, a combination of these effects is possible (for example, with the so-called whiplash injury of the cervical spine, when, after flexion of the spine, its extension occurs).

As a result of the action of these mechanical forces , various changes in the spine are possible :

- sprain and rupture of ligaments;
- damage to the intervertebral discs;
- subluxation, dislocation of the vertebrae;
- vertebral fractures;
- dislocation fractures.

There are the following types of vertebral fractures:

- fractures of the vertebral bodies (compression, comminuted, explosive);
- fractures of the posterior semicircle;
- combined with a simultaneous fracture of bodies, arches, articular and transverse processes;
- isolated fractures of the transverse and spinous processes.

The state of stability of the spine is of particular importance. Its instability is characterized by the pathological mobility of its individual elements. Spinal instability can cause additional serious injury to the spinal cord and its roots.

It is easier to understand the causes of spinal instability if we turn to Denis's concept, who distinguishes 3 support systems (columns) of the spine: the anterior support complex (column) includes the anterior longitudinal ligament and the anterior segment of the vertebral body; the middle pillar unites the posterior longitudinal ligament and the posterior segment of the vertebral body, and the posterior pillar - articular processes, arches with yellow ligaments and spinous processes with their ligamentous apparatus. Violation of the integrity of two of the mentioned support complexes (pillars), as a rule, leads to instability of the spine.

Spinal cord injury. The causes of spinal cord injury from spinal cord injury are varied. They can be trauma to the spinal cord and its roots with a bone fragment, a dislocated vertebra, a prolapsed intervertebral disc, a hematoma formed at the site of a fracture, etc.

The consequence of trauma can be a rupture of the dura mater and direct injury of the spinal cord with a bone fragment.

Similarly to traumatic brain injury in traumatic spinal cord injury, concussion, contusion and compression are distinguished. The most severe form of local lesion of the spinal cord is its complete anatomical hiatus with diastasis of the ends at the site of injury.

Pathomorphology. In the pathogenesis of spinal cord injury, circulatory disturbance arising from trauma is of great importance . It can be ischemia of significant areas of the spinal cord due to compression or rupture of the radicular arteries, the anterior artery of the spinal cord. There may be hemorrhages in the substance of the spinal cord itself (hematomyelia) or the formation of meningeal hematomas.

Edema is a common and dangerous consequence of spinal cord injury. An increase in the volume of the spinal cord as a result of edema can lead to an increase in its compression, a secondary violation of blood circulation, a vicious circle of pathological reactions arises that can lead to irreversible damage along the entire diameter of the spinal cord.

In addition to the listed morphological structural changes, pronounced functional disorders also occur , which in the acute stage of trauma can lead to a complete cessation of motor activity and reflex activity, loss of sensitivity - spinal shock.

Spinal shock symptoms can persist for weeks or even months.

Clinical manifestations of spinal cord injury in spinal cord injury. The clinical symptoms of a complicated spinal fracture are determined by a number of reasons, primarily the level and degree of spinal cord injury.

There are syndromes of complete and partial transverse spinal cord injury.

In the *syndrome of complete transverse lesion of the spinal cord* downward from the level of the lesion, all voluntary movements are absent, flaccid paralysis is observed, tendon and skin reflexes are not triggered, all types of sensitivity are absent , control over the functions of the pelvic organs is lost (involuntary urination, defecation disorders, priapism), vegetative innervation (sweating, temperature regulation are disturbed). Over time, flaccid muscle paralysis can be replaced by their spasticity, hyperreflexia, and the automatism of the functions of the pelvic organs is often formed.

Features of the clinical manifestations of spinal cord injury depend on the level of the lesion. In case of damage to the upper cervical part of the spinal cord (CI-IV at the level of I-IV cervical vertebrae), tetraparesis or tetraplegia of a spastic nature develops with the loss of all types of sensitivity from the corresponding level. If there is concomitant damage to the brain stem, then bulbar disorders (dysphagia, aphonia, respiratory and cardiovascular disorders) appear .

Damage to the cervical thickening of the spinal cord (CV-ThI at the level of the cervical vertebrae) leads to peripheral paraparesis of the upper limbs and spastic paraplegia of the lower ones. There are conduction disorders of all types of sensitivity below the level of the lesion. Possible radicular pain in the hands. The defeat of the ciliospinal center causes the appearance of the

Bernard- Horner symptom , a decrease in blood pressure, and a slowdown in the pulse.

Injury of the thoracic spinal cord (ThII-XII at the level of I-IX thoracic vertebrae) leads to lower spastic paraplegia with the absence of all types of sensitivity, loss of abdominal reflexes: upper (ThVII-ThVIII), middle (ThIX-ThX) and lower (ThXI - ThXII).

In case of damage to the lumbar thickening (LI-SII at the level of X-XII thoracic and I lumbar vertebrae), peripheral paralysis of the lower extremities occurs, anesthesia of the perineum and legs from top to bottom from the inguinal (pupartovoy) ligament, the cremaster reflex falls out .

In case of trauma to the spinal cord cone (SIII-V at the level of I-II lumbar vertebrae), there is “saddle” anesthesia in the perineal region.

Injury of the cauda equina is characterized by peripheral paralysis of the lower extremities, anesthesia of all types in the perineum and on the legs, and sharp radicular pain in them.

Spinal cord injuries at all levels are accompanied by disorders of urination, defecation and sexual function. With transverse lesions of the spinal cord in the cervical and thoracic parts, there are dysfunctions of the pelvic organs as a syndrome of "hyperreflex neurogenic bladder". In the first time after the injury, urinary retention occurs, which can be observed for a very long time (months). Bladder sensitivity is lost. Then, as the segmental apparatus of the spinal cord is disinhibited, urinary retention is replaced by spinal automatism of urination. With a hyperreflex bladder, involuntary urination occurs with a slight accumulation of urine in it. When the cone of the spinal cord and the roots of the cauda equina are damaged, the segmental apparatus of the spinal cord suffers and the syndrome of "hyporeflex neurogenic bladder" develops . It is characterized by urinary retention with symptoms of paradoxical ischuria. Disorders of defecation in the form of stool retention or fecal incontinence usually develop in parallel with urinary disorders.

Defeats of the spinal cord in any parts are accompanied by bedsores that occur in areas with impaired innervation, where bone protrusions are located under the soft tissues (sacrum, iliac crests, heels). Decubitus ulcers develop especially early and quickly with gross (transverse) damage to the spinal cord at the level of the cervical and thoracic regions . Pressure ulcers quickly become infected and cause sepsis.

When determining the level of spinal cord injury, it is necessary to take into account the interposition of the vertebrae and spinal segments. It is easier to compare the location of the spinal cord segments with the spinous processes of the vertebrae (with the exception of the lower thoracic region). To determine the segment, add 2 to the vertebra number (so, at the level of the spinous process of the III thoracic vertebra , the V thoracic segment will be located).

This pattern disappears in the lower thoracic and upper lumbar regions, where II segments of the spinal cord are located at the ThXI-XII-LI level (5 lumbar, 5 sacral and 1 coccygeal).

There are several syndromes of partial spinal cord injury.

Syndrome of a half lesion of the spinal cord (Brown-Séquard syndrome) - paralysis of the limbs and impairment of deep types of sensitivity on the side of the lesion with loss of pain and temperature sensitivity on the opposite side. It should be emphasized that this syndrome in its "pure" form is rare, usually its individual elements are revealed.

Anterior spinal syndrome is bilateral paraplegia combined with a decrease in pain and temperature sensitivity. The reason for the development of this syndrome is a violation of blood flow in the anterior spinal artery, which is injured by a bone fragment or a prolapsed disc.

Central spinal cord syndrome (often occurs with a sharp overextension of the spine). It is characterized mainly by paresis of the hands, weakness in the legs is less pronounced, there are varying degrees of severity of sensitivity disorders below the level of the lesion, urinary retention.

In some cases, mainly with trauma, accompanied by a sharp bending of the spine, a *syndrome of damage to the posterior cords of the spinal cord* may develop - loss of deep types of sensitivity.

Damage to the spinal cord (especially with complete damage to its diameter) is characterized by dysregulation of the functions of various internal organs: respiratory disorders in cervical lesions, intestinal paresis, dysfunction of the pelvic organs, trophic disorders with the rapid development of pressure ulcers.

In the acute stage of trauma, cardiovascular disorders and a drop in blood pressure are often observed . In case of vertebral fracture, an external examination of the patient and identification of such changes as concomitant damage to soft tissues, reflex muscle tension , sharp pain when pressing on the vertebrae, and finally, external deformity of the spine (for example, kyphosis with a compression fracture in the thoracic region).

Spinal concussion. It is characterized by a lesion of the spinal cord of the functional type in the absence of obvious structural damage. Macro- and microscopically, edema of the substance of the brain and its membranes, single point hemorrhages are usually found . Clinical manifestations are due to neurodynamic changes, transient disorders of hemo- and cerebrospinal fluid dynamics. There are short-term, mild paresis, paresthesias, impaired sensitivity, disorders of the functions of the pelvic organs. The cerebrospinal fluid is not changed, the patency of the subarachnoid space is not impaired. Concussion of the spinal cord is rare. A much more common and serious injury is spinal cord injury.

Spinal cord injury. The most common type of lesion in closed and non-penetrating spinal cord injuries. Contusion occurs when a vertebra is fractured with its displacement, prolapse of the intervertebral disc, subluxation of the vertebra. With a spinal cord injury, structural changes always occur in the substance of the brain, roots, membranes, vessels (focal necrosis, softening, hemorrhages). The damage to the brain tissue is accompanied by spinal shock. The nature of movement and sensory disorders is determined by the location and extent of the injury. As a result of spinal cord injury, paralysis, impaired sensitivity, pelvic organ functions and autonomic functions develop . Trauma often leads to the emergence of not one, but several foci

of injury. Secondary circulatory phenomena can lead to the development of foci of myelomalacia several hours or even days after injury. Spinal cord injuries are often accompanied by subarachnoid hemorrhage. In this case, an admixture of blood is found in the cerebrospinal fluid. The patency of the subarachnoid space is usually not impaired.

Depending on the severity of the injury, restoration of impaired functions occurs within 3-8 weeks. However, in severe contusions with a complete anatomical break of the spinal cord, the lost functions are not restored.

Spinal cord compression. It occurs with a fracture of the vertebrae with mixing of fragments or with dislocation, herniated intervertebral disc. The clinical picture of spinal cord compression can occur immediately after injury or be dynamic (growing with the movements of the spine) with its instability and the presence of movable bone fragments.

Allocate the so-called *hyperextension injury of the cervical spine* (whiplash), which occurs during car accidents, diving, falling from a height. The mechanism of this spinal cord injury consists in a sharp hyperextension of the neck, exceeding the anatomical and functional capabilities of this section and leading to a sharp narrowing of the spinal canal with the development of ischemia or compression of the spinal cord. Clinically, hyperextension trauma is manifested by various severity of spinal cord lesion syndromes - radicular, partial dysfunction of the spinal cord, complete transverse lesion, anterior spinal artery syndrome.

Spinal hemorrhage. Most often, hemorrhage occurs when the vessels rupture in the region of the central canal and posterior horns at the level of the lumbar and cervical thickenings. Clinical manifestations of hematomyelia are caused by compression of the posterior horns of the spinal cord with outflowing blood, spreading to 3 segments. In accordance with this, there are acute segmental dissociated disturbances of sensitivity (temperature and pain), located on the body in the form of a jacket or half jacket. When blood spreads to the area of the anterior horns, peripheral flaccid paresis with atrophy is revealed. With the defeat of the lateral horns, vegetative-trophic disorders are noted . Very often in the acute period, not only segmental disturbances are observed , but also conduction disorders of sensitivity, pyramidal symptoms due to pressure on the lateral cords of the spinal cord. With extensive hemorrhages, a picture of a complete transverse lesion of the spinal cord develops . Cerebrospinal fluid may contain blood.

Hematomyelia is characterized by a regressive course. Neurological symptoms begin to decrease after 7-10 days. Restoration of impaired functions can be complete, however, neurological disorders often remain.

Hemorrhage into the spaces surrounding the spinal cord. It can be both epidural and subarachnoid. As a result of epidural hemorrhages (from the venous plexuses), an epidural hematoma forms, gradually compressing the spinal cord. Epidural hematomas are rare.

Clinical manifestations. Epidural hematomas are asymptomatic after injury. A few hours after it, radicular pain occurs with different irradiation depending on the localization of the hematoma. Then the symptoms of transverse compression of the spinal cord appear and begin to increase.

The clinical picture of intrathecal (subarachnoid) hemorrhage in spinal cord injury is characterized by acute development of symptoms of irritation of the membranes and spinal roots. Intense pain in the back, limbs, stiff neck muscles, Kernig's and Brudzinsky's symptoms appear. Very often these symptoms are accompanied by paresis of the extremities, conduction disturbances of sensitivity and pelvic disorders due to damage or compression of the spinal cord by the flow of blood. The diagnosis of hematorrachis is verified by lumbar puncture: the cerebrospinal fluid is intensely stained with blood or xanthochromic. The course of hematorrachis is regressive, and full recovery often occurs. However, hemorrhage in the cauda equina region can be complicated by the development of an adhesive or cystic arachnoiditis.

Diagnostics. X-ray research methods, including computed tomography and magnetic resonance imaging, are critical in determining the nature of spinal and spinal cord injury and in choosing an adequate method of treatment. These studies must be carried out with some caution so as not to cause additional trauma to the spinal cord.

If a fracture of the 1st and 2nd vertebrae is suspected, images are taken with a special positioning of the patient - images through the mouth.

To identify the instability of the spine, a series of images is performed with its gradual (by 5-10 °) flexion and extension, which makes it possible to identify the initial signs of instability and not cause a deterioration in the patient's condition.

Computed tomography, aimed at the level of the alleged injury, provides more complete information about damage to bone structures, intervertebral discs, the state of the spinal cord and its roots.

In some cases, myelography with a water-soluble contrast is used, which makes it possible to clarify the nature of the lesion of the spinal cord and its roots, to determine the presence of a block of the subarachnoid space. In the acute stage of trauma, this study must be performed with great care, since the introduction of contrast can increase the compression of the spinal cord in the area of the block.

In these cases, it is preferable to use magnetic resonance imaging, which provides the most complete information about the state of the spinal cord and structures of the spine.

Treatment. All victims who have suffered severe trauma should be treated as if they had possible damage to the spinal cord and spine, especially in cases of impaired consciousness, in the presence of signs of respiratory distress or characteristic symptoms of spinal lesion (paresis of the extremities, sensory disturbances, priapism, deformity of the spine etc.).

First aid at the scene is primarily in the immobilization of the spine: neck collar, shield. Special care is required when transferring and transporting the patient.

In case of severe injuries, a complex of intensive therapy measures is carried out aimed at maintaining blood pressure and normalizing breathing (if necessary, artificial ventilation of the lungs).

Patients with damage to the spine and spinal cord should, if possible, be hospitalized in specialized institutions.

Intensive anti-shock therapy continues in the hospital. Until the nature of the lesion is clarified and an adequate method of treatment is selected, immobilization remains.

The variety of pathophysiological mechanisms, clinical manifestations of spinal cord injury determines the approach to *drug therapy,* which depends on the nature and level of damage.

The acute period may be accompanied (in addition to symptoms of spinal cord injury) shock reactions with a drop in blood pressure and impaired microcirculation, which requires anti-shock therapy under the control of electrolyte levels, hemoglobin, hematocrit, and blood proteins.

For the prevention of secondary changes in the spinal cord caused by the development of edema and circulatory disorders in the acute period, some authors consider the use of large doses of glucocorticoid hormones (dexamethasone, methylprednisolone) justified .

Damage to the spinal cord at the level of ThII-ThVII segments can cause arrhythmias of cardiac activity, a decrease in the functional capacity of the myocardium, and ECG changes. In these cases, the appointment of cardiac glycosides is indicated.

To improve microcirculation, prevent thrombosis, reduce vascular permeability, angioprotectors are prescribed, anticoagulants, vasodilator drugs.

With violations of protein metabolism, cachexia, poor wound healing, the use of anabolic hormones is indicated . All victims are shown the appointment of nootropics, especially in the acute period of trauma.

Prevention and treatment of inflammatory complications is carried out by the introduction of antibacterial agents, taking into account the sensitivity of the microflora.

Both in acute and in subsequent periods, patients need the appointment of sedative, tranquilizing and neuroleptic drugs.

Prevention of complications. Dysfunction of the gas organs is one of the most common complications of spinal cord injury.

With complete transverse lesion of the spinal cord in the acute period (in conditions of development of spinal shock), paralysis of the detrusor, spasm of the sphincter of the bladder, and the absence of its reflex activity are noted. The consequence of this is urinary retention (atony and hyperextension of the bladder).

For the *prevention of dysfunction of the pelvic organs* from the first hours of hospitalization, it is necessary to clearly determine the state of urination and establish an adequate excretion of urine. In the first weeks after injury, an indwelling catheter must be inserted. Subsequently, 4-time periodic catheterization of the urinary bladder is carried out with simultaneous washing with aseptic solutions. Manipulations must be accompanied by the strictest adherence to the rules of asepsis and antisepsis.

When the phenomena of spinal shock pass, the reflex activity of the bladder is restored : it is automatically emptied at a certain filling.

More severe disorders of urination with the absence or inhibition of its reflex activity and urinary incontinence can be observed with damage to the spinal centers

of the pelvic organs (ThXII-LI) or with damage to the cauda equina roots. In these cases, in the presence of a large amount of residual urine, periodic catheterization of the bladder is indicated.

One of the main tasks in the treatment of patients with spinal cord injury is the development of reflex mechanisms that ensure automatic emptying of the bladder when it is full. The use of electrical stimulation of the bladder can help achieve this goal.

Disorder of the act of defecation, which always develops with a spinal cord injury, can be the cause of low-grade fever and intoxication. To restore the function of the rectum, it is recommended to prescribe a diet, various laxatives, suppositories, and in some cases, a cleansing enema.

For the timely and successful rehabilitation of patients, the prevention of pressure ulcers in the sacrum, ischial tubercles, greater trochanters of the femur, and heels is of paramount importance . It is necessary to choose a rational position of the patient on the stomach, sides. Indispensable conditions are the hygienic maintenance of the bed, gentle turning (every 2 hours), wiping the skin with ethyl, camphor or salicylic alcohol. Special mattresses are effective , providing automatic redistribution of pressure to the surface of the body. Various spacers are advisable, allowing you to give a physiological or necessary position for the body and limbs in a particular case.

For the *prevention of contractures of the extremities* , para-articular and paraossal ossifications, correct laying of the extremities, massage and therapeutic exercises are of great importance .

In the acute and early periods, especially with lesions of the cervical spinal cord, the *prevention of inflammatory pulmonary complications is of* great importance . It is necessary to normalize the functions of external respiration, to aspirate discharge from the respiratory tract. Aerosol inhalation of medicines, active and passive gymnastics are useful . In the absence of chest and lung injury , banks and mustard plasters are recommended . Vibration massage, ultraviolet irradiation, electrical stimulation of the diaphragm are prescribed .

For the prevention of pressure ulcers, UFOs of the lower back, sacrum, buttocks and heels are used in suberythemal doses.

In the presence of pain syndrome, diadynamic currents (DDT), sinusoidally modulated currents (SMT), ozokerite or mud applications are used in combination with electrophoresis of analgesic drugs, exercise therapy, massage.

Treatment of patients with spinal cord and spinal cord injury or its consequences should always be comprehensive. Adequate rehabilitation and spa treatment are important conditions for increasing the effectiveness of treatment of these patients.

Treatment for complicated spinal fractures. The main goals that are pursued in helping patients with complicated spinal fractures are elimination of compression of the spinal cord and its roots and stabilization of the spine.

Depending on the nature of the injury, this goal can be achieved in different ways:

• surgical method;

• using external immobilization and reduction of the spine (traction, neck collars, corsets, special fixation devices).

Spine immobilization. Prevents possible dislocation of the vertebrae and additional damage to the spinal cord; creates conditions for the elimination of the existing deformation of the spine and the fusion of damaged tissues in a position close to normal.

One of the main methods of spinal immobilization and elimination of its deformity is traction, which is most effective for trauma to the cervical spine .

Traction is carried out using a special device consisting of a brace fixed to the skull and a system of traction blocks.

The Catchfield Brace is secured with two pointed screws to the parietal tubercles. Traction with weights is carried out along the axis of the spine. Traction usually starts with a small load (3-4 kg) and gradually increases to 8-12 kg (in some cases more). The change in spinal deformity under the influence of traction is controlled by repeated X-ray images.

In case of damage to the cervical spine, immobilization of the spine can be carried out using a special device consisting of a special corset such as a vest, a metal hoop rigidly fixed to the patient's head, and rods connecting the hoop with the vest. In cases where complete immobilization is not required for injuries of the cervical spine, soft and hard collars are used. Corsets of a special design are also used for fractures of the thoracic and lumbar spine.

When using methods of external immobilization (traction, corsets), it takes a long time (months) to eliminate the deformation of the spine and the fusion of damaged structures in the required position.

In many cases, this method of treatment is unacceptable, especially when it is necessary to immediately eliminate the compression of the spinal cord. In such a situation, there is a need for surgical intervention.

The purpose of the operation is to eliminate the compression of the spinal cord, correct the deformity of the spine and reliably stabilize it.

Surgery. Various types of operations are used: with an approach to the spinal cord from the back through laminectomy, from the side or in front with resection of the vertebral bodies. Various metal plates, bone screws, and wires are used to stabilize the spine . The resected fragments of the vertebrae are replaced with bone fragments taken from the patient's ilium or tibia, special metal and ceramic prostheses, and bone taken from a corpse.

Indications for surgical intervention for spinal and spinal cord injury.

•When determining surgical indications, it should be borne in mind that the most dangerous spinal cord injuries occur immediately at the time of injury and many of these injuries are irreversible. So, if the victim immediately after the injury has a clinical picture of complete transverse lesion of the spinal cord, then there is practically no hope for an urgent operation that can change the situation. In this regard, many surgeons consider surgical intervention in these cases unreasonable.

•An exception may be the presence of symptoms of a complete break in the roots of the spinal cord. Although on the severity of damage in these cases a

surgical operation is justified first of all in connection with the fact that the possible conduction
restoration of damaged roots, and when they break, which happens rarely, a positive result can be obtained when microsurgical suturing the ends of damaged roots.

•If there are even the slightest signs of the safety of part of the functions of the spinal cord (slight movement of the fingers, the ability to determine the change in the position of the limb, the perception of strong pain stimuli) and there are signs of compression of the spinal cord (the presence of a block, displacement of the vertebrae, bone fragments in the spinal canal, etc.) , then the operation is shown.

• In the late period of injury, surgery is justified if compression of the spinal cord persists and the symptoms of its lesion progress.

• The operation is also indicated for severe deformity and instability of the spine, even with symptoms of complete transverse lesions of the spinal cord. The purpose of the operation in this case is the normalization of the supporting function of the spine, which is an important condition for more successful rehabilitation of the patient.

The choice of the most adequate treatment method - traction, external fixation, surgical intervention, the combination of these methods is largely determined by the location and nature of the injury.

In this regard, it is advisable to separately consider the most typical variants of trauma to the spine and spinal cord.

Cervical spine injury. The cervical spine is the most susceptible to injury and the most vulnerable. About 40-60% of all spinal injuries occur in the cervical spine, especially often in children, which can be explained by the weakness of the cervical muscles, significant extensibility of the ligaments, and the large size of the head.

It should be noted that trauma to the cervical vertebrae is more often than other parts of the spine accompanied by damage to the spinal cord (40-60% of cases).

Damage to the cervical spine leads to the most severe complications and, more often than with trauma to other spine, to the death of the patient: 25-40% of victims with trauma localization at the level of the three upper cervical vertebrae die at the scene.

The peculiarity of the structure and functional significance of the I and II cervical vertebrae make it necessary to separately consider their damage. I cervical vertebra (atlas) can be damaged in isolation or together with the II vertebra (40% of cases). Most often, due to trauma, the atlas ring breaks in its various links. In case of damage to the II cervical vertebra (epistrophy), a fracture and displacement of the odontoid process usually occur. A peculiar fracture of the II vertebra at the level of the articular processes is observed in the hanged ("hangman's fracture").

The CV-ThI vertebrae account for over 70% of injuries - fractures and dislocations with concomitant severe, often irreversible spinal cord injuries.

For fractures of the 1st cervical vertebra, traction is usually successfully applied by rigid external stabilization with halo vest, followed by the use of cervical

collars. In case of combined fractures of the I and II cervical vertebrae, in addition to these methods, surgical stabilization of the vertebrae is used, which can be achieved by wire tightening the arches and spinous processes of the first three vertebrae or fixing with screws in the area of the articular processes.

In some cases, to eliminate the compression of the spinal cord and medulla oblongata by a fractured tooth of the II cervical vertebra, the anterior approach through the oral cavity can be used .

Surgical fixation is indicated for fracture-dislocations of the CIII-ThI vertebrae. In dependence on the characteristics of damage it can be performed with a posterior approach fixation of vertebrae via a wire or other metallic constructions for bow and spinous processes. In case of anterior compression of the spinal cord by fragments of a fractured vertebra, a prolapsed disc, or hematoma, it is advisable to use an anterior approach with resection of the affected vertebral bodies and stabilization of the spine with a bone graft. The operation technique is similar to that used for prolapse of the median cervical discs.

Injury of the thoracic and lumbar spine. With injuries to the thoracic and lumbar spine, compression fractures often occur with the formation of the Urban wedge. More often than not, these fractures are not accompanied by spinal instability and do not require surgery.

With comminuted fractures, compression of the spinal cord and its roots is possible. In this case, there may be indications for surgery. To eliminate compression and stabilize the spine, complex lateral and anterolateral approaches, including transpleural, may be required.

Treatment of patients with consequences of spinal cord injury. One of the frequent consequences of spinal cord injury is a sharp increase in the tone in the muscles of the legs and trunk, which often complicates the conduct of restorative treatment.

To eliminate muscle spasticity when drug treatment is ineffective, in some cases it is necessary to perform an operation on the spinal cord (myelotomy), the purpose of which is to separate the anterior and posterior horns of the spinal cord at the level of the LI - SI segments (myelotomy according to Bischoff, Rotballer, etc.).

With persistent pain syndromes, which often occur with damage to the roots, and the development of an adhesive process, there may be indications for surgery on the pathways of pain afferentation.

In the event of pressure ulcers, dead tissue is excised, drugs are used that promote rapid cleansing and wound healing (solcoseryl). Local ultraviolet or laser irradiation is effective.

Ability to work. Clinical and labor prognosis depends on the level and degree of spinal cord injury. So, all surviving patients with a complete anatomical break of the spinal cord at any level are disabled in group I, but sometimes they can work in individually created conditions. With a concussion of the spinal cord, persons of mental labor are determined to be temporarily disabled for 3-4 weeks. Persons engaged in manual labor need to be released from work for at least 5-8 weeks, followed by release from lifting weights up to 3 months. The latter is due to the fact that a spinal cord injury occurs in most cases when

the vertebrae are displaced, and this involves a rupture or stretching of the ligamentous apparatus.

With a slight injury of the spinal cord, the sick leave is extended until the restoration of functions, less often the transition of the patient to a disability of the III group is advisable .

In case of moderate injury, it is desirable to prolong the temporary disability, and then transfer to the III group of disability, but not to the II, since this will not stimulate the clinical and labor rehabilitation of the patient.

In case of severe bruises, compression and hematomyelia, ischemic necrosis of the spinal cord, it is more rational to transfer patients to disability and continue treatment and rehabilitation with subsequent re-examination taking into account the neurological deficit.

The problems of medical and social rehabilitation are of particular importance. The task of the doctor is to teach the patient to make the most of the remaining motor abilities to compensate for the defects that have developed after the injury. For example, you can use the system of training the muscles of the trunk, shoulder girdle in patients with lower paraparesis. Many patients need the supervision of psychologists to help them find new stimuli in life. A difficult task is to return the sick to work: this usually requires retraining the sick, creating special conditions for them, and supporting society.

2.2. Open injuries of the spine and spinal cord

In peacetime, open wounds with the penetration of a wounding object into the cavity of the spinal canal are rare. In the treatment, the same principles are used as in providing assistance to victims with closed injuries, with the exception of measures related to surgical treatment of the wound, removal of the injured object and anti-inflammatory treatment.

Test questions on the topic: Traumatic damage to the nervous system.

1. Statistics of the frequency and structure of craniocerebral injuries.
2. Biomechanics of traumatic brain injury (outline the most basic).
3. Classification of TBI.
4. Concussion of a part of the brain.
5. Brain contusion, mild. Clinic, diagnostics.
6. Severe contusion of the brain. Clinic, diagnostics, treatment tactics.
7. Contusion of the brain with compression. Clinic, diagnostics, treatment tactics.
8. Basic principles of conservative treatment of TBI.
9. Stages of intracranial hematomas. Surgical treatment tactics.
10. Subdural hydromas. Pathogenesis, Clinic, treatment tactics.
11. Fractures of the skull vault and base. Clinic, diagnostics, treatment tactics.
12. Types of spinal injuries. Types of injuries to the spinal column.
13. Injury to the spinal cord. Damage types. Diagnostics, clinical picture.
14. Principles of surgical treatment of complicated spine and spinal cord injuries.
15. Basic principles of first aid for patients with SCI. The principles of conservative therapy in a hospital.

Situational tasks:

1: A 45-year-old patient was admitted to the hospital with complaints of headache. It is known that about an hour ago he hit his head in a fall. There was a short-term loss of consciousness, nausea, single vomiting. Upon admission: the condition is relatively satisfactory. Omniscience, contact, correctly oriented in space and time. Disturbing headache more pronounced in the right side of the head. The right parietal-temporal region is painful on palpation and percussion. There are no meningeal symptoms. From the side of the cranial nerves without pathology. There are no motor, sensory and coordination disorders. The patient was left under observation in the emergency room. A few hours later, neurological symptoms began to appear and progress in the form of impaired consciousness to the level of stunning - stupor, slight diverging strabismus due to the right eyeball, anisocoria (due to the expansion of the right pupil), and a decrease in photoreaction. In the left extremities , a decrease in muscle strength up to 3.5-4 points was noted. Computed tomography reveals a high-density structure in the right parietal-temporal region adjacent to the inner bone plate.

Make a diagnosis. What is the name of the developing syndrome? What diseases can lead to the development of such a syndrome? Determine the tactics of patient management.

2: A 41-year-old patient was found unconscious on the street after drinking alcohol. Having come to his senses, he amnesified the events that had happened to him. Since that time, I have worried about a headache , mainly in the morning, I preferred to lie face down, my memory significantly decreased , I was confused, and could not work. On admission, the patient was in a state of moderate severity, conscious, partially disoriented in time, stiff neck muscles, Kernig's symptom on both sides. Percussion of the skull is painful, more on the right, the zygomatic symptom of ankylosing spondylitis on the right, the pupils are uniform, the photoreaction is preserved. The left nasolabial fold is smoothed, right-sided hemiparesis with a decrease in strength to 3.5 points, hyperkinesis in the fingers of the left hand, tendon reflexes are revived, more on the right, grasping foot marks on both sides, does not clearly perform coordination tests with the left extremities. On the X-ray of the skull, bone-destructive changes are not determined. The fundus of the eye: the disks of the optic nerves are moderately edematous, the borders are blurred by single streak-like hemorrhages, the veins are noticeably dilated, full-blooded. On MRI (T I-weighted images), performed 12 days after injury, there is a formation with a high signal intensity adjacent to the cortex of both hemispheres .

Make a diagnosis. Determine the tactics of patient management

3: Patient 50 years old, engineer. He was admitted to the neurological department with complaints of recurrent headaches that occur several times a month, of a diffuse nature, of moderate intensity, more disturbing in the morning hours, after sleep. These phenomena have been observed over the past 2 years. At the age of 14, there was a severe traumatic brain injury (falling from a height, hitting the head) with loss of consciousness, accompanied by dizziness, nausea,

and vomiting. I did not see a doctor, these symptoms disappeared after 3 days, headaches subsequently worried rarely. When viewed in a neurological status, no meningeal and focal symptoms are detected. The patient was examined by the ECHO-ES method. The following results were obtained : MS = MO = Tr = 80 cm. No displacement of the median structures of the brain was revealed. The width of the third ventricle is 7 mm. MRI of the brain showed no focal changes in the brain. There is a significant expansion of all ventricles, smoothing of the cracks and grooves of the brain. Examination by a neuro-ophthalmologist: the contours of the SZN are blurred, the veins are full-blooded from 2 sides.

Rice.
About what the pathological process in question? What is the tactics of surgical treatment

1

Tumors of the nervous system

The purpose of the lesson : to reveal the main provisions of the pathogenesis, clinical picture, diagnosis, surgical treatment of the most common forms of neuro-oncological pathology in the work of a general practitioner. Determine the purpose, information content, indications and contraindications for the main methods of instrumental diagnostics of neurooncological diseases.

The student should know:

1. Prevalence and classification of tumors of the nervous system. Features of neoplastic lesions of
the nervous system in contrast to other oncological diseases.

2. The main neurological syndromes in tumor lesions of the brain:
primary (focal) and secondary (intracranial hypertension, dislocation disorders, etc.).

3. Clinical picture and diagnosis of tumors of hemispheric and subtentorial localization.
4. Clinical picture and diagnosis of tumors of the chiasmatic-sellar region.
5. Brain tumors in children.
6. Features of metastatic brain lesions.
7. The most informative methods for diagnosis of tumors of the brain brain, plan examination of patients.

8. Principles, possibilities and outcomes of surgical treatment. Radiation therapy, chemotherapy, symptomatic treatment.

The student must be able to.

1. Possess the technique of neurological examination of patients with neuro-oncological diseases.
2. Localize lesions of the nervous system.
3. To highlight the leading neurological symptom complex, which determines the patient's condition and the scheme of the next therapeutic and diagnostic measures.

4. Develop a management scheme and a plan for examining patients with neoplastic diseases of the nervous system.

5. Diagnose conditions requiring emergency and urgent surgical intervention.

1. Brain tumors. Surgery

Brain tumors are one of the most serious human diseases.

Not only malignant tumors infiltrate and destroy the brain, leading to the death of the patient. Benign neoplasms, due to their steady growth in the limited space of the skull, constantly squeeze the brain and sooner or later also lead to such damage that is incompatible with the patient's life.

Among all neoplasms, brain tumors account for about 10%.

Primary brain tumors in our country are detected annually in about 30 thousand people, approximately the same number of secondary (metastatic) tumors are diagnosed.

It is important to note that brain tumors often occur in childhood (among all tumors in children, about 20% are tumors of the nervous system).

The etiology of most brain tumors is the same as that of neoplasms of other organs and systems.

Genetic predisposition has been established only in relation to some tumors of the nervous system, belonging mainly to the group of phakomatoses: neurofibromatosis, tuberous sclerosis, Hippel-Lindau disease.

In the emergence of a number of tumors, the role of dysembryogenesis (craniopharyngiomas, dermoid and epidermoid cysts, teratomas, etc.) is undoubted . Classification. There are different approaches to grouping tumors of the nervous system. The most common is the WHO classification.

The classification is quite complicated and is mainly needed for specialists.

In everyday practice, brain tumors are easier to divide into intra- and extracerebral.

For *intracerebral* are tumors that develop from the cellular elements, which form the stroma of the brain: mostly gliomas - astrocytomas, oligodendrogliomas, ependymomas, glioblastomas; as well as tumors arising from embryonic cells of the nervous system: medulloblastomas, neuroblastomas, ependymoblastomas and some others.

The main feature of these tumors is that they arise in the brain tissue itself and there is no real border between tumor cells and brain cells: tumor elements can be found in brain tissue at a considerable distance from the main cluster of tumor cells. These tumors, as they grow, replace and destroy various structures in the brain. Consequently, intracerebral tumors, as a rule, cannot be completely removed by surgery .

Histological classification of tumors and tumors of the central nervous system

1. Neuroepithelial tumors:
- Astrocytic tumors
- Oligodendroglial tumors
- Mixed gliomas
- Ependymal tumors
- Choroid plexus tumors
- Neuronal and mixed neuronal-glial tumors
- Embryonic neuroepithelial tumors
- Tumors of the pineal parenchyma

2. Tumors of the cranial and spinal nerves
3. Tumors of the meninges:

- Tumors from meningothelial cells
- Mesenchymal non-meningothelial tumors
- Melanocytic tumors of the membranes

4. Pituitary tumors
5. Tumors of the remains of the pituitary tract
6. Germ cell tumors (germ cell)
7. Tumors of the hematopoietic tissue

8. Tumors invading the cranial cavity and spinal canal
9. Metastatic tumors

10. Tumors of unknown origin
11. Cysts
12. Vascular tumor lesions
13. Reactive and inflammatory processes that mimic tumors

Extracerebral tumors develop outside the brain from its membranes, cranial nerves, from the epididymis - the pituitary gland. They mainly compress the brain; there is usually a fairly clear border between the tumor and the brain. This is a large group of shell tumors (meningiomas), neurinomas of the cranial nerve roots (mainly III, V, VIII).

An intermediate position is occupied by tumors that have arisen as a result of disturbances in the process of brain development - *dysembryogenetic tumors.* They can penetrate deeply into the brain, but in general they are quite well delimited from it.

An independent group consists of *metastatic tumors.*

Brain tumors can vary dramatically in grade. For malignant brain tumors characterized by infiltrative growth and rapid progression of disease. Unlike malignant tumors of other organs, brain tumors do not give distant metastases, but if metastasis occurs, then the tumor is disseminated through the cerebrospinal fluid spaces (the most malignant gliomas, embryonic and germinal tumors metastasize).

The duration of the disease in benign and malignant tumors is different.

So, patients with benign gliomas live 8-10 years, sometimes more, and with malignant gliomas usually no more than a year.

One of the main features of brain tumors is that they are located in a confined space, as a result of which, as they grow, they lead to a change in the volumes of intracranial structures, which is expressed primarily in an increase in intracranial pressure and the development of dislocation syndromes.

In addition, the tumor has a direct effect on the areas of the brain where it is located.

In accordance with these pathogenetic mechanisms, there are 3 main groups of symptoms of brain tumors: 1) cerebral, caused by increased intracranial pressure; 2) local and 3) symptoms "at a distance" arising from the displacement of the brain and compression of its stem sections in the tentorial and large occipital foramen.

General cerebral symptoms. Symptoms of increased intracranial pressure are most pronounced in tumors that cause occlusion of the cerebrospinal fluid (tumors of the posterior cranial fossa, ventricles of the brain), tumors of the temporal lobe (often accompanied by dislocation of the brain and impaired CSF circulation at the level of the tentorial foramen), tumors that compress the main venous outflow tract (parasagittal meningiomas).

Headache often the first symptom of a tumor due to an increase intracranial pressure. The headache may be general, not having a clear localization. It occurs as a result of irritation of the dura mater, which is innervated by the trigeminal, vagus and glossopharyngeal nerves, and the walls of blood

vessels; violation of venous outflow in the diploic vessels of the bone. Morning pain is characteristic of hypertensive syndrome. Over time, the pain intensifies, becomes permanent. The predominance of pain in any area of the head may be a symptom of a local effect of a tumor on the dura mater and blood vessels.

Vomiting is one of the characteristic symptoms of increased intracranial pressure. It is repeated, often at the height of the headache. It should be noted that vomiting may be a local symptom of a tumor affecting the fundus of the IV ventricle.

Congestive optic discs one of the typical and striking manifestations intracranial hypertension. First, there is a short-term blurred vision, it can increase with stress, physical exertion. Then the visual acuity begins to decrease . The end result is blindness due to so-called secondary atrophy of the optic nerves.

Epileptic seizures - an increase in intracranial pressure and concomitant changes in the blood circulation in the brain, can cause general epileptic seizures. However, more often the appearance of seizures, especially focal ones, is the result of a local effect of the tumor on the brain.

Mental disorders in the form of lethargy, apathy, memory loss, ability to work, irritability can also be caused by an increase in intracranial pressure.

Dizziness , occurs in patients with brain tumors may Life 1 consequence of stagnation in the maze.

The consequence of intracranial hypertension can be changes in cardiovascular activity (increased blood pressure, bradycardia) and respiratory disorders.

Focal symptoms. Determined by the localization of the tumor. Will be considered when describing the most common tumors. Remote symptoms are also possible - dislocation symptoms

Diagnostics. Features of the anamnesis, the steady progression of the disease, a combination of cerebral, focal and dislocation symptoms most often give reason to suspect a brain tumor. However, an accurate topical diagnosis and clarification of such important details for a possible operation as the nature of tumor growth (infiltrative or nodular), blood supply, attitude to cerebrospinal fluid, etc., can be made after a comprehensive examination of the patient.

Laboratory and functional research data. The study of the cerebrospinal fluid reveals symptoms that are very typical for some tumors: an increase in pressure and the presence of protein-cell dissociation in the cerebrospinal fluid (a high level of protein with a normal content of cellular elements). However, it should be remembered that lumbar puncture in patients with suspected brain tumor should be performed with great care and limited to the withdrawal of a small amount of cerebrospinal fluid. Lumbar puncture is contraindicated in cases of dislocation, with suspicion of a tumor of the posterior cranial fossa, temporal lobe, and cerebral ventricular system.

Examination of the fundus reveals symptoms of increased intracranial pressure and signs of primary damage to the optic nerves.

Craniographic examination can reveal characteristic signs of increased intracranial pressure and local changes in the skull caused by the tumor itself , such as bone destruction, its infiltration by the tumor.

The contrasting of liquor spaces with air (pneumoencephalo-, pneumoventriculography) or a radiopaque substance, which was widely used until recently, is now practically not used.

Radioisotope scanning allows diagnosing tumors with the ability to accumulate radiopharmaceuticals (meningiomas, malignant gliomas, metastases).

X-ray computed tomography, magnetic resonance imaging and angiography are of course crucial.

Treatment. The main treatment for most tumors is surgery: many extracerebral tumors can be removed completely. With intracerebral tumors, it is often necessary to be limited to partial excision or to perform palliative operations. For malignant tumors, in addition to surgical treatment, radiotherapy is used, chemotherapy drugs are used.

From a practical point of view, it is customary to distinguish several groups of tumors, the clinical manifestation of which and the methods of treatment used have a certain originality.

- Tumors of the cerebral hemispheres:
- extracerebral;
- intracerebral;
- intraventricular.
- Tumors of the chiasmatic-sellar region.
- Tumors of the posterior cranial fossa.
- Metastatic tumors.
- Tumors of the bones of the skull.

1.1. Tumors of the cerebral hemispheres

With tumors of the cerebral hemispheres, a variety of symptoms are noted, due to the specifics of those areas in which they are located. Before proceeding to the description of individual tumors of the cerebral hemispheres, let us dwell on the syndromes of lesion of its lobes.

Tumors of the frontal lobes. The main symptoms are mental disorders, epileptic seizures, aphasia (with left-sided lesion), movement disorders. Mental disorders are characteristic, which appear early and are more pronounced than with tumors of other localization. Lethargy, lethargy, apathy, lack of initiative, irritability, depression are noted . Memory and attention are impaired, thinking, the ability to synthesize are impaired , symptoms such as eroticism, bouts of unreasonable rage are not uncommon . In the late stage, the intellect suffers more, patients do not assess the severity of their condition, untidiness appears, control over the functions of the pelvic organs disappears . Sometimes patients are euphoric, prone to "flat" jokes, foolishness (moria), commit ridiculous acts. In half of the cases, epileptic seizures are observed.

When the tumor is localized in the posterior parts of the frontal region, adversive seizures occur : turning the head and eyes in the opposite direction to the focus in combination *with* clinical and tonic convulsions in the opposite limbs.

With large tumors, astasia and abasia sometimes occur (inability to walk and stand), trunk ataxia (the patient is not able to sit up in bed on his own) due to damage to the frontal-bridge pathways.

Other movement disorders include poor movement and facial expressions, lack of motor initiative, and muscle stiffness. Sometimes it can be observed
unmotivated laughing or crying, grasping phenomenon (involuntary clenching of the hand into a fist when touching the palmar surface of the hand and fingers). Exposure to the cortical-spinal fibers can lead to the development of muscle weakness on the opposite side of the body, especially in the muscles of the face and tongue.

When the tumor is localized on the basis of the frontal lobe, due to the effect on the olfactory tract, a weakening or loss of smell is observed on the side of the lesion. Tumors located in the posterior parts of the base of the frontal lobe can compress the optic nerve, leading to its atrophy on the side of the tumor and congestion in the fundus from the opposite side as a result of increased intracranial pressure (Foerster-Kennedy syndrome).
With left-sided localization (in right-handers), motor aphasia is possible.

Tumors of the precentral gyrus. As a result of irritation of the cortical structures of this area, clonic convulsions occur in the opposite limbs (Jacksonian seizures). Convulsions can be limited to only one muscle group, or spread to the entire limb or half of the body, or go into a generalized tonic-clonic seizure. With the progression of the disease, hemiparesis of the opposite limbs develops , up to hemiplegia.

Tumors of the postcentral gyrus. They are characterized by irritation and subsequent loss of sensitivity. Local seizures begin with paresthesia in a specific area, spreading in a sequence of cortical representation of sensitivity in the postcentral gyrus.

Tumors of the temporal lobe. Symptoms of increased intracranial pressure with tumors
The temporal lobes appear early. Headaches are constant and intense. Often noted
stagnant discs of the optic nerves. Focal symptoms are often mild, especially with a right-sided tumor. Epileptic seizures are often observed, which are preceded by an aura in the form of auditory, gustatory or olfactory hallucinations, often accompanied by involuntary movements (licking, smacking the lips, chewing and swallowing), as well as unpleasant sensations in the epigastric region. The seizure can proceed in the form of a secondary generalized tonic-clonic. Psychomotor automatisms, erratic inferences, memory disorders and emotional paroxysms, including feelings of fear and depression, are sometimes observed . Sometimes there are peculiar states when everything around it seems to the patient to be distant and unreal, but already once previously seen or never seen. Auditory hallucinations (noise, whistling, musical melody, etc.), gustatory and olfactory ("strange" taste or smell) can be noted outside of seizures. The defeat of the uncinate gyrus causes a deterioration in taste and smell, although, as a rule, does not lead to their complete loss. When the tumor is localized in the deep parts of the temporal lobe , an upper quadrant homonymous hemianopsia is often observed, which later turns into a complete one. Vestibular disorders often occur: a feeling of instability and rotation of surrounding objects. Sometimes there are symptoms of

damage to the oculomotor nerve in the form of ptosis and pupil dilation. Deep tumors can cause paresis on opposite limbs due to pressure on the inner capsule and peduncle. For lesions of the temporal lobe of the left hemisphere in right-handers, speech disorders (sensory and amnestic aphasia, paraphasia) are characteristic.

Tumors of the parietal lobe. With tumors of the parietal lobe, there are sensitivity disorders on the opposite side of the body, disorientation in one's own body, and with damage to the left hemisphere , loss of reading, writing, and counting skills. The most affected are complex types of sensitivity - stereognosis, a sense of localization, etc. Difficulty is noted when performing habitual, automated actions. When the tumor spreads to the inferior parietal lobe, disorientation in space and body schema occurs : the ability to distinguish right from left is impaired, ignorance of his paretic limb (usually left) is noted, or the patient believes that he has 3-4 arms or legs.

Tumors of the occipital lobe. Characteristic local tumor syndrome occipital share are visual field defects in a contralateral homonymous hemianopsia and livestock When the tumor in calcarine furrows can be observed quadrant hemianopsia. Tumors of the occipital lobe can manifest as epileptic seizures in the form of turning the head and eyes to the side opposite to the tumor, with a preceding visual aura. Sometimes there are visual hallucinations, various forms of optical agnosia,
subject agnosia, metamorphopsia (the surrounding objects seem to be angular, irregular in shape, their sizes in comparison with the real ones - larger or smaller). The phenomenon of visual agnosia often develops with bilateral lesions of the occipital lobes.

Tumors of the basal nodes and the optic tubercle. With tumors in this area , symptoms of intracranial hypertension, paresis and impaired sensitivity of the opposite limbs (damage to the inner capsule) develop early . Changes in muscle tone may be observed . Signs of compression of the midbrain are often noted.

Tumors of the third ventricle. First of all, these tumors lead to impaired CSF circulation and increased intracranial pressure.

With damage to the bottom of the third ventricle (hypothalamus), polydipsia, polyuria, hyperglycemia, glucosuria, obesity, sexual weakness, an increase or decrease in body temperature occur. Memory disorders (Korsakov's syndrome), sleep disturbances, and decreased mental activity are often detected.

Tumors of the pineal region and posterior parts of the third ventricle. They are characterized by the early development of hydrocephalus and intracranial hypertension due to occlusion of the cerebral aqueduct (Sylvian aqueduct), as well as symptoms of damage to the lining of the midbrain. There are paresis of the gaze along the vertical, paresis of convergence, the disappearance of the reaction of the pupils to light, vertical nystagmus. More gross oculomotor disorders, as well as hearing loss, are possible.

The listed symptoms can be manifested to one degree or another in tumors with different histological structures, but these signs are more pronounced in intracerebral, especially malignant tumors that destroy the brain.

1.1.1. Extracerebral tumors

Meningiomas. Meningiomas are usually slow-growing tumors separated from the brain. They develop from the arachnoendothelial cells of the meninges. Meningiomas account for 15-20% of all primary brain tumors, usually appear at the age of 35-55 years, and are almost twice as common in women.

Several types of meningiomas are distinguished histologically. Of these, the most common are meningotheliomatous, consisting of layers of polygonal cells, and fibroblastic (cell clusters are separated by the connective tissue stroma). Meningiomas are often located along the venous sinuses (superior sagittal, cavernous, transverse). They affect the dura mater over a long distance and often infiltrate the bone, spreading in it along the Haversian canals. As meningiomas grow, they first squeeze the brain, then deeply penetrate into it.

There are meningiomas located on the covexital surface of the brain, while a significant part of them are tumors that develop from the lateral lacunae of the walls of the sagittal sinus and sickle process. These are the so-called parasagittal meningiomas.

A significant part of meningiomas develops in the region of the base of the skull - basal meningiomas. These are meningiomas in the area of the olfactory fossa, the wings of the sphenoid bone, the tubercle of the sella turcica, the clivus.

Clinical manifestations. The nature of the clinical symptoms depends on the location of the meningiomas. The initial manifestation of superficial hemispheric meningiomas is often focal seizures and their equivalents. So, the first manifestation of parasagittal meningiomas located in the motor region of the cerebral cortex is often convulsions in the opposite leg. Later, as the tumor grows, symptoms of prolapse join - hemiparesis, speech disorders and a number of other cortical disorders.

Meningiomas that develop in the anterior fossa often become very large. They cause a loss of smell, later mental disorders join (decrease in criticism, disinhibition), there is a decrease in vision due to an increase in intracranial pressure (secondary atrophy of the optic nerves) or as a result of direct pressure of the tumor on the optic chiasm and optic nerves (primary atrophy of the optic nerves).

Meningiomas in the area of the Turkish saddle tubercle rarely reach a large size, they early cause a decrease in vision as a result of compression of the optic nerves by the tumor and
visual chiasm. Meningiomas in the area of the wings of the sphenoid bone squeeze the basal parts of the temporal and frontal lobes of the cerebral hemispheres, causing epileptic seizures with a visceral aura, speech disorders with left-sided lesions, and other symptoms.

Diagnostics. Craniography reveals the characteristic signs of bone infiltration by a tumor, its sharp thinning, and a change in structure. As with other tumors, CT and MRI can accurately determine the location, size and shape of a meningioma.

With meningiomas located near large vessels of the base or near the sinuses, angiography is of great diagnostic value. It is important for clarifying the sources of blood supply and the degree of tumor vascularization. In basal meningiomas, it allows you to identify tumor overgrowth of the carotid artery and

its branches, and in parasagittal meningiomas - sinus patency. This data is extremely important when planning an operation.

Treatment. Most meningiomas are benign, well-defined tumors that can be successfully removed with surgery.

With convexital meningiomas, a skin incision and craniotomy are done according to the location of the tumor. The dura mater is usually opened with a circular incision along the edge of the tumor node. In this case, the main vessels supplying the tumor are coagulated and intersected . At the border of the tumor , vessels are coagulated and intersected , mainly veins passing to the brain. The brain is separated using cotton strips moistened with isotonic sodium chloride solution. When the mobilization of the tumor is completed, the latter is removed in a single block along with the dura mater. In case of large tumors, in order to avoid trauma to the brain, it is first advisable to enucleate the tumor and then remove it in parts, which makes it possible to almost completely avoid traction of the brain. If the tumor infiltrates the bone, the latter is resected to the border of the unaltered bone.

After removal of the tumor, plastic surgery of the dura mater is performed using a canned cadaveric membrane, aponeurosis or fascia lata of the thigh. If there is a defect to the bone, it can be closed with styracryl or a homoscopy graft. When removing parasagittal meningiomas, the most important task is to preserve the parasagittal veins in the area of the central gyri. Resection of the tumor-infiltrated sagittal sinus in the middle and posterior regions is justified only if it is completely occluded. If, when removing the tumor, it is necessary to open the sinus, bleeding from it is stopped by suture on its wall. In some cases, it is necessary to perform plastic surgery of defects in the walls of the sinus using a flap from the dura mater.

The most difficult is the removal of basal meningiomas that extend into the cavernous sinus and overgrow the carotid artery and the vessels departing from it, as well as localized in the region of the medial sections of the wings of the sphenoid bone or clivus.

Radical tumor resection requires adherence to a number of principles: the lowest possible resection of the basal parts of the skull in the frontotemporal region, the use of complex modern approaches (for example, with the reaction of the zygomatic arch and different parts of the temporal bone pyramid), which allows exposing the tumor with minimal traction of the brain. The operation requires a long dissection of the tumor under a microscope, sometimes it cannot be performed simultaneously, and then a second intervention is required to achieve complete removal of the tumor.

The results of treatment depend on the radicality of the operation and the histological structure of the tumor. If the meningioma is not completely removed, its recurrence may occur after a few years.

It is technically difficult to remove the crescent meningioma and the tentorium of the cerebellum due to their remoteness from the end of the brain surface.

1.1.2. Intracerebral tumors

Intracerebral tumors of the cerebral hemispheres are mainly gliomas of various degrees of malignancy - astrocytomas, oligodendrogliomas, ependymomas, glioblastomas.

Hemispheric astrocytomas. Astrocytomas are most common at the age of 30-50 years and account for 1/3 of all glial brain tumors. Astrocytomas are localized more often in the white matter of the temporal and frontal lobes.

Depending on the predominance of cellular elements or connective tissue of the stroma

there are protoplasmic, fibrillar and mixed astrocytomas. Other histological variants of astrocytomas are less common.

Poor (anaplastic) astrocytomas are distinguished, characterized by a denser arrangement of cells, cellular and nuclear polymorphism, an increase in the number of vessels and proliferation of their endothelium.

They can vary significantly in their structure and growth patterns. Part of astrocytomas grows diffusely, occupying a significant area of the hemisphere and having no clear boundaries with it. With computed tomography, it is possible to identify only the zone of density change corresponding to the area of the tumor, compression of the ventricles and cerebrospinal fluid spaces, displacement of the midline. The disease can last for a number of years and manifest itself as focal epileptic seizures and an increasing intracranial pressure syndrome, increasing in severity .

Some astrocytomas are more compact in structure, in some places they have relatively clear boundaries with the brain. On computed tomograms, the tumor is detected by a density different from the brain, which can be higher, lower than the brain density, or is heterogeneous. Often, the tumor contains cysts.

Astrocytomas also differ in the degree of blood supply: some are almost avascular, others are richly vascularized. With compact astrocytomas, in addition to the symptom of increased intracranial pressure, clear symptoms of brain damage can be detected, corresponding to the localization of the tumor.

The growth rate of a tumor largely depends on the degree of its malignancy: with benign astrocytomas, the duration of the disease is 7-8 years, with anaplastic - 1-2 years.

Treatment. With compact nodular astrocytomas, surgery is indicated, the tumor can be radically removed, especially if it is located in functionally less significant areas, for example, in the right temporal lobe.

It is more difficult to resolve the issue of the expediency of surgery for widespread diffuse gliomas. If there are no pronounced symptoms of intracranial hypertension, it is preferable to clarify the histological structure of the tumor using stereotaxic biopsy followed by radiation. With severe intracranial hypertension, partial removal of the tumor is performed for the purpose of internal decompression.

During the operation, to determine the localization of the tumor, it may be necessary to use an ultrasound scan or a radioisotope method.

An important method for localizing a tumor located under the cortex is a brain puncture. This simple technique allows the surgeon to detect increases or decreases in tissue density and locate cysts in the tumor.

For anaplastic astrocytomas in the postoperative period, irradiation and chemotherapy with special drugs, vincristine and some others are carried out .

Oligodendrogliomas. Tumors are mostly benign, relatively slow growing. They consist of cells containing the same type of round nuclei. In more than 70% of cases, microscopic calcifications are found in tumors. More often, oligodendrogliomas are located in the deep parts of the hemispheres in the paraventricular region, sometimes on both sides, and may also have a predominantly intraventricular arrangement. Oligodendrogliomas are poorly vascularized, prone to calcification. Manifested by epileptic seizures and slowly progressive symptoms of damage to certain parts of the brain.

Removal is usually partial. Some authors consider radiation treatment to be justified .

Ependymomas. Ependymomas are benign, relatively slow-growing tumors. Histologically, they are characterized by capillary cell structures containing small round nuclei. Cell clusters are often rosette-like. Ependymomas are localized in the ventricles of the brain, but can also spread paraventricularly.

Malignant ependymomas - ependymoblastomas are characterized by rapid growth, more often they occur in children, localized not only in the ventricles, but also in the white matter of the hemispheres. Cysts form in the tumor tissue , alternating with foci of necrosis and hemorrhage.

Glioblastoma. These are the most malignant glial brain tumors, localized mainly in the cerebral hemispheres, they occur more often at the age of 40-60 years,
characterized by rapid infiltrative growth. Glioblastomas can be located in different parts of the brain, but more often in the temporal and frontal regions. Often the tumor affects the corpus callosum and spreads to both hemispheres.

Characterized by cellular polymorphism, the presence of multinucleated giant cells and an abundance of dividing cells. Glioblastomas are characterized by the presence of many pathological newly formed vessels, the formation of arteriovenous shunts. There are extensive areas of necrosis and hemorrhages in the tumor tissue . These morphological features of glioblastomas are also noted in diagnostic studies. Angiography often reveals abnormal vasculature and early contrast filling of tumor veins. On computed tomograms, glioblastoma corresponds to a zone of inhomogeneous density. In the center of the tumor , large areas of low density are found - areas of necrosis.

Surgical treatment is ineffective, since radical removal of the tumor is impossible, especially if it spreads to the midline structures of the brain and overgrows large vessels. Nevertheless, the removal of the tumor within the available limits is justified, especially if it is located in functionally less significant areas (in the right temporal lobe, the pole of the right frontal lobe). A somewhat more lasting clinical effect can be obtained with combined treatment (tumor removal, radiation and chemotherapy).

The question of the indication for surgery for glioblastomas is decided strictly individually, depending on the age, the severity of the patient's condition and the location of the tumor.

1.1.3. Intraventricular tumors

Ventricular tumors are divided into primary, arising from the vascular plexuses and walls of the ventricles, and secondary - these are tumors of nearby structures that grow into the cavity of the ventricles.

Primary ventricular tumors include ependymomas, choroid papillomas, meningiomas. The primary tumors of the third ventricle also include the so-called colloid cysts and some types of craniopharyngiomas that develop in the bottom of the third ventricle, and ectopic tumors. Most of these tumors are fairly well demarcated and can be radically removed.

Approaches to ventricular tumors are determined by their localization within the ventricular system. Tumors of the anterior and middle parts of the lateral ventricles are removed using access through the premotor cortex of the frontal lobe or by dissecting the corpus callosum . When the tumor is located in the area of the ventricular body, a posterior temporal or parietal approach is used.

From a technical point of view, it is most difficult to remove tumors of the third ventricle. Various approaches have been proposed , but transcallosal approaches are more preferable - removal of the tumor through the interventricular (Monroe's) opening and access through the anterior wall of the third ventricle - the terminal plate.

The choice of access for secondary ventricular tumors can primarily be determined by the location of its extraventricular part.

1.2. Tumors of the chiasmatic-sellar region

Most of the neoplasms localized in the area of the sella turcica and optic mountains of the chiasm (chiasm) are extracerebral tumors: the already mentioned meningiomas of the sella turcica , pituitary tumors, tumors of a dysembryogenetic nature - craniopharyngiomas, cholesteatomas, etc.

A special group is made up **of pituitary tumors** . In turn, they can be subdivided into *hormone-active* and *hormone-inactive* tumors.

The symptom complex that develops with these tumors is very characteristic. It consists of symptoms of dysfunction of the pituitary gland (its hyper- or hypofunction), decreased vision due to compression of the optic nerves and optic chiasm. Large tumors with pronounced intracranial growth can affect the hypothalamic parts of the brain and even disrupt the outflow of cerebrospinal fluid from the ventricular system, causing compression of the third ventricle.

Hormone-active tumors of the pituitary gland rarely reach a large size, since they cause characteristic endocrine symptoms that contribute to their early recognition.

Depending on the type of endocrine active cells from which the tumor is formed, prolactin-secreting adenomas are distinguished; growth hormone producing adenomas; ACTH-secreting and some other tumors.

Prolactin-secreting adenomas (prolactinomas) cause lactorrhea. violation of the menstrual cycle and some other symptoms.

Growth hormone-producing adenomas at a young age are the cause of gigantism, and in adult patients they cause characteristic symptoms of acromegaly: an increase in the size of the hands, feet, coarseness of facial features, and enlargement of internal organs.

With *ACTH-secreting adenomas* , Cushing's syndrome develops: increased blood pressure, characteristic fat deposits on the trunk, striae gravidarum, hirsutism.

Many of these tumors are detected in the initial stage, when their size does not exceed a few millimeters, they are completely located within the Turkish saddle - these are microadenomas.

With hormone-inactive adenomas that compress the pituitary gland, symptoms of panhypopituitarism are noted (obesity, decreased sexual function, decreased performance, pallor of the skin, low blood pressure, etc.). Often these tumors are almost asymptomatic until they grow far beyond the Turkish saddle and cause decreased vision.

Complex methods (. Radiography, computed tomography, MRI level of various hormones) allows to determine the form of the pituitary tumor, its size and the direction of growth One of the most common diagnostic signs - ballonovidnos extension Turkish saddle, which is easily detected at craniography, CT and MRI studies ...

Treatment. The growth of small prolactin-secreting pituitary tumors can be halted with dopamine agonist drugs (bromocriptine).

In most cases, surgical removal of the pituitary tumor is the most reasonable treatment. Small pituitary tumors, predominantly located in the sella turcica, or tumors with moderate suprasellar growth, are usually removed using a transnasal transsphenoidal approach.

The sphenoid sinus is opened through the nose, the upper wall of which is the bottom of the sella turcica. Under a microscope, it is removed to differentiate the tumor from normal pituitary tissue and radically remove it. At the same time, X-ray control is carried out , which makes it possible to determine the depth of penetration of instruments into the cranial cavity and the radicality of tumor removal.

Pituitary adenomas with pronounced supra- and parasellar growth are removed using the frontal or frontotemporal approach.

By lifting the frontal lobe, the surgeon reaches the area of the optic chiasm. The optic nerves and chiasm are usually sharply displaced by a tumor emerging from the sella turcica. The adenoma capsule is opened between the optic nerves and the tumor is removed intracapsularly with a surgical spoon and by aspiration. When the tumor spreads parasellarly into the cavernous sinus or retrosellarly into the cisterna cisternae, the operation becomes difficult and risky, primarily due to the tumor overgrowth of the carotid artery and its branches.

If the tumor is partially removed, it is advisable to carry out radiation therapy. Irradiation is also indicated for recurrent tumor growth.

Craniopharyngiomas are dysembryogenetic tumors that are more common in childhood and adolescence. The emergence of these tumors is associated with incomplete reverse development of the so-called Rathke's pocket - the outgrowth of the embryonic pharyngeal epithelium, which takes part in the formation of the anterior pituitary gland. These tumors can develop in the Turkish saddle and beyond - in the region of the pituitary gland and the bottom of the third ventricle. Tumors consist of dense tissue, often containing calcified areas -

petrification, and cystic cavities. Cysts contain a brown or xanthochromic fluid rich in cholesterol, they are often multiple and can be gigantic.

Clinical manifestations. For craniopharyngiomas, *endocrine disorders are* very characteristic , growth retardation (nanism), obesity, menstrual irregularities, diabetes insipidus, lethargy, weakness, in severe cases - exhaustion, cachexia; *visual impairment* , up to blindness; *intracranial hypertension* ; this syndrome occurs when tumors penetrate into III
ventricle and squeezing interventricular (Monroe) openings. The characteristic manifestations of craniopharyngiomas also include the presence of petrificates over the Turkish saddle (they are found on X-ray of the skull or computed tomography).

Treatment. A temporary effect can be achieved by puncturing the cysts and emptying their contents or by draining the ventricles in case of hydrocephalus caused by compression of the third ventricle.
The generally accepted method is radical removal of the tumor.

Craniopharyngiomas, mainly located in the cavity of the sella turcica, are removed in the same way as pituitary tumors, using transsphenoidal or subfrontal approaches.

Removal of craniopharyngiomas located in the cavity of the third ventricle, behind the optic chiasm, is one of the most complex neurosurgical interventions. The complexity is due to the deep penetration of the tumor into the hypothalamic region, where the most important centers of autonomic and endocrine regulation are located, the common blood supply to the tumor and the hypothalamus, and the frequent inclusion of large vessels in the tumor capsule.

To achieve a radical removal of such tumors, it is necessary to use combined approaches: basal-frontal or frontal-temporal one- and two-sided in combination with transcallosis access to the upper sections of the tumor located in the third ventricle.

No less difficulties arise when removing giant cystic craniopharyngiomas, which overgrow the large vessels of the base of the brain, cranial nerves and protrude deeply into the basal surface of the brain. For treatment, intratumoral administration of radioactive drugs (radioactive iodine) or oncostatics is also used, leading to the death of cells that produce cystic fluid, to a decrease and obliteration of cysts.

It should always be remembered that patients with craniopharyngiomas have severe endocrine insufficiency, which requires constant correction both before the operation and especially after it.

In the chiasmal-sellar region, there are other dysembryogenetic tumors - epidermoid cysts (cholesteatomas, consisting of masses of deflated epithelium), dermoid cysts, consisting of skin derivatives, and mature teratomas, in which formed teeth and even fragments of jaw bones can be found .

In this area, meningiomas occur relatively often, compressing the optic nerves and optic chiasm.

Turkish saddle tubercle meningiomas. Their main manifestation and practically the only symptom is a progressive decrease in vision.

When removing meningiomas of the sella turcica tubercle, a certain sequence of actions is important : removal of the anterior part of the tumor, its maximum separation by means of coagulation from the site of attachment, after removal of the central part of the tumor, careful separation of its outer and posterior sections from the optic nerves, optic chiasm and the pituitary stalk. The greatest difficulties are associated with the isolation of the carotid and anterior cerebral arteries, which may be located in the tumor tissue.

Tumors of the **optic nerves and optic chiasm are** especially common in childhood. They are characterized by a progressive decrease in vision up to complete blindness.

Indications for tumor removal arise when these formations cause compression of the adjacent parts of the brain and the remaining intact visual pathways.

1.3. Tumors of the posterior cranial fossa

Tumors of the posterior cranial fossa can be both intra- and extracerebral. For intracerebral tumors include cerebellar and IV ventricle, primary tumors of the brain stem.

Among the extracerebral, the most common neuromas of the VIII pair of cranial nerves, meningiomas.

Some extracerebral tumors of the posterior cranial fossa mainly affect the bones of the base and are located extradurally.

Tumors of the cerebellum and IV ventricle. These tumors can be both benign (astrocytomas, characterized by slow growth), and malignant, infiltrative growing (medulloblastomas). And astrocytomas, and especially medulloblastomas, are more common in childhood.

Cerebellar tumors often affect the worm, fill the cavity of the IV ventricle and compress the brain stem. In this regard, the symptomatology is caused not so much (and often not only) by damage to the nuclei and pathways of the cerebellum, as by compression of the brain stem.

A feature of cerebellar tumors is that they often lead to a violation of the outflow of cerebrospinal fluid, closing the exit from the IV ventricle or squeezing the cerebral aqueduct.

Hydrocephalus of the lateral and third ventricles, rapidly growing with acute occlusion, leads to dislocation of the brain with the danger of acute infringement of the brain stem in the area of the tentorial foramen.

By itself, a tumor that develops in the cerebellum leads to an increase in its volume and can cause wedging in both the tentorial and occipital foramen.

The initial symptoms of a cerebellar tumor are often impaired coordination, ataxia, adiadochokinesis, and decreased muscle tone. Early, especially with cystic or rapidly growing tumors, symptoms of compression of the structures of the bottom of the IV ventricle may appear: nystagmus (usually horizontal), bulbar disorders, vomiting and hiccups. With the development of an infringement of the brain stem in the occipital foramen, breathing disorders occur until it stops, a violation of cardiovascular activity: bradycardia, an increase in blood pressure , followed by its fall.

Cerebellar astrocytomas, in contrast to hemispheric astrocytomas, can be well demarcated from the surrounding cerebellar tissue and contain

cysts. Histologically, these tumors belong to the most benign type of pilocytic astrocytomas, which occur mainly in childhood.

Computed tomography and MRI scan reveal tumors with clear contours and cysts contained in them.

These tumors can be radically removed along the border with the cerebellar tissue, the CPT is compressed , but not germinated by the tumor. Operations can lead to complete recovery of the patient or long-term, many years of remission.

Along with this, there are infiltratively growing cerebellar tumors, some of which grow into the brain stem.

On a computed tomogram, the tumor is indistinct, blurred outlines. In these cases, only partial resection of that part of the tumor, which in its structure is most different from the normal cerebellar tissue , is possible .

Removal of cerebellar astrocytoma, as well as other tumors, is carried out by trepanation of the posterior fossa, usually using a midline soft tissue incision in the cervico-occipital region.

Hemangioblastomas (angioreticulomas) *are* richly vascularized tumors, often leading to cyst formation (in 70% of cases). Most of the hemangioblastomas are located in the hemispheres of the cerebellum or vermis. Occasionally, the tumor is located in the medulla oblongata and pons. Hemangioblastomas can also affect the spinal cord. Most often, hemangioblastomas develop at the age of 3 (MO years. It should be borne in mind that in about 20% of cases, tumors are multiple and are a manifestation of Hippel-Lindau disease (hereditary autosomal dominant type). In these cases, in addition to tumors of the central nervous system (cerebellum, spinal cord), retinal angiomatosis, tumors and cystic changes in the kidneys and other internal organs, polycythemia are often detected .

With the formation of a cyst, the rapid development of the disease is sometimes noted with the appearance of formidable symptoms of compression of the brain stem.

Treatment. Surgical removal of solitary cerebellar hemangioblastomas in most cases leads to almost complete recovery of patients.

In some cases, the main part of the neoplasm is a cyst, while the tumor itself is negligible and may go unnoticed. In this regard, after emptying the cyst, it is necessary to carefully examine all its walls from the inside in order to detect a tumor, which is distinguished by a bright red color.

Removal of solid tumors, especially those that penetrate into the trunk, is difficult: these tumors are very richly supplied with blood and, if the main sources of blood supply are not turned off at the beginning of the removal , the operation can be very traumatic. In case of illness

Hippel-Lindau disease relapses are possible due to multifocal tumor growth.

Medulloblastomas malignant, rapidly growing tumors occurring

mainly in childhood. Medulloblastomas, localized in the posterior fossa, account for 15-20% of all brain tumors in children. More often, medulloblastoma develops from the worm, fills the IV ventricle, can infiltrate its bottom and grow into the trunk, early leads to a violation of the outflow of cerebrospinal fluid from the IV ventricle and hydrocephalus. Metastasizes to cerebrospinal fluid spaces.

The most common symptoms are headache, vomiting, ataxia in the limbs, unsteadiness of gait, nystagmus. With the germination of the bottom of the IV ventricle, tabloid symptoms, impaired sensitivity on the face, and oculomotor disorders appear . Computed tomography reveals a tumor located in the region of the IV ventricle, worm and medial parts of the cerebellum (it is usually a heterogeneous structure), and signs of hydrocephalic expansion of the lateral and III ventricles.

Treatment. Surgical treatment consists in removing the tumor as completely as possible (only the areas growing into the brain stem are not removed) and restoring normal circulation of cerebrospinal fluid.

The tumor is often soft and is removed by aspiration with conventional or ultrasonic suction. After the operation, the posterior cranial fossa is irradiated in combination with general irradiation of the brain and spinal cord in order to prevent tumor metastasis. A positive result can be obtained from the use of chemotherapy (nitrosourea preparations, vincristine, etc.).

Primary tumors of the IV ventricle (ependymomas, choroid papillomas) are accompanied by symptoms of impaired CSF circulation and compression of the brainstem. Ependymomas can infiltrate the fundus of the IV ventricle, spread into the occipital cistern and craniospinal, which often makes their radical removal impossible. Choroid papillomas, on the other hand, are well-demarcated tumors that can be removed totally.

Patients with tumors of the cerebellum and IV ventricle require special attention, since with these tumors a sharp deterioration is always possible with the development of phenomena of dislocation and suppression of the brain: increased headache, hiccups, vomiting, depression of consciousness, impaired respiration and cardiovascular activity.

The appearance of these symptoms requires urgent measures, first of all, ventricular puncture with the establishment of an external drainage system and resuscitation measures.

Brain stem tumors. Glial tumors of varying degrees of malignancy make up the bulk of the stem tumors .

Most often, stem tumors develop in childhood. They cause damage to both nuclear formations and the pathways of the brain stem. Often, alternating syndromes are detected with a predominance of motor and sensory disorders on the opposite side, and on the side of the predominant location of the tumor , the lesions of the cranial nerves and cerebellar disorders are more pronounced.

Unlike tumors of the cerebellum, tumors of the trunk relatively rarely lead to a violation of the outflow of cerebrospinal fluid from the IV ventricle, therefore hydrocephalus and intracranial hypertension are late symptoms of brain stem tumors, with the exception of those that develop in the midbrain near the sylvian aqueduct.

Benign tumors of the trunk are characterized by slow growth, which can last for years (in some cases, 10-15 years or even more). Malignant, which make up the majority, lead to the death of patients within several months or 1-2 years (the differences are determined by the degree of malignancy).

Tumors can be localized in different parts of the trunk, but more often the bridge grows.

Since the time of R. Virkhov, it was believed that tumors of the trunk diffusely infiltrate all its structures, and, therefore, are inoperable. Recent research has partially changed this concept of stem tumors. In addition to diffusely growing tumors, which, unfortunately, constitute the majority of neoplasms, there are also nodular, fairly well- demarcated, and tumors containing cysts.

The experience of a number of surgeons has shown that benign, delimited tumors of the trunk can be successfully removed. In these cases, the operation can significantly prolong the patient's life and improve his condition.

Such tumors are detected only in 20-25% of patients. In other cases, with diffuse In growing trunk gliomas, radiation therapy may be recommended, the effectiveness of which has not yet been accurately studied.

Neurinoma of the VIII cranial nerve. This is a fairly common (7-10%) intracranial neoplasm. A benign, slowly growing tumor arises from the cells of the Schwann sheath of the vestibular cochlear nerve. Primary symptom. which patients pay attention to - hearing loss, later there are coordination disorders, change in gait, impaired sensitivity or pain in the face, damage to the facial nerve. With large tumors, the outflow of cerebrospinal fluid from the IV ventricle is disturbed, secondary hydrocephalus develops and intracranial pressure increases with edema of the optic disc and decreased vision.

A typical craniographic symptom is enlargement of the internal auditory canal, in which the tumor is located. Computed tomography reveals a well-defined tumor, sometimes containing cysts in the area of the lateral cistern.

Treatment. Timely microsurgical technique allows, in most cases, to achieve successful removal of the neuroma. The approach to the tumor is most often carried out from the side of the posterior cranial fossa. The soft tissues are dissected with a vertical incision, posterior to the mastoid process (the so-called paramedian incision), the scales of the occipital bone and partly the mastoid process are resected up to the sigmoid sinus. The cerebellar hemisphere is displaced medially. To reduce the tension of the brain, the arachnoid membrane of the brain is opened near the IX and X pairs of cranial nerves. Cerebrospinal fluid is excreted. These nerves are isolated from the lower pole of the tumor. Initially, intracapsular removal of the tumor is performed .

This is easier to do with ultrasonic suction. Then trepanation of the posterior wall of the internal auditory canal is performed - the site of the initial tumor growth, as well as the facial nerve, is exposed . Further removal of the tumor is done with extreme caution so as not to damage the facial nerve, which can be sharply thinned, difficult to differentiate in the tumor capsule. The tumor capsule is sequentially separated from the medial parts of the cerebellum, VII nerve, brainstem, trigeminal nerve. It is very important to preserve all the vessels supplying the trunk, some of which pass in the tumor capsule. Particularly noteworthy is the anterior inferior cerebellar artery, which forms a loop on the tumor capsule and gives off a branch that goes into the internal auditory canal. Microsurgical technique allows in most cases to completely remove

the tumor, while preserving the facial nerve. Only with small tumors and intact hearing is it possible to remove the tumor while preserving the auditory nerve.

In elderly people and patients in serious condition, it is advisable to limit ourselves to partial intracapsular removal of the tumor.

Along with the described technique, in recent years, a translabyrinth approach has been widely used , which, however, requires special skills in operations on the temporal bone pyramid .

Posterior cranial fossa meningiomas. These tumors can develop from the dura mater of the convexital surface of the cerebellum, the tentorium of the cerebellum, in the area of the pyramid of the temporal bone, clivus, foramen magnum .

Meningiomas in the area of the surface of the cerebellum and tentorium are mainly manifested by symptoms of increasing intracranial hypertension and mild cerebellar symptoms. Basally located meningiomas cause damage to the corresponding localization of the tumor of the cranial nerves and symptoms of compression of the trunk. Radical removal of meningiomas of the posterior surface of the cerebellum and the tentorium of the cerebellum usually does not present significant difficulties. On the contrary, the removal of meningiomas in the area of the pyramid and clivus is a technically very difficult task, the solution of which requires the use of special approaches with resection of the basal parts of the skull, including the pyramid. If the tumor is closely fused with the brain stem, then you have to limit yourself to its partial removal.

A difficult task is the treatment of patients with extracerebral tumors developing in the region of the base of the skull. This group includes chordomas, glomus and some other tumors. These tumors can spread supratentorially in the area of the cavernous sinus and the lower wall of the middle cranial fossa.

Chordomas develop from the remains of the primary chord, they destroy the bones of the skull base in the clivus, *sella turcica* , affect the cranial nerves and compress the brain stem.

Tumors often spread to the nasopharynx.

Only partial removal of chordomas is possible. In case of recurrent tumors, patients are operated on again. To approach the chordomas, it is necessary to use different approaches: from the side of the posterior or middle cranial fossa, transoral, and some others.

Glomus tumors of the bulb of the jugular vein. These tumors develop from the so -called glomus bodies, more often in the area of the bulb of the jugular vein. They infiltrate the pyramid of the temporal bone, cause damage to the VII, VIII, IX, XI, XII pairs of cranial nerves. The initial symptoms are pulsating noise in the ear, hearing loss, dizziness, followed by ataxia and symptoms of damage to the nerves passing through the jugular foramen.

Tumors grow along the vessels, can spread into the cavernous sinus, and not very often - along the jugular vein. Glomus tumors are very richly supplied with blood from the branches of the external and internal carotid arteries. They can invade the eardrum and cause dangerous external bleeding.

On examination, a bright red swelling is visible in the ear canal.

Treatment. Radical removal is difficult. A wide resection of the pyramid, isolation of the tumor on the neck is required . To reduce bleeding before surgery, embolization of the adductor vessels is often performed.

In some cases, preference is given to embolization of the tumor vessels with its subsequent irradiation.

Neurinoma of the V cranial nerve. This tumor, which develops from the roots of the trigeminal nerve, can be attributed to neoplasms that can simultaneously be located in the posterior cranial fossa (subtentorial) and above the tentorium of the cerebellum.

These tumors often reach very large sizes, destroy the bones of the base of the skull, grow into the cavernous sinus, and deform the brain stem. They are manifested by impaired sensitivity on the face, damage to the oculomotor nerves, symptoms of compression of the brain stem. Their removal requires modern basal approaches from the side of the middle or posterior cranial fossa, transection of the tentorium of the cerebellum, and careful dissection of the tumor near the brain stem.

1.4. Metastatic tumors

Metastatic tumors are common (30-50%) brain tumors. Cerebral metastases are found in 15-20% of cancer deaths.

There is a tendency towards an increase in the number of metastatic brain tumors, which is associated with an increase in the life expectancy of patients with cancer. Also significant is the fact that not all chemotherapy drugs, which are increasingly important in the treatment of cancer patients, penetrate the blood-brain barrier and, therefore, can prevent tumor metastasis to the brain. In addition, the methods for recognizing metastases have become much more sophisticated.

The most common cause of metastases to the brain is lung cancer (more than 40%), followed by the frequency of metastases to the brain - breast cancer and kidney cancer.

Up to 80% of metastases are localized in the cerebral hemispheres, about 15% in the cerebellum.

Clinical manifestations. Brain metastases are characterized by a rather acute onset, headache, the appearance of symptoms of local brain damage (paresis of the limbs, impaired sensitivity, speech), which are steadily progressing. Recognition of cerebral metastases, of course, is facilitated by anamnestic data on the primary oncological disease. However, it should be borne in mind that in 15% of cases, the primary lesion, which was the cause of metastasis, cannot be detected.

From the point of view of possible surgical treatment, it is very important to find out whether the patient has a single brain metastasis or multiple metastases. On computed tomograms, metastases look like rounded formations of increased density with enlightenment in the center (ring-like). This form is explained by the frequent necrosis of the central part of the tumor. It is not so easy to identify small metastases. They can be suspected by concomitant cerebral edema, which is usually pronounced with metastases .

Gamma encephalography and magnetic resonance imaging may be more informative.

tomography.

Treatment. Initially, steroid drugs (dexamethasone) are prescribed, which, having a pronounced anti-edema effect, can contribute to a rapid improvement in the patient's condition (headache passes, the severity of movement disorders decreases, etc.).

With single megastases and a relatively satisfactory general condition of the patient, surgical removal of the tumor is advisable. It may be justified in some cases to remove several nodes, if they are located in the same area of the brain.With a subcortical location, it is very important to have an accurate idea of the location of the metastatic node in the brain, otherwise the operation may be unjustifiably traumatic. For this purpose, such methods of intraoperative localization of a tumor as ultrasound scanning or the use of a radioisotope method can be applied. Removal of deep metastases can be successfully performed using stereotaxic access.

For multiple metastases, whole-brain irradiation may be recommended.

Life expectancy is often determined by the general condition of the patient and the dissemination of the tumor to other organs.

With single metastases and a successfully removed primary focus, in some cases , a long-term improvement in the patient's condition can be achieved.

1.5. Tumors of the bones of the skull

Tumors of the skull can cause compression of the brain and the appearance of neurological symptoms, primarily local headache and symptoms of brain irritation.

Osteomas. These are benign, slow-growing tumors. The indications for their removal arise when tumors reach a significant size and begin to compress the brain or when they are located in the paranasal sinuses.

Osteomas of this localization can cause inflammatory complications - osteomyelitis, abscesses.

Hemangioma. This is a benign tumor, which is more often localized in the parietal and frontal bones. On craniograms, rounded formations with a characteristic honeycomb structure or trabecular structure are determined . The operation is indicated when neurological symptoms appear.

Epidermoids and dermoids. They look like intraosseous cystic formations with compacted walls. Surgical removal is indicated.

Tumors that destroy the base of the skull (chordomas, glomus tumors; have already been described.

Malignant tumors of the base of the skull include chondro- and osteosarcomas and some others. Their treatment is ineffective and usually consists of partial removal in combination with radiation and chemotherapy.

Pseudotumors of the brain (benign intracranial hypertension). The disease is characterized by a persistent increase in intracranial pressure, accompanied by nausea, dizziness, sometimes double vision and congestion in the fundus. The most dangerous manifestation is a progressive decrease in vision, in some cases - to complete blindness.

The etiology of this syndrome is not fully understood. It is assumed that it may be based on endocrine-metabolic disorders, since there is a dependence of benign intracranial hypertension, which is more often observed in women, from obesity, menstrual irregularities, and postpartum complications.

With long-term intracranial hypertension, a very careful examination of patients is necessary in order to identify other possible causes of an increase in pressure: chronic inflammatory processes in the membranes (including fungal ones), blood diseases ; violation of venous outflow with sinus thrombosis; the toxic effect of certain drugs, etc.

CT, MRI studies and craniography give negative results, with the exception of indirect signs of intracranial hypertension: collapsed ventricles, hypertensive changes in the bones of the skull.

Treatment. Aimed at normalizing intracranial pressure: limited intake of fluid and salt is recommended , diuretics (furosemide, diamox), indicated steroid therapy.

With a steady decline in vision, they resort to surgical treatment: lumboperitoneal shunting, decompression of the optic nerves.

2. Tumors of the spinal cord. Surgery

Tumors of the spinal cord are found 8-10 times less frequently than tumors of the brain and are observed in patients mainly at the age of 20 to 60 years. Spinal tumors are usually subdivided into *primary* and *secondary.* The group of **primary tumors** includes neoplasms originating from the medulla *(intramedullary* tumors) and developing from the meninges, roots, vessels *(extramedullary* tumors). Extramedullary tumors are much more common than intramedullary tumors. Extramedullary tumors can be either subdural (located under the dura mater) or epidural (outside the dura mater). Most extramedullary tumors are subdural. In rare cases, there are tumors, some of which are located subdurally, and some are located epidurally (subdural-epidural tumors), as well as epidural-extravertebral tumors.

To *extramedullary tumors* spinal cord include: 1) meningioma (arachnoid endothelioma), originating from the meninges; 2) neurinoma, which develops from Schwann cells, predominantly of the posterior roots of the spinal cord, which occur with approximately the same frequency as meningiomas; 3) hemangioblastomas (angioreticulomas) - richly vascularized tumors, which in some cases can be multiple (Hippel-Lindau disease); 4) lipomas, usually in combination with spina bifida or other malformations .

The last two types of tumors are relatively rare.

Intramedullary tumors of the spinal cord are mainly represented by gliomas (astrocytomas and ependymomas). Less commonly, multiforme spongioblastomas, medulloblastomas, and oligodendrogliomas are found. Astrocytomas are characterized by infiltrative growth, localized in the gray matter and are highly spread along the length of the brain. Ependymomas arise most often from the ependyma of the central canal at the level of the cervical and lumbar enlargements. They can also develop from the terminal thread and are located between the roots of the cauda equina.

In addition to primary tumors of the spinal cord, **secondary** tumors are also possible , growing into the spinal canal from the surrounding tissues or metastasizing with primary damage to other organs. Sources of metastases are mainly lung, breast, thyroid and prostate cancer .

The defeat of the spinal cord can also be observed with tumors of the spine - both primary (vertebral hemangiomas, sarcomas) and secondary (metastatic), as well as with volumetric processes located in the epidural space (lymphogranulomatosis).

Neoplasms sharply disrupt the functioning of the spinal cord, squeezing (meningioma, neurinoma), destroying (cancer metastasis) or sprouting (glioma) its substance. Any tumors reduce the space in the spinal canal, disrupt blood and cerebrospinal fluid circulation.

Spinal cord tumors are characterized by the progressive development of the syndrome of transverse spinal cord injury and mechanical blockade of the subarachnoid space. Features of the clinical picture in each case depend on the level of the tumor, its nature and extra- or intramedullary localization.

The syndrome of partial and then complete transverse lesions of the spinal cord in extramedullary tumors is a consequence of its compression. the emergence of functional and dynamic disorders, then irreversible degenerative changes, first in the pathways, and as the tumor pressure increases - in the gray matter. In intramedullary tumors, this syndrome is caused by the destruction or compression of the gray matter in the corresponding segment and the increasing compression from the inside of the white matter of the spinal cord. The development of the syndrome of transverse spinal cord injury to paraplegia can last from several months (for malignant tumors) to 1.5-3 years (for benign tumors).

The second most important sign of a spinal cord tumor is an increase in the blockage of the subarachnoid space. As the tumor grows, it sharply narrows, and then obliterates the subarachnoid space at its location. As a result, the circulation of cerebrospinal fluid stops and stagnant changes develop in it.

Clinical manifestations of extramedullary tumors. During this disease , 3 stages are distinguished: 1) radicular; 2) the stage of half lesion of the spinal cord (Brown-Séquard syndrome); 3) the stage of complete transverse lesions of the spinal cord.

The earliest manifestation of extramedullary tumors is radicular pain and paresthesia caused by irritation of the dorsal root by a tumor growing from its Schwann membrane (neurinoma) or located near the root (meningioma). At first, the pain is one-sided, then often becomes bilateral, which is due to the tension of the roots on the side opposite to the tumor. With tumors in the region of the roots of the cervical and lumbar thickenings, pain spreads longitudinally in the upper and lower extremities; with tumors in the thoracic region, the pain is shrouded in nature. Pain syndrome at the onset of the disease occurs periodically, and later becomes constant, aggravated by coughing, sneezing, sometimes at night, with long lying. Radicular pains are usually long-term, strictly localized, which determines their diagnostic value. In the zone of innervation of the affected roots, sensitivity disorders in the form of hypesthesia are revealed, tendon, periosteal and skin reflexes decrease and fall out, the reflex arcs of which pass through them. Soreness is found with pressure on the spinous processes and paravertebral points at the site of tumor localization. However, more pathognomonic for extramedullary tumors are

increased radicular pain and the appearance of conductive paresthesias during percussion along the spinous process at the tumor level (Razdolsky's symptom).

As the tumor grows, symptoms appear that indicate spinal cord compression. When the tumor is localized on the anterolateral, lateral, posterolateral surfaces of the spinal cord, Brown-Sequard syndrome is formed: on the side of the tumor and below it, deep sensitivity is impaired and central paresis occurs, and on the opposite side, superficial sensitivity is impaired . However, as a result of compression, both halves of the spinal cord suffer to a greater or lesser extent, therefore, usually we are talking about elements of the Brown-Sequard syndrome (central paresis is more pronounced on the side of the tumor, 1 disturbance of superficial sensitivity on the opposite). At the level of spinal cord lesions , segmental disorders can also develop: motor - in the form of atrophy of the corresponding muscles, paresis in them, decreased reflexes, sensitive - in the form of radicular hyperalgesia, paresthesia, hypesthesia.

With further growth, the tumor gives a picture of transverse compression of the spinal cord: lower paraplegia or tetraplegia, bilateral conduction disorders of sensitivity (hypesthesia or anesthesia), dysfunction of the pelvic organs. Decreased strength in the extremities and hypesthesia first appear in the distal extremities and then rise to the level of the affected spinal cord segment.

A characteristic feature of extramedullary tumors of the spinal cord is the early appearance and severity of blockade of the subarachnoid space and changes in cerebrospinal fluid.

Clinical manifestations of intramedullary tumors. The picture is distinguished by the absence of a stage of radicular pain. An early sign is segmental sensory disorders of a dissociated nature. As the tumor grows and the lateral cords of the spinal cord are compressed , conductive bilateral motor and sensory disorders join in the zones located below the segmental "sensory disorders". For conduction hypesthesia with an intramedullary tumor, spreading from top to bottom is characteristic due to the law of the eccentric arrangement of longer conductors in the spinal cord. As a result of the defeat of the anterior and lateral horns, bilateral peripheral paresis occurs , expressed vegetative-trophic disorders. Characterized by the late appearance of signs of blockade of the subarachnoid space, the absence of symptoms of the cerebrospinal fluid and the spinous process of Razdolsky. The course of intramedullary tumors in comparison with extramedullary tumors is faster.

Clinical manifestations of spinal cord tumors at the level of different segments have their own characteristics. Tumors of the *upper cervical level (CI-CIV) are* characterized by pain in the neck and back of the head, tension of the neck muscles, forced position of the head, spastic tetraparesis, conduction disorders of sensitivity. The defeat of the CIV segment is accompanied by
paresis of the diaphragm, which is manifested by hiccups, shortness of breath, difficulty coughing, sneezing.

With tumors at the level of the *cervical thickening,* there are peripheral paresis of the upper extremities in combination with spastic paresis of the lower extremities. In the hands, radicular disorders of sensitivity and pain may appear . Horner's syndrome (ptosis, miosis, enophthalmos) is characteristic of

the lesion of *segments at the level of CVIII-ThI* . Disorders of the functions of the pelvic organs with tumors of cervical localization are usually absent for a long time and have the character of an urgent urge or automatic emptying of the bladder.

Tumors of the *thoracic region* cause conduction disorders of sensitivity, lower spastic paraparesis, and dysfunction of the pelvic organs. The hands remain intact. Radicular pains are of a girdle nature, mimicking diseases of the internal organs. Segmental abnormalities can be manifested by the loss of abdominal reflexes, which helps to establish the level of damage.

With tumors of the *upper lumbar segments* , spastic paresis of the lower extremities is observed in combination with atrophy in their proximal regions, radicular pain in the area of innervation of the femoral nerve.

With tumors of the *epicone* (LIV-SII), radicular pain occurs in the lumbar region, "saddle" hypesthesia, flaccid paresis of the gluteal muscles, muscles of the posterior surface of the thigh, lower leg and foot. Dysfunctions of the sphincters appear early in the form of urinary and fecal incontinence.

Tumors of the *cerebral cone* (SII-SV) are characterized by early and severe dysfunction of the bladder, rectum, and genitals. There are no paralysis of the lower extremities, tendon reflexes are preserved. In the crotch region there are sensory disorders of a dissociated nature in the form of "rider pants". Large pressure ulcers often appear in the lumbosacral region.

Tumors of the *cauda equina* are manifested by intense radicular pain radiating to the buttock, leg, aggravated in the supine position. First, unilateral, then bilateral pains appear . There are asymmetric radicular sensory disorders. Movement disorders occur in the form of flaccid paresis and paralysis in the distal parts of the lower extremities; trophic disorders are also found there. Pelvic disorders are expressed in urinary retention. Tumors of the cauda equina (neurinomas) develop slowly and, due to the vastness of the subdural space and displacement of the roots, can reach large sizes before causing gross spinal disorders. The so-called implantation cholesteatomas - epidermal tumors that develop from elements of the epidermis brought into the subarachnoid space during repeated lumbar punctures - are also accompanied by clinical symptoms of lesions of the cauda equina .

Laboratory and functional research data. In the diagnosis of spinal cord tumors, the study of cerebrospinal fluid and CSF dynamic tests are of great importance . A spinal cord tumor is characterized by an increase in the protein content in the cerebrospinal fluid with a normal number of cells (protein-cell dissociation). In some tumors (cauda equina neurinoma, filamentous ependymoma) , a particularly high protein content and spontaneous coagulation in a test tube are found. With the development of reactive arachnoiditis near the tumor in the cerebrospinal fluid , a slight pleocytosis (20-40 cells) may appear, which is also likely with subdural malignant neoplasms. Often, the cerebrospinal fluid is xanthochromic due to hemolysis of erythrocytes that enter it due to compression of the veins of the spinal cord or from the vessels of the tumor itself.

CSF dynamic tests help to reveal a partial or complete blockage of the subarachnoid space : an artificial increase in the pressure of the cerebrospinal

fluid above the tumor by compressing the vessels of the neck (Kvekenstedt's test), tilting the head forward (Pussep's test), pressing on the abdominal region (Stukey's test). The degree and rate 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 subarachnoil space. A complete block is also characterized by a rapid and sharp (to zero) pressure drop when a small amount of cerebrospinal fluid is withdrawn . During CSF dynamic tests, a symptom of Razdolsky's CSF impulse (increased pain in the area of the affected root) and conductive paresthesias may appear.After a lumbar puncture, a wedge syndrome may be detected
(a sharp increase in conduction disorders up to the development of complete transverse compression of the spinal cord), which is based on an increase in the pressure of the tumor displaced in the distal direction on the lower parts of the spinal cord. The symptom of the cerebrospinal fluid impulse and the wedging syndrome are also of great diagnostic value, being pathognomonic for tumors of the spinal cord, especially extramedullary ones.

Due to possible complications, lumbar puncture should be performed with great care and according to strict indications. When using magnetic resonance imaging to diagnose a tumor, there is practically no need for a spinal puncture and performing CSFD tests.

If a spinal cord tumor is suspected, the examination of the patient must necessarily begin with an X-ray of the spine in order to exclude its diseases that can lead to compression of the spinal cord, as well as in order to identify bone changes inherent in spinal tumors. The latter include the expansion of the intervertebral foramen in epidural-extravertebral tumors, as well as atrophy of the roots of the arches and an increase in the distance between them (Elsberg-Dyck symptom). Often, radiological changes in metastases in the spine appear later than clinical manifestations, in particular radicular pain, and are found only at a later stage.

To determine the block of the subarachnoid space and the level of the tumor, contrast myelography is shown .

Diagnostics and differential diagnosis. The most accurate information can be obtained with computed and especially magnetic resonance imaging.

The diagnosis of a spinal tumor is based on the presence of symptoms of a progressive transverse lesion of the spinal cord, a block of the subarachnoid space, and characteristic changes in the cerebrospinal fluid. When making a diagnosis, it is necessary to determine the localization of the tumor, its intra- or extramedullary location, primary or secondary nature.

In topical diagnostics, the upper border of the tumor can be used to determine the localization of radicular pain and sensory disorders, symptoms of the spinous process and cerebrospinal fluid , persistent conduction disturbances of sensitivity. It should be borne in mind that the level of hypesthesia is usually below the tumor, firstly, due to the eccentric arrangement of the pathways of superficial sensitivity in the spinal cord and, secondly, because the fibers that make up the pathways of surface sensitivity pass through 2-3 segments on their side; in addition, there is an "overlap" of adjacent segments. Therefore, the upper border of

the tumor is localized 2-3 segments above the border of anesthesia. The lower border of the tumor is much more difficult to determine . The decrease in tendon and periosteal reflexes, the arcs of which pass through the compressed segments, as well as the level of protective reflexes, are important . The descending and ascending myelography, made according to the indications, allows to determine the upper and lower borders of the tumor.

Determining the nature of the tumor (primary or secondary) is important for prognosis and treatment .

The main clinical sign of metastases of malignant tumors in the spine are pains that do not disappear at rest and in any forced position of the patient, resistant to treatment.

Neuralgic pains are in the nature of secondary radiculitis without deep sensory disturbances and movement disorders, at least until the moment of a compression fracture or compression of the spinal cord by a growing tumor. Metastases are usually localized in the spinal column. The development of cerebrospinal symptoms often occurs rapidly against a background of severe previous pain. History may include indications of 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 (for example, myelomatosis) is usually made by x-ray and confirmed by appropriate laboratory tests. Secondary tumors are always malignant and within a year or even several months can lead to the syndrome of complete transverse spinal cord injury. Secondary tumors are usually located extradurally.

In the diagnosis of secondary tumors of the spinal cord, a detailed history, a thorough examination of internal organs, repeated clinical blood tests, and especially
X-ray of the spine.

Differential diagnosis of a spinal cord tumor depends on the stage of the process. Differentiation of neuromas and meningiomas in the radicular stage follows from diseases of the internal organs (pleurisy, duodenal and stomach ulcers, cholecystitis, nephrolithiasis, etc.), as well as from radicular syndromes of osteochondrosis. Tumors of the spinal cord, giving a clinical picture of the growing transverse lesion of the spinal cord, are differentiated from the spinal form of multiple sclerosis.

Acute myelitis or epiduritis is usually suspected in malignant tumors that impair the spinal circulation. For a correct diagnosis , a thorough study of the history (presence of infection), the onset of the disease (prodromal period with general infectious symptoms, fever), examination of cerebrospinal fluid (pleocytosis) are important .

Differentiating intramedullary tumor from syringomyelia is quite difficult. It should be borne in mind a slower (years) increase in spinal symptoms in syringomyelia, especially pronounced trophic disorders with less significant lower spastic paraparesis and pelvic disorders, dysraphic status, no signs of spinal cord compression and changes in cerebrospinal fluid.

Spinal cord tumors are differentiated from other diseases that also cause spinal cord compression, for example, from tuberculous spondylitis, which is characterized

by local tenderness of the affected vertebrae, pain under axial load, deformity of the spine and limitation of its mobility, the presence of cold abscesses - swelling near the affected vertebra, change in shape or destruction of the vertebra, detected on x-ray.

Discogenic myelopathy differs from a spinal tumor in a very slow involvement of the spinal cord in the process. MR imaging and contrast myelography are of decisive importance in the diagnosis of vertebral myelopathy .

Surgery. The only radical method for the treatment of a significant part of spinal cord tumors is their surgical removal. The effectiveness of the operation is primarily determined by the nature of the tumor.

Benign extramedullary tumors (meningiomas, neuromas) can be radically removed with a favorable outcome for the patient, if the operation is performed at that stage of the disease, while irreversible damage to the spinal cord has not yet developed.

To remove the tumor, according to the level of its location, a laminectomy is performed .

Neurinomas that develop from the spinal root have an ovoid shape, a pronounced capsule, and are well delimited from the spinal cord. With large tumors, it is advisable to initially enucleate the tumor to reduce its size, and then, with less trauma, separate it from the spinal cord. The spinal root, on which the tumor has formed, is coagulated and transected, after which the entire tumor is removed. Removal of tumors extending along the root extradurally and beyond the spinal canal is very difficult .

These tumors consist of two parts (intra- and extradural) and are shaped like an hourglass. The extradural and especially the extravertebral part can become large, causing destruction of the vertebrae and forming large nodes in the chest or abdominal cavity.

In some cases, to remove these tumors, it is necessary to use combined approaches both from the side of the spinal canal and from the side of the thoracic or abdominal cavity.

Meningiomas often develop from the dura mater of the lateral and anterior surfaces of the spinal canal; they can be very dense and have a wide area of attachment. Removal of these tumors should be done with the utmost care to avoid injury to the spinal cord and its vessels.

The use of an ultrasonic aspirator and laser allows these tumors to be removed with minimal trauma. To prevent relapses, the dura mater, from which the tumor has developed, must be excised or carefully coagulated. A defect in the membrane that has arisen after removal of the tumor can be closed with a canned membrane or fascia.

Intramedullary tumors , more often astrocytomas, do not have clear boundaries with the spinal cord and are significantly widespread along its length, therefore, the possibility of their successful
removals are sharply limited. Nevertheless, modern neurosurgical techniques make it possible to achieve a sufficiently radical removal of these tumors in some cases, especially in tumors containing cysts.

Indications for such operations are strictly individual and are made on the basis of a thorough analysis of the clinical picture of the disease and an assessment of the data of magnetic resonance imaging. The operation consists in exposing the tumor by cutting the posterior surface of the spinal cord along the midline (or above the site of the preferred location of the tumor), emptying the cyst and partially removing the tumor in order to decompress and restore CSF circulation.

Carrying out such an operation creates the best conditions for radiation and chemotherapy in those cases when it is indicated.

Ependymomas develop in the region of the central canal and differ in that they sometimes have a fairly clear border with the brain, the structures of which they seem to push apart.

These features make it possible to achieve, in a number of cases, a very radical removal of the central drip by the epidymus.

Above the location of the tumor, an incision of the spinal cord is made along the posterior commissure. The posterior surface of the tumor is exposed. The volume of the tumor decreases after removing its central part, after which it is possible to separate it from the adjacent brain structures, coagulate and cross the vessels suitable for the tumor, and remove the tumor in whole or in most of it. Technically, it is simpler to remove ependymomas developing from the terminal filament and located in the region of the cauda equina roots.

It should be emphasized that the removal of a spinal cord tumor requires the mandatory use of microsurgical techniques.

With tumors affecting the spine, it is possible to remove them with partial or complete resection of the affected vertebrae, replacing them with a graft and appropriate stabilization of the spine.

For malignant tumors of the spinal cord, radiation therapy and the use of chemotherapy drugs in combination with hormone therapy are indicated .

Regardless of the surgical treatment, patients with spinal cord compression require careful care due to the tendency to form pressure ulcers and the development of an ascending urogenic infection.

Forecast and working capacity. The prognosis is determined by the degree of malignancy and localization of the tumor. Benign tumors usually develop slowly (1.5-2.5 years). After the operation, especially in the early stages, work capacity is restored in 60-80% of patients . With intramedullary tumors, the prognosis for recovery is much worse. The prognosis is poor for metastatic tumors.

Situational tasks:

1: 55 years old, retired. Complains of headache, mainly frontal-occipital localization. A year ago, he underwent radical surgery for bladder cancer, after which he felt satisfactory. He fell ill about a month ago when he began to notice staggering while walking. Gradually, the indicated phenomena increased, practically could not walk due to imbalance, speech became indistinct. Hospitalized in the neurological department. On examination: restless, with difficulty finding a place in bed. He takes a forced position - lying down, since he cannot walk or sit - loses his balance and falls. In the neurological status: the level of consciousness is mild stunning. The patient is communicative, critical, oriented correctly. Moderate meningeal symptoms in the form of a mild Kernig symptom on both sides. There are no

paresis of the limbs. Muscle tone in the limbs is diffusely reduced, more on the left. Anisoreflexia is not clearly detected. Bilateral Babinsky symptom. Rough trunk ataxia, ataxia in the limbs, more on the left. There are no sensitive violations. The speech is dysarthric, with elements of chanting. The patient underwent MRI of the brain. On the obtained MRI scans in the T2 mode in the projection of the left hemisphere of the cerebellum, a rounded zone of the heterogeneous MR signal (with areas of hyperintense and hypointense signal) is determined , signs of the volumetric effect of this formation on the structures of the brain stem are revealed. When examining organs and systems, no other pathology was revealed.
Make a diagnosis, determine the tactics of patient management.

No. 2: A 65-year-old patient was admitted to the clinic with complaints of headaches, weakness, rapid fatigability. According to relatives, over the past several months, the patient's memory began to decline, he became indifferent to what was happening, apathetic. A few days before hospitalization, relatives noted that the patient developed weakness in the left extremities, which the patient himself did not notice. Examination reveals a left-sided homonymous hemianopsia, mild left-sided hemiparesis, impairments of all types of sensitivity on the left. The patient is disoriented in place and time, confabulation is revealed. Memorization of current events is grossly impaired, the patient does not always correctly name the names of relatives, cannot remember the name and patronymic of the doctor, although he himself can assess his memory as satisfactory. When performing voluntary movements, he does not use his left limbs, as if ignoring them. The proposed texts are read from the middle of the page. Does not notice objects located to his left. The patient underwent computed tomography of the brain with contrast enhancement, on which in the right parieto-occipital region to reveal a large formation adjacent to the meninges, with clear, uneven contours, intensively accumulating contrast agent. Multiple hypertrophied vessels, apparently from the systems of the right middle cerebral and posterior cerebral arteries, "fit" to the formation. The formation is accompanied by massive perifocal edema, extending to the temporal and frontal regions of the right hemisphere.
Make a diagnosis, determine the tactics of patient management.

3: 58 years old. He was admitted to the neurological department in a planned manner with complaints of staggering while walking, impaired coordination of movements. From the anamnesis it is known that since 1978 the patient began to be bothered by a persistent cough, was observed by a therapist for chronic bronchitis. However, despite the treatment, the cough continued to bother me, shortness of breath, more expiratory in nature, asthmatic syndrome gradually joined. He underwent X-ray examination several times. A chest X-ray from 1985 reveals multiple confluent foci infiltrates in both lungs, more in the hilar regions. Severe pneumosclerosis. Consulted by a phthisiatrician, a differential diagnosis was made between tuberculosis and sarcoidosis of the lungs (more data are in favor of the latter). Several times he underwent courses of inpatient treatment in the pulmonary department, received courses of hormonal therapy (solumedrol intravenous cap., Later on prednisolone per os). The present deterioration of the patient's condition has been noted during the last two years, when unsteadiness

appeared when walking, ataxia of the trunk and extremities appeared, and speech became indistinct. Also, the patient's memory deteriorated, changes appeared in the psycho-emotional sphere (became irritable, conflicts). On examination upon admission with a neurological status, there were no cerebral and meningeal symptoms. From the pathology of the cranial innervation, a horizontal medium-sweeping

Control questions on the topic: Tumors of the nervous system.

1. Prevalence of tumors of the nervous system.
2. Histological classification.
3. Classification of tumors according to the localization of the initial growth.
4. Features of tumors of the nervous system downstream.
5. The main syndromes of the clinical picture of tumors of intracranial tumors.
6. Diagnostic methods, research objectives, contraindications.
7. Selar tumors. Types, clinic, diagnostics and treatment methods.
8. Tumors of parosellar localization. The predominance of what syndrome and its constituent symptoms.
9. Tumors of the posterior cranial fossa. Clinic (main common syndromes, their etiology).
10. Tumors of the brain stem. The prevalence of which syndrome is most common.
11. Metastatic tumors. Types, prevalence, course features, treatment.
12. Tumors of the spinal cord. Localization classification.
13. Diagnostics, clinical presentation, indications for surgical treatment.
14. Features of the clinical picture by stages of the course of the disease.
15. Methods of treatment, substantiation of the method of choice. Forecast.