NEUROSURGERY

STUDY GUIDE

Edited by
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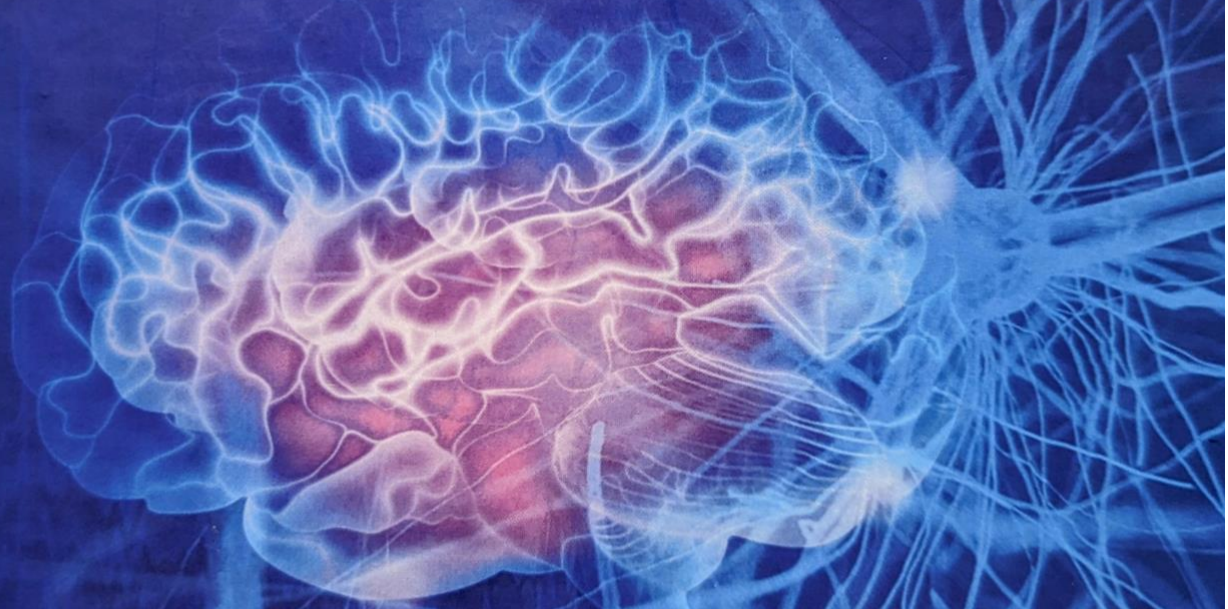

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The study guide is compiled in accordance with the curriculum in neurosurgery. The basics of diagnosis and treatment of patients with the most common neurological diseases of the brain and spinal cord, spine and its structures, peripheral nerves are highlighted. The publication uses modern European and American protocols for the management of such patients. Each section contains a list of recommended reading.

For students of higher medical education establishment, interns.

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Навчальний посібник складено відповідно до навчальної програми з діаг-ностики і лікування пацієнтів із неврологічними захворюваннями головного й спинного мозку, хребта та його структур, периферійних нервів. У виданні використано сучасні європейські й американські протоколи ведення таких хворих. В кінці посібника подано список рекомендованої літератури.

Для студентів медичних закладів вищої освіти.

Contents

Topic 1

TRAUMATIC BRAIN INJURY

Traumatic brain injury is the most common and severe type of trauma that is the main cause of death for victims aged 20—40. Skull and brain injuries constitute over one-third of injuries and rank the top among the reasons of lethality and disability of the victims.The number of disabled people due to brain damage in the late twentieth century reached around 150 million people worldwide. Every year, 1.5 million people die from traumatic brain injury, and 2.4 million become disabled. The incidence of traumatic brain injury is on average 3—4 cases per 1000 population (E.G. Pedachenko et al., 2007; B.V. Zadorozhnaya, 2012; V.V. Kosharnyi et al., 2014; M.E. Polishchuk et al., 2015).

One of the main causes of high morbidity and mortality from a severe traumatic brain injury is irreversible changes in brain structures, particularly in the brainstem, resulting in their displacement and compression with subsequent development of acute stroke, edema, swelling and dislocation of the brainstem.

In Ukraine, patients with traumatic injuries of the nervous system account for more than 70 % of the total number of patients admitted to the neurosurgical departments. Severe forms occur in over 30 % of patients with traumatic brain injury. 30 to 50 % of patients with severe traumatic brain injury die; complete survival of the central nervous system is relatively rare among survivors. In the unfavorable course, the mortality rate may reach more than 80 % (N.E. Polishchuk et al., 2000; V.M. Shevag et al., 2009; N.N. Zavadenko et al., 2010; V.G. Poltoratsky, 2013; M.H. Morgalla et al., 2014).

Organizations such as the NICE (National Institute for Clinical Excellence) and BTF (Brain trauma foundation) regularly issue protocols and guidelines for patients with severe brain injury. These protocols are based on multi-center, prospective, randomized trials and are consistent with the principles of evidence-based medicine.

Classification of traumatic brain injury

For the first time, the classification of traumatic brain injury was proposed by J.L. Petty in 1774. He described three major clinical forms of brain damage:

1) brain concussion (*commotio cerebri*);
2) brain contusion (*contusio cerebri*);
3) brain compression (*compressio cerebri*).

This classification was used for over three centuries and became the basis for many other classifications of traumatic brain injuries.

Modern clinical classification of acute traumatic brain injury includes seven main clinical forms (A.N. Konovalov, 2001):

1) concussion;
2) contusion of a mild degree;
3) middle-level contusion;
4) severe contusion;
5) diffuse axonal brain damage;
6) brain compression;
7) head compression.

Considering infection of the brain injury is divided into closed (70—75 %) — not initially infected, and open — initially infected with the presence of open paths for penetration of infection into the cranial cavity.

Closed brain injury — there is no violation of the integrity of the soft tissues of the head or there are such disorders, but there is no violation of the integrity of the aponeurosis or no damage to the bones.

Open brain injury — is a trauma characterized by 1) damage to all layers of soft tissues of the head and underlying bone (fractures linear, fragmentary, punctured); 2) fracture of the skull bones in the area of the air-bearing sinuses or fracture of the base of the skull; 3) wounds of the soft tissues of the head with damage to the aponeurosis.

Open traumatic brain injuries are penetrating (damaged dura mater accompanied by liquorrhea) and nonpenetrating (without damaging the dura mater).

An important diagnostic and prognostic criterion is the level of consciousness.

In our country there are seven gradations of the state of consciousness:

1) clear consciousness;
2) stunning moderate;
3) stunning deep;
4) sopor;
5) moderate coma;
6) deep coma;
7) terminal coma.

However, in most countries around the world, the Glasgow coma scale is more widely used to quantify consciousness disorders. On this scale, the degree of impaired consciousness is assessed by three parameters: 1) eye response; 2) motor response; 3) verbal response.

Eye response (E)

Four grades are starting with the most severe:

Eyes opening spontaneously — 4 points.

Eye-opening to speech. — 3 points. (Not to be confused with the awakening of a sleeping person; such people receive a score of 4, not 3.)

Eye-opening in response to pain stimulus — 2 points (a peripheral pain stimulus, such as squeezing the lunula area of the person's fingernail is more effective than a central stimulus such as a trapezius squeeze, due to a grimacing effect).

No opening of the eye — 1 point.

Verbal response (V)

Five grades are starting with the most severe:

Oriented — 5 points. (Person responds coherently and appropriately to questions such as the person's name and age, where they areand why, the year, month, etc.)

Confused — 4 points. (The person responds to questions coherently but there are some disorientation and confusion.)

Inappropriate words — 3 points. (Random or exclamatory articulated speech, but no conversational exchange. Pronounces words without sentences.)

Incomprehensible sounds — 2 points. (Moaning but no words.)

No verbal response — 1 point.

Motor response (M)

There are six grades:

Obeys commands — 6 points (the person does simple things as asked).

Localizes to pain — 5 points (purposeful movements towards painful stimuli; e.g., brings a hand up beyond chin when supraorbital pressure applied).

Withdrawal from pain — 4 points (absence of abnormal posturing; unable to lift hand past chin with supraorbital pain but does pull away when nailbed is pinched).

Decorticate posturing accentuated by pain — 3 points (flexor response: internal rotation of the shoulder, flexion of forearm and wrist with a clenched fist, leg extension, plantarflexion of foot).

Decerebrate posturing accentuated by pain — 2 points (extensor response: adduction of the arm, internal rotation of the shoulder, pronation of forearm and extension at the elbow, flexion of wrist and fingers, leg extension, plantarflexion of foot).

No motor response — 1 point.

According to the Brain Trauma Foundation (BTF) protocols https://www.braintrauma.org/ (2016), the management of patients with severe brain injury is conducted in three categories: procedures, monitoring, and indicators.

Procedures

1. Decompressive craniectomy.
2. Prophylactic hypothermia.
3. Hyperosmolar therapy.
4. Cranial-brain drainage of fluid.

5. Ventilation therapy.
6. Anesthetics, analgesics, and sedatives.
7. Steroids.
8. Nutrition.
9. Prevention of infection.
10. Prevention of deep vein thrombosis.
11. Rash prevention.

Monitoring

12. Intracranial pressure.
13. Excessive brain perfusion.
14. Advanced brain monitoring.

Indicators

15. Blood pressure.
16. Intracranial pressure.
17. Brain perfusion.

The Glasgow outcome scale (B. Jennet, M. Bond) is used to evaluate the results of treatment after a traumatic brain injury.

1. Death	Severe injury or death without recovery of consciousness
2. Persistent vegetative state	Severe damage with a prolonged state of unresponsiveness and a lack of higher mental functions
3. Severe disability	Severe injury with the permanent need for help with daily living
4. Moderate disability	No need for assistance in everyday life, employment is possible but may require special equipment
5. Low disability	Light damage with minor neurological and psychological deficits

Basic clinical forms and their characteristics

Brain concussion (*commotio cerebri*) is a mild form of closed traumatic brain injury, characterized by a prevalenceof reversible functional changes in the brain. It is believed that there are no macroscopic

pathomorphological changes in the case of concussion. Electron microscopic studies have made it possible to find damage to neurons in the form of destruction of their cell membranes, mitochondria, and other changes.

Brain concussion occurs in 70—80 % of the affected and is accompanied by loss of consciousness after trauma of 1—2 to 10—20 min. There is a presence of characteristic cerebral symptoms such as headache, dizziness, general weakness, poor sleep, increased sensitivity to bright light, noise. Also note the presence of autonomic disorders — blood flow to the face, redness of the face, asthenia. In neurological status — divergent strabismus, oscillating pendulum-like nystagmus, moderately manifested Kernig's symptom. These symptoms disappear within 5—7 days after the injury.

One manifestation of a concussion is the presence of various types of memory impairment:

a) retrograde amnesia — no memories of events that happened before the traumatic brain injury. There is a memory loss of events occurring in the coming days, months, or years before the injury, and memory is retained about events of 5—10 years ago;

b) anterograde amnesia — the loss of memories of events that occurred with and around the patient immediately after a traumatic brain injury, but this memory loss occurs after the patient has already developed signs of clear consciousness. Thus, the patient after a traumatic brain injury had a clear consciousness, he was examined by a doctor, and after 2 weeks the patient did not recognize the doctor and could not recall the content of the conversation with him;

c) congrade amnesia — memory loss for the period of disturbed consciousness due to traumatic brain injury.

In the case of brain concussion, there is no damage to the skull bones, examination of the cerebrospinal fluid reveals the absence of significant changes in its composition and intracranial pressure.

The clinical symptoms caused by a concussion disappear within 1—2 weeks after the injury, and the general condition of the patients improves significantly. Surgical treatment is not indicated.

A brain contusion (*contusio cerebri*) is characterized by the presence of areas of brain death that are determined macroscopically; clinically detect various focal symptoms of lesions of the central nervous system. There are three degrees of brain contusion: mild, moderate, and severe.

Mild brain contusion occurs at 10—15 % of victims. Longer loss of consciousness (from 20 to 40 min) is noted after traumathan in comparison to patients with concussion. The mild degree contusion belongs to mild traumatic brain injury and is characterized by the presence of not clearly expressed focal symptoms such as slight anisocoria, clonic nystagmus, signs of pyramidal insufficiency, meningeal symptoms.

Along with focal, there are also cerebral symptoms. The severity of cerebral symptoms usually depends on the mechanism of trauma — in the presence of shock mechanism of trauma there are significant cerebral manifestations, and in local trauma they are insignificant. Often there is retro- or anterograde amnesia. Violations of vital functions are not observed. In the victims, there is increasedblood pressure and bradycardia or tachycardia. There may be a blood admixture in the cerebrospinal fluid. As a rule, there is a slight increase in intracranial pressure. All clinical symptoms regress within three weeks.

Brain contusion of moderate severity is found in 10—15 % of patients with traumatic brain injury. It is characterized by clear focal symptoms of central nervous system damage, depending on the localization of brain trauma. Aphasia, hemianopsia, hemiparesis, hemiplegia, hemihypesthesia, motile disorders, and others are

usually noted. Often, focal symptoms are combined with cerebral symptoms in the form of headache with vomiting, dizziness, and meningeal symptoms. Lost of consciousness lasts from 10—20 minutes to 2—4 hours. Impaired consciousness observed from moderate to deep stun. Retro- and anterograde amnesia are often observed. In the victims, there are vital functions disturbances: tachy- or bradycardia, increased blood pressure, tachypnea without disturbance of rhythm — which do not require hardware correction.

Mild edema of the brain stem manifested by the presence of secondary brainstem symptoms — nystagmus, disturbance of convergence, sluggish pupil reaction to light. These symptoms regress as brainstem edema disappears. In the case of moderate brain contusion during lumbar puncture, is often exhibits hemorrhage into the subarachnoid space.The majority of patients (62 %) have fractures of the bones of the vault and the base of the skull: fractures of the bones of the vault — in 35 %, bases of the skull — in 15 %, vault and bases — in 50 % of the victims. On the first day often there is a psychomotor excitation, caused by the presence of hemorrhage. In the cerebrospinal fluid — an admixture of blood (from 10,000 to 100,000 erythrocytes in 1 μl). After 1.5—2 weeks the impurity of blood disappears. The amount of protein in the cerebrospinal fluid reaches 0.8 g/l or more and depends on the severity of hemorrhage in the subarachnoid space. The amount of protein in cerebrospinal fluid can be significantly higher — from 1.0 to 2.0 g/l — in the case of acute hydrocephalus, which is accompanied by hemorrhage in the ventricles of the brain. At the same time, it is possible to observe liquor hypertension or hypotension. Surgical treatment is not indicated.

Severe brain injury is characterized by prolonged loss of consciousness — from several hours to several weeks, the presence of vital function abnormalities, which is quite life-threatening, and gross focal neurological disorders. The incidence of severe brain injury is 7—10 %. At the moment of trauma, foci of the destruction of brain tissue are characterized by the formation of detritus and the presence of multiple parenchymal hemorrhages. Great importance has the localization of foci of contusion destruction of the brain. Preferably, the foci of the brain contusions are located in the basal divisions of the frontal and temporal lobes and, more rarely, in the convexital areasof the cerebral hemispheres with a spread to subcortical formations. Often there is a primary lesion of the brainstem, especially the medulla oblongata, which is life-threatening. Patients are in a moderate or deep coma since the injury. Note the presence of primary stem symptoms — bilateral mydriasis or myosis, sluggish pupil reaction to light, reduced corneal reflexes, swimming movements of the eyeballs, oscillatory tonic nystagmus, a symptom of Gertwig—Mazandi, bilateral pathologicalplantarreflexes, decerebrate rigidity, etc. Also noted disturbances of cardiovascular system activity — bradycardia, tachycardia, fluctuations of blood pressure. In the case of atonic coma, blood pressure is unstable and can be corrected only by medication, respiratory disorders of the central type.

Four clinical forms are distinguished by the localization of contusion: extrapyramidal, diencephalic, mesencephalic-bulbar, and cerebrospinal.

Extrapyramidal form is mainly observed in lesions of the brain cortex and subcortical structures. It is characterized by the appearance in the clinical picture of hyperkinesis, hypomymia, increased muscle tone, which is often altered by hypotension. In comatose patients, the recovery of consciousness occurs slowly, through the phase of akinetic mutism.

At the diencephalic form of brain contusion, the signs of affection of hypothalamic structures of a brain occur: deep stunning, a sopor or a coma, hyperthermia to 39 °C and above, tachypnoea, increase of blood pressure, tachycardia, an increase of residual nitrogen content in the blood. At the background of cerebral and dural symptoms, focal and stem disorders have varying degrees of severity.

The most severe is *the mesencephalic-bulbar form*. In the clinical picture with of gross disturbance of consciousness down to a coma, except cerebral and focal symptoms, there are signs of involvement in the process structures of the midbrain: suppression of the pupils' reaction to the light, divergence of the eyeballs vertically, anisocoria, reduction of corneal external reflexes.

The cerebrospinal form is characterized by that on the background of cerebral symptoms occur the paresis by type of para- and tetraplegia with sphincter disorders.

Diffuse axonal brain damage. Usually, such trauma is caused by angular or rotational acceleration-deceleration injury (auto-trauma, catatrauma (fall from height), barotrauma). The basis of diffuse axonal brain damage lies in the tension and ruptures of the axons in the white matter of the hemispheres and the brainstem. This type of TBI is more common in children and young people.

Clinic and laboratory-instrumental diagnosis of diffuse axonal damage of the brain:

- Coma is often accompanied by symmetric or asymmetric decerebration or decortication.
- Extreme variability in changes of muscle tonus — from diffuse muscle hypotension to hormetonia. There are gross brainstem symptoms.

Typical motor tetransindroms of a pyramidal extrapyramidal character, often with asymmetry of paresis of the extremities.

- Vegetative disorders are clearly manifested.
- The transition from a coma to a transient or persistent vegetative state is often noted, which onset is caused by opening eyes spontaneously or in response to various irritations.

Pyramidally extrapyramidated tetrasyndrom with bilateral changes in muscle tone and tendon reflexes.

- Mental disturbances.

Brain compression (*compressio encephali*) can be caused by intracranial hematomas (epidural, subdural, intracerebral, intraventricular), hydromas, depressed fractures, and increased edema of the brain, pneumo-cephalus, or foreign bodies.

Main clinical triad in patients with brain compression:

1. Loss of consciousness.
2. Ipsilateral anisocoria.
3. Contralateral hemiparesis.

According to the velocity of brain compression development,there are:

— acute compression — the development of clinical manifestations during the day after injury;

— subacute compression — the development of clinical manifestations of compression within 2—14 days after injury;

— Chronic compression — Threatening clinical symptoms develop 15 days or more after traumatic brain injury.

The epidural hematoma is a traumatic hemorrhage that is located between the inner surface of the bone and the dura. The hematoma results in local and general compression of the brain. The cause of the epidural hematoma is a rupture of the main trunk of the middle meningeal artery, its anterior or posterior branch, isolated damage to the membranous veins, sinuses. The volume of epidural hematomas that are clinically manifested is from 30 to 80 ml. They can be localized in the frontal, temporal, parietal, and temporal, parietal, and occipital (posterior) areas. Clinically, the epidural hematoma may show both cerebral symptoms and focal symptoms with sensory or movement disorders. During the surgical treatment, an extremely important task is to completely evacuate the hematoma and stop bleeding from the affected vessel. Usually, the best operative access is wide access («trauma flap») to the convex surface of the cerebral hemispheres for the evacuation of thrombotic masses, control of bleeding, and establishment of definitive hemostasis (Fig. 1.1). The bone flap is put back, and the dura mater stitched up to it.

Fig. 1.1. **Soft tissue incision (*A*), bone flap formation (*B*) and blood clot removal (*C*):**
1 — temporal craniotomy; *2* — the edge of the bone tissue; *3* — incision of the scalp (dotted line); *4* — temporalis muscle; *5* — middle meningeal artery; *6* — subcutaneous tissues of the scalp; *7* — aponeurotic helmet; *8* — parietal bone; *9* — epidural hematoma; *10* — dura mater; *11* — spinous hole; *12* — opening of the middle cerebral artery; *13* — membrane covering the temporal lobe; *14* — area of the middle cerebral artery in the bone; *15* — membrane covering the frontal lobe

A subdural hematoma is a hemorrhage that lies between the dura and arachnoid mater and causes general or local compression of the brain. Brain compression develops in the case of local brain contusion and rupture of pia mater vessels or cortical vessels. A feature of the subdural hematoma is the formation of hemorrhage on the opposite side of the applied traumatic force by the mechanism of the counterstroke. In the clinical picture, a three-phase change of consciousness is observed. Primary fainting occurs immediately after the injury and is usually short. The light interval lasts from 10—20 minutes to several hours. Then the stunner deepens, drowsiness or psychomotor agitation appears. Orientation in space is inadequate, headache increases, vomiting occurs. Usually, there are focal symptoms (homolateral mydriasis, contralateral pyramidal insufficiency, and sensitivity disorders develop). Development of brainstem syndrome with bradycardia and increased blood pressure, changes in breathing rhythm, bilateral vestibular and oculomotor disorders, tonic convulsions is possible.

During surgical removal of the hematoma, a wide incision of the dura mater is performed for maximum access to the subdural space. It is necessary to remove a blood clot, identify a damaged vessel and achieve

hemostasis by coagulation or using a Gelfoam / Surgicel tamponade (Fig. 1.2). The defect of the dura mater must be closed to watertightness.

Fig. 1.2. **Opening of the dura mater (*A*). The incision begins above the temporal lobe. Blood clot removal (*B*):**
1 — membrane covering the frontal lobe; *2* — middle meningeal artery; *3* — membrane covering the temporal lobe; *4* — temporal lobe; *5* — unfolded shell; *6* — subdural hematoma

Brain contusion. Contusional foci of the brain are areas of an irreversibly damaged brain that cause edema of the brain and subsequent bleeding. As a rule, contusions over the frontal and temporal areas with a diameter of more than 1—2 cm should be surgically removed. Wide resection is limited in functionally important areas of the brain.

Intracerebral hemorrhage. Intracerebral hematomas usually occur in the frontal and temporal areas, but often they are located deeper than the brain contusion. Computed tomography can accurately determine the location of the hematoma. Because hematomas are predominantly associa-ted with other traumatic lesions, standard wide craniotomy is usually chosen. Hematomas near the cortical substance and hematomas greater than 1—2 cm in diameter need to be evacuated; hematomas of deeper localization if they do not cause significant displacement of the median structures, increased intracranial pressure, or neurological deficiency need conservative treatment.

Intraventricular hemorrhage. Such damage is mainly observed in patients with severe traumatic brain injury. It is associatedwith multiple hemorrhages, brain injuries, and skull fractures. Intraventricular hemorrhage may be primary or secondary, resulting from intracerebral hematoma breakthrough in the ventricular system.

Primary intracranial hemorrhage is predominantly caused by vascular plexus trauma, where the force of the shockwave of the cerebrospinal fluid plays a significant role. The blood vessels of the lateral ventricles are damaged due to the occurrence of a cavitation effect, especially in the event of a car accident.

Significant destruction of the lateral ventricles and plexuses leads to the development of cerebrospinal and hemodynamic disorders with the subsequent development of edema — swelling of the diencephalic and mesencephalic areas of the brainstem, impaired vital functions, vegetative-vascular disorders and tachypnea, arterial hypertension, hyperthermia to 39—40 °C, hyperhidrosis, hormetonias, development of sopor, comatose state. Patients are rapidly exhausted due toimpaired protein metabolism, severe intoxication. Secondary intraventricular hemorrhages lead to a rapid deterioration of patients in the event of a breakthrough traumatic hematoma in the ventricular system. Focal symptoms and brainstems symptoms grow rapidly comatose state is occured.

Comprehensive diagnosis of traumatic brain injury

For the correct and timely diagnosis of traumatic brain injury, it is necessary to studythe anamnesis carefully, to clarify the mechanism of injury of the skull and soft tissues of the head, to take into account clinical symptoms such as loss of consciousness, nausea, or vomiting, amnesia. Examination of the head and the whole body and detailed neurological examination should be performed to establish the cerebral and focal neurological symptoms. After determining the pathological changes, a set of additional methods of examination is prescribed, which includes skull radiography, echoencephalography, electroencephalography, CT, MRI, and lumbar puncture in the absence of contraindications.

Radiography of the skull. Traditional craniography is mandatory for patients with traumatic brain injury. This technique is informative for detecting various skull fractures, especially linear ones. Craniography is a fairly accessible method of examining skull bones under different conditions. Almost 2/3 of skull bone fractures occur in patients with closed traumatic brain injury.

Neurosurgeons subdivide skull fractures into the following types: 1) skull vault fractures; 2) fractures of parabasal bones of the skull; 3) fractures of the skull base; 4) combined vault fractures and skull bases.

By the frequency of skull fractures parietal bone is on the first place, then — the frontal and much less — temporal and occipital bones. In cases where the force of impact accounts for a limited area of the skull vault, arising out of fractures. In the case of dull objects on a large section of the skull vault, there are fragmented and multi-shaped fractures, as well as large transverse (from convex to base of the skull) linear fractures. Quite often incomplete fractures are detecting when it is damaged only the inner plate of the skull bones. Among the fractures of the skull base are most often occurring fractures of the middle cranial fossa and a bit less often — fractures of the anterior and posterior cranial fossas. In the area of the skull base sometimes there are multiple cracks throughout the skull or two adjacent cranial fossas.

Bone fragments can damage the integrity of the dura mater and its vessels or sinuses. Skull base fractures are often accompanied by cranial nerve damage and intracranial hemorrhage.

Echoencephalography and ***electroencephalography*** in patients with traumatic brain injury should be performed repeatedly — in the first hours and days after traumatic brain injury.

Computed tomography (CT) is a very effective method of diagnosis for traumatic brain injury. The use of CT makes it possible to assemblea detailed description of fractures of the skull, the degree of damage to the brain substance in the acute period of traumatic brain injury. But, according to M. Greenberg (2019), the value of craniography in the detection of fractures of the skull has not significantly decreased.

The use of CT in a special «bone» mode (visualization only of bone structures of the skull) reveals linear fractures of the vault and the base of the skull, as well as bone fragments of the depressed fracture. The CT determines the size of the depressed fracture and the degree of its delusion. In the case of epidural hematomas, it is possible to find a double-convex area of high density adjacent to the skull vault. Subdural hematomas for CT are preferably detected in the form of a sickle-shaped area of homogeneous density that occupies part or all of the cerebral hemisphere.

Magnetic resonance imaging (MRI) is essential in the diagnosis of traumatic brain injury. However, in the acute period of traumatic brain injury, its diagnostic capabilities are less informative compared to CT. This concerns the detection of damaged skull bones and intracranial hemorrhage.

Intracranial hemorrhages in the first hours after traumatic brain injury are isointensewith the brain so that they are almost indistinguishable. However, the value of MRI, unlike CT, is significantly increased in the diagnosis of subacute, chronic intracranial hemorrhage. The presence of the characteristic intensity of the MR signal on T1- and T2-weighted images, which is detected by MRI, allows setting the timing of formation, localization, and size of the intracranial hemorrhage and brain contusion unmistakably.

The use of MRI in victims with craniocerebral trauma in T1- and T2-weighted modes in three mutually perpendicular planes allows you to clearly identify the topographic location of the pathological center, its size, relationship with subcortical structures, ventricular system of the brain, as well as the presence of damage to the brainstem structures.

MRI is one of the informative methods of diagnosing diffuse axonal brain damage. Small-focal hyperintensive sections on the border of gray and white matter and in the corpus callosum are detected at T2-weighted tomograms.

Lumbar puncture is one of the invasive methods of a comprehensive diagnosis of traumatic brain injury. It is performed in the absence of contraindications for its use (the presence of volumetric formation in the temporal lobe and the posterior cranial fossa) after carrying out all the complex methods of diagnosis (including CT and MRI), which allowed to assume the possibility of subarachnoid hemorrhage due to traumatic brain injury.

In most cases, patients with concussion have normal cerebrospinal fluid pressure, much less have cerebrospinal fluid hypotension (100 mm Hg or less) or cerebrospinal fluid hypertension (200 mm Hg or more). Hemorrhage into the subarachnoid space in such victims is usually not detected. In the case of brain contusion, cerebrospinal fluid hypertension (400—600 mm) of water is determined. Art.) and hemorrhage into the subarachnoid space of varying intensity — a light pink color of cerebrospinal fluid or intense red, if the hemorrhage is massive. Laboratory data indicate the content of erythrocytes in the cerebrospinal fluid.

Implantation of **diagnostic burr holes (DBH)** is an invasive method of diagnosis of traumatic intracranial hematoma. According to M. Greenberg (2010), indications for the imposition of DBH are the progressive deterioration of the patient's condition, the appearance of dislocation symptoms, and the impossibility of brain CT.

Algorithm of choosing the side of the overlay DBH:

1. Determine the dilated pupil.
2. If both pupils are enlarged, start from the side from which the pupil has expanded earlier.
3. If the pupils are the same, start from the side where the injury occurred.
4. If there are no signs that can determine the location of the injury, start on the left side. To do this, the author proposes the following method:

a) start from the zygomatic arch by stepping 1 cm from the tragus of the ear (not to damage the branch of the facial nerve and the anterior branch of the superficial temporal artery);

b) the further direction of the incision line — upwards, and then with the bend back at the level of the upper edge of the ear;

c) then turn the incision line up again 4—6 cm behind the ear;

d) then turn the incision line forward, not reaching 1—2 cm to the midline on its side, and terminate at the border with the scalp.

It is necessary to impose burr holes in such a way that if necessary a craniotomy can be performed.

Decompression trepanation. According to the Brain Trauma Foundation (BTF) protocol https://www.braintrauma.org/ (2016), to reduce mortality and improve neurological outcomes in patients with severe brain contusion it is recommended to use a large size of frontal-temporal-parietal decompression trepanation (at least 12 × 15 cm or 15 cm in diameter).

Complications of closed traumatic brain injury

1. Late intracranial hemorrhage.
2. Pachymeningitis.
3. Hydrocephaly.
4. Epilepsy.
5. Meningoencephalitis.
6. Purulent leptomeningitis.
7. Thrombophlebitis.
8. Brain abscess.

Complications of open traumatic brain injury

I. Early:

1) bleeding;
2) intracranial hematomas;
3) liquor;
4) prolapse of the brain;
5) shock.

II. Late:

1) meningitis;
2) meningoencephalitis;
3) CSF fistulas;
4) osteomyelitis;
5) granulomas;
6) scar-commissural processes;
7) Late prolapse of the brain;
8) abscesses;

9) hydrocephalus.

Combined brain injury

Simultaneous damage to one or more types of damaging energy by two or more anatomical systems is called combined trauma (craniovertebral, cranio-transabdominal).

Multiple trauma is the simultaneous destruction of one or more parts of the same functional system by one type of damaging energy (multiple brain concussions, multiple fractures of the lower extremities).

Combined trauma — the damage to the body by different types of damaging energy acting simultaneously (mechanical, thermal, ionizing radiation, etc.).

Classification by localization:

Damage to the facial skull.

Damage of the chest and organs of the respiratory system.

Damage of abdominal organs.

Damage of spine and spinal cord.

Damage to the musculoskeletal system.

Damage to the facial skull:

— single fractures of the mandible;

— traumatic tooth extraction;

— soft tissue lesions without significant bleeding;

— lesions by Le Fore type 2, 3;

— multiple lesions of the facial skull.

Damage to the chest and respiratory system:

— fracture of the clavicle;

— fracture of up to three ribs without damage to the chest, nerves, and blood vessels;

— fractures of ribs with damage of vessels;

— damage of the chest cavity;

— hemopneumothorax;

— damage of the mediastinum.

Damage of abdominal organs:

— damages of the cavity of the abdominal cavity;

— damages of parenchymal organs.

Damage of spine and spinal cord:

— fractures of the bodies and roots of the vertebrae without damaging the spinal cord;

— fractures of vertebral bodies and roots with damage to the spinal cord.

Damage to the musculoskeletal system:

— single fractures of the upper extremities;

— pelvic, hip, multiple limb lesions.

Independent work on the topic

Write down definitions of the following terms:

1. Dislocation syndrome.
2. Brain edema.
3. Diagnostic milling hole.
4. Resection trepanation of the skull.
5. Bone-plastic trepanation of the skull.
6. Patients' quality of life.

Answer the question:

1. Name the clinical criteria for different degrees of consciousness disorder.
2. What are the methods of additional examination of a patient with traumatic brain injury and their diagnostic capabilities?
3. Name the indications for conservative and surgical treatment of traumatic brain injury.
4. Name the main stages of surgical treatment for meningeal and intracerebral intracranial hematomas.
5. What scales allow to evaluate the effectiveness of treatment of traumatic brain injury?
6. What complications of traumatic brain injury can develop and when?

Tasks for self-control

1. Epidural hematomas are often accompanied by:

a) fractures of the skull bones;
b) nasal liquorrhea;
c) auricular liquorrhea;
d) ventricular hematomas;
e) pneumocephalus.

2. Epidural hematomas are often limited by:

a) the suture of bones of the vault;
b) the cerebral cortex;
c) choroid plexuses;
d) transparent membrane;
e) crescent-shaped process.

3. Nasal liquorrhea is often accompanied by:

a) damaging of the venous sinuses;
b) damaging of the lattice plate of the vomer bone;
c) fracture of the parietal bone;
d) epidural hematoma of the posterior cranial fossa;
e) fatty embolism of cerebral vessels.

4. A concussion is characterized by:

a) hemorrhage into the subarachnoid space;
b) retrograde amnesia;
c) hemiparesis;
d) paresis of upgaze;
e) formation of the carotid-cavernous joint.

5. Acute traumatic intracranial hematomas are characterized by:

a) platybasia;
b) Bruns syndrome;
c) Foster—Kennedy syndrome;
d) pulsating exophthalmos;
e) the presence of a «light gap».

Topic 2

SPINAL CORD INJURIES

Vertebrae, in the number of 33—34 are overlapping-ring-shaped bones and are gathered into a single column — a spine or a vertebral column (columna vertebralis).

The spine is divided into the following sections: cervical (pars cervicalis), thoracic (pars thoracica), lumbar (pars lumbalis), sacral (pars sacralis), and coccygeal (pars coccygea). Accordingly, vertebrae are divided into five groups: cervical vertebrae (vertebrae cervicales), thoracic vertebrae (vertebrae thoracicae), lumbar vertebrae (vertebrae lumbales), sacral vertebrae (vertebrae sacrales) and coccygeal vertebrae (vertebrae coccygeae). The spine is characterized by mobility, which is most noticeable in cervical and lumbar divisions. The thoracic spine is relatively motionless. Spinal dislocation is a displacement of the vertebral bodies along with each other. It occurs more frequently in its most movable divisions — the cervical and lumbar ones. On the border of movable and sedentary parts of the spine, there are often closed fractures, which, due to the spongy structure of the vertebral bodies takes the form of compression or a comminuted-compression fracture.

Parts of the spine of an adult form four curvatures (curvaturae) in the sagittal plane: cervical, thoracic, lumbar (abdominal), and sacral (pelvic). The cervical and lumbar curves are convexly anteriorly (lordosis), thoracic and pelvic — posteriorly (kyphosis).

Described curvatures of the spine should be taken into account during laminectomy at different levels of the spine, since the nature of the curvature effects the standing of spinous processes and the depth of the surgical wound.

All vertebrae also are divided into two groups: the so-called true and false vertebrae. The first group includes cervical, thoracic, and lumbar vertebrae, the second — sacral vertebrae, fused into the sacral bone (os sacrum), and coccyxes,fused into the coccygeal bone (os coccygis). Sometimes the first sacral vertebra is separated by the cartilage plate from the rest of the sacral vertebrae and looks like the sixth lumbar vertebra. This is the lumbarisation of the first sacral vertebra.

The fusion of the fifth lumbar vertebra with the first sacral is called sacralization (thus, the transverse processes of the last lumbar vertebra merge with the lateral mass of the I sacral). The shortening of the neck, caused by the fusion of the cervical vertebrae, is called Klippel—File syndrome. The merge of the Atlas with the occipital bone is called occipitalization.

Epidemiology. In recent years, the number of patients with backbone and spinal cord lesions has increased significantly. In Ukraine, 1500—2000 cases of spinal cord injury are reported annually (M.E. Polishchuk, E.I. Slinko, 2001).

In 50 % of patients, spinal cord injuries are combined with multiple tubular bones and skull traumas, and 20 % have injuries to abdominal organs. Among patients with traumatic spinal cord lesions, 80 % are people under 40 years of age. The point is that in 50 % of cases mortality from spinal trauma is not due to the initial severity of the trauma but to its poor diagnosis and inadequate management of the patient at the pre-hospital and hospital stages.

According to official data in the USA, 18 000—38 000 people get spinal traumas annually, with an average of 4700 cases (or about 20 %) accompanied by paraplegia. Furthermore, 65 % of patients have cervical spine injuries, 20 % of the thoracic spine, and 15 % of the lumbar spine. In 5—20 % of the affected, spine injuries are characterized by multiple fractures.

Classification of spinal injuries

Depending on the involvement of adjacent to the spine tissues and the spinal cord,first of all, mark out three groups: uncomplicated, complicated, and combined lesions. The *uncomplicated lesions* are limited only by bone and soft tissue structures that directly make the spine. In case of *complicated* ones, bone fragments of vertebrae damage adjacent to the spine tissue and organs. *Combined trauma* is characterized by simultaneous damage to the spine and other organs by the direct action of the damaging factor.

According to the mechanism of the damaging, there are bending, extending, rotational dissecting injuries and damages arising from axial pressure. L. Bohler (1956), E.A. Nicoll (1949), and F.W. Holdsworth (1970) based the classification of spinal injuries on the condition of the locking appliance and the violation of the mechanical stability of the spine that occurs (or does not occur) at the moment of damage. Accordingly, the authors marked

lesions as stable (simple anterior compression fractures, burst fractures, and extensional damage) and unstable, which included distraction and rotational dislocations, fracture-dislocations, and vertebral fractures.

All of the above classification principles are included in consolidated classifications of spinal injuries.

The combined classification of G.P. Saldun (1983) includes 8 major groups and 46 signs of vertebral segment damage, according to which injuries are subdivided into follows (terminology, numbering, and lettering are given by G.D. Nikitin, G.P. Saldun, et al., 1998).

I. By localization of the lesion:
   a) cervical division;
   b) thoracic division;
   c) lower thoracic and lumbar divisions;
   d) sacrococcygeal division.

II. By character and degree of the damage to the spinal cord and its elements:
   1) uncomplicated fractures;
   2) complicated fractures:
   a) spinal cord rupture («anatomical interruption»);
   b) compression of the spinal cord;
   c) contusion of the spinal cord;
   d) compression or damage of elements of the spinal cord (its roots).

III. By the mechanism of damaging:
   1. Compression fractures;
   2. Compression-flexural fractures;
   3. Flexural fractures;
   4. Compression-rotational fractures;
   5. Rotational damage;
   6. Extensible fractures.

IV. By the degree of wedge-shaped deformation of the vertebra:
   1. Marginal fractures;
   2. Deformation up to 1/4 of the normal height of the vertebral body;
   3. Deformation up to 1/3 of height;
   4. Deformation up to 1/2 height;
   5. Deformation over 1 height;

V. By character of damage to the vertebra:
   1. Penetrating fractures:
   a) with neurological symptoms;
   b) without neurological symptoms.
   2. Vertical fractures.
   3. Horizontal fractures.
   4. Fragmentary («burst fractures»).

5. Multiple vertebral fractures:

a) adjacent;

b) remote;

c) combined with damage to other areas of the musculoskeletal system.

6. Fractures of arches:

a) one-sided (with the shift, without shift);

b) on both sides (with the shift, without shift).

7. Fractures of the articular processes:

a) one-sided (with the shift, without shift);

b) on both sides (with the shift, without shift);

c) adjacent vertebrae.

8. Full separation of posterior support complex.

9. Damage (rupture) of ligaments.

10. Fracture-dislocations:

a) complete;

b) incomplete;

c) complicated;

d) uncomplicated.

11. Fractures of spinous processes, fractures of transverse processes (single, multiple).

VI. By character of resistance:

1. Stable lesions:

a) non penetrating compression fractures of vertebral bodies without signs of damage to the posterior support complex, with a wedge-shaped deformation up to 1/3;

b) extensible fractures.

2. Conditional-stable lesions:

a) uncomplicated compression fractures of vertebral bodies with wedge-shaped deformation up to 1/2 without signs of damage to the posterior support complex;

b) multiple fractures of vertebral bodies with a total wedge-shape up to 1/2 of one of them;

c) penetrating fractures with persistent pain syndrome.

3. Unstable lesions:

a) fractures of vertebrae with a wedge-shaped deformation of 1/2 or more complicated or uncomplicated character;

b) less expressed wedge-shaped deformation, but with signs of damage to the posterior support complex or deformation of the spinal canal;

c) fractured dislocations, complicated and uncomplicated;

d) multiple fractures of vertebrae with a total wedge-shape of more than 1/2 of one of them;

e) fragmentary, vertical and horizontal fractures;

f) complicated and uncomplicated fractures after laminectomy.

VII. Vertebral fractures in the elderly.

VIII. Combined fractures (with damage to internal organs, brain, etc.).

The classification of spine injuries by F. Denis (1983) is based on the theory of three columns. Unlike F. Holdsworth's (1970) theory of two columns, the boundary between which was the frontal plane extending along the posterior longitudinal ligament, F. Denis distinguished a middle column, which adjoins directly to the spinal canal. The anterior column by F. Denis consists of the anterior longitudinal ligament, anterior parts of vertebral bodies, and intervertebral discs. The middle column is made up of posterior parts of vertebral bodies, intervertebral discs, and posterior longitudinal ligament adjacent to the spinal canal; the posterior column is formed out of arches, transverse, articular, and spinous processes, as well as the posterior musculoskeletal and ligament apparatus of the spine.

Clinical symptoms and severity of spine damage according to F. Denis determined by a) mechanism of injury; b) affected area (damaged column); c) stability (or instability) of the damaged segment. Wherein, the concept of «instability» has a dual interpretation and includes mechanical and neurological components.

Mechanical instability (the author also uses the term «first-degree instability») is characterized by abnormal spine motility (or threat), occurring at the level of the injured segment immediately at the time of injury, or progression of spinal deformation in the distant period (so-called dynamic or delayed instability).

Neurological instability (or second-degree instability) is damage or a theoretical possibility of damage to the spinal cord and its elements by bone fragments of affected vertebrae directly at the moment of trauma or in case of inadequate treatment. The author attributes the combination of mechanical and neurological instability to third-degree instability.

Different authors interpret the concept of spine instability differently. It is appropriate to cite the classic triad of clinical signs of chronic posttraumatic spine instability, developed by I. Posner et al. (1981): 1) dynamic (progressive and/or transient) neurological disorders; 2) pain; 3) progressive deformation of the spine.

Damage to the spinal cord, its roots, and meninges (S.N. Nekhlopochin et al., 2001) is divided into:

1. Traumatic radiculitis.

1.1. Primary:

a) contusion;

b) brain stem hemorrhage;

c) stretching;

d) anatomical damage.

1.2. Secondary radiculitis due to compression by:

a) bone fragments of the spinal canal;

b) fallen disc;

c) damaged ligamentum flavum;

d) epidural hematoma;

e) hemorrhage into the subarachnoid space.

2. Concussion.

3. First-degree contusion.

4. Second-degree contusion.

5. Third-degree contusion.

6. Haematomyelia (by localization):

a) ventral;

b) dorsal;

c) lateral;

d) ventrolateral;

e) dorsolateral;

f) tubular.

7. Anatomic damage to the spinal cord:

a) partial;

b) transverse.

8. Compression of the spinal cord

8.1. By the time of occurrence:

a) acute;

b) early (hours or days after injury);

c) late (months and years after injury).

8.2. By localization:

a) anterior;

b) internal;

c) posterior.

8.3. By compression ratio:

a) partial (partial functional block);

b) complete (full functional block).

8.4. By character of development:

a) acute progression;

b) chronic development;

c) stabilized.

F. Denis and L. Krach (1984) differ the following clinical variants of spinal cord injuries:

— complete tetraplegia — total loss of movements of upper and lower extremities in case of injury to the cervical division of the spinal cord;

— incomplete tetraplegia — partial loss of movements of upper and lower extremities in case of trauma to the cervical division of the spinal cord: a) anterior—cerebral—artery syndrome; b) Brown—Sequard syndrome; c) central damage syndrome;

— complete paraplegia — total loss of movements in lower extremities;

— incomplete paraplegia (paraparesis) — partial loss of movement in lower extremities;

— pseudo complete paraplegia — the complete absence of movements in lower extremities in case of injury of the epiconus and cone of the spinal cord;

— rising paraplegia (in updated literature, this type of disorder is described as «rising myelopathy»), growing in dynamics, extending beyond the level of vertebral damage; neurological symptoms are observed in the first 4 days after the injury.

V.P. Bersnev et al. (1998), describing different periods of the clinical course of spinal trauma, gave additional specific clinical and morphological features:

— acute period (up to 3 days): morphologically — soft tissue swelling, primary necrosis and myeloid ischemia of the injury zone; unstable clinical picture, including symptoms representing spinal shock;

— early period (2—3 weeks) corresponds to the time of occurrence of primary complications: meningitis, myelitis, pneumonia, urosepsis, exacerbation of chronic infectious-inflammatory diseases;

— intermediate period (up to 3 months) — the persistence of purulent complications, on the background of which fibrotic processes develop in the damaged brain tissue, a bone callus is formed in the fracture zone, and the bedsores begin to heal;

— the late period (from 3 months to 1 year) corresponds to the period of late complications: pyelonephritis, enterocolitis, trophic disorders, bedsores, sepsis;

— residual period (more than 1 year after injury) — a period of residual effects and consequences.

In 1969, Frankel et al. developed a scale for qualitative assessment of neurological complications of spinal injuries for the first time, which is now used for the rough estimation of myelopathy of different genesis. On this scale, five types of neurological spinal disorders are distinguished:

Type A — paraplegia with complete sensory impairment (clinical picture of complete transverse spinal cord lesion).

Type B — paraplegia with partial sensitive disorders.

Type C — paraparesis with marked impairment of motor function.

Type D — paraparesis with a slight limitation of motor functions.

Type E — the absence of neurological complications or the presence of minimal neurological symptoms.

Considering features of pediatric patients, O.Yu. Mushkin et al. (1998) modified the Frenkel's scale for use in pediatric practice and found it possible to attribute a complete absence of pathological neurological symptoms to type E, whereas lesions of the anterior columns of the spinal cord, which are detected only by a direct neurological examination and which does not significantly restrict arbitrary movement are type D. Also, the type R is additional radicular (pain) syndrome.

The Frenkel's scale is used for qualitative characterization of injuries, which are accompanied by damage to the spinal cord below the level of the cervical thickening.

To increase the objectivity of the evaluation of movement disorders, the American Association for Study of the Spinal Cord Injury NASCIS and ASIA have introduced quantitative schemes based on the determination of the strength of muscles innervated by a certain spinal segment — the so-called key muscles.

The strength of each key muscle is evaluated on a 5-point scale first proposed by the Nerve Injury Committee in 1943. 0 points stand for paralysis; 1 is for palpable or visible muscle contractions; 2 — active movements with the incomplete volume of counteracting the force of gravity; 3 — the full volume of movements to counteract the force of gravity; 4 — the full range of movements with moderate counteraction; 5 — unlimited movement.

ASIA summarizes the function of 10 muscles, evaluated on both sides. The maximum total amount is 100 points. Unlike NASCIS, they summarize the functions of the 14 muscles on the right side (taking into account the predicted symmetry of neurological disorders).

Clinical symptoms of spinal cord injury

Spinal cord concussionis characterized by reversible pathological changes in the form of a syndrome of total or partial conductivity disorders. Short-term paresis, sometimes paralyses, fleeting sensitivity disorders, rarely limited by paresthesias; temporary disorders of the function of the pelvic organs. There are no structural changes in the brain. Pathological phenomena disappear from a few minutes to several days after the injury (depending on the severity of the concussion). Cerebrospinal fluid, generally, without pathological changes. The passability of subarachnoid space is not disturbed.

Contusion of the spinal cord leads to organic changes in the brain tissue (necrosis, hemorrhage, etc.) and functional disorders (spinal shock). The «physiological interruption» of the spinal cord, which occurs at the time of trauma, is difficult to differentiate from the anatomical one in the first days and weeks. It is also characterized by the development of flaccid paralysis, paresis, sensitivity disorders, impaired function of pelvic organs, and vegetative nervous system. In the cerebrospinal fluid, there may be an admixture of blood, which is a sign of hemorrhage into the subarachnoid space and is usually accompanied by moderate meningeal symptoms. The passability of the subarachnoid space is not disturbed. Partial restoration of functions is caused by reduction of spinal shock, elimination of edema and swelling, compensation of circulatory disorders, and other impaired functions of the spinal cord.

The recovery time of the motor, sensory functions and function of pelvic organs depends on the severity of the lesion and usually lasts for 3—8 weeks. Initially, tendon reflexes restore and pathological reflexes appear; flaccid paresis or paralysis replace by the spastic one if the damage is higher than the lumbar or cervical thickness. Residual spinal cord dysfunction may be observed.

Spinal cord compression can occur at the time of injury (acute), one hour, and days after the injury (early), for months and years (late). According to localization, there are the following types of compression: posterior (by an arch of a vertebra, epidural hematoma, ruptured ligamentum flavum), anterior (by a body of the damaged vertebra, fallen intervertebral disc), internal (by an intracerebral hematoma, detritus at the center of softening with swelling). It can be total or partial. In distant periods, compression is caused by a scar, bone callus, and so on.

Tubule- of sphere-shaped **hemorrhage into the gray matter of the spinal cord** over several segments, often in the cervical and lumbar thickness, is called hematomyelia. The spilled blood, spreading through or near the central canal, often destroys the gray matter and compresses pathways. The symptoms of hematomyelia occur almost immediately after the injury and progress within hours. Clinically, hematomyelia manifests by the syndrome of total or partial conductivity disorders. After reduction of spinal shock, hematomyelia reveals with a focus of dissociated sensitivity disorders, usually symmetrical (retention of deep and partial tactile sensitivity, in conjunction with pain and temperature disorders), combined with attenuation or total loss of reflexes, paresis, or paralysis of the muscles, innervated by the affected segments of the spinal cord.

Spinal shock is a pathophysiological condition characterized by impaired motor, sensory functions, and reflexes of the spinal cord below the site of injury. Spinal shock manifests by a spontaneous reduction of excitability and inhibition of the activity of all reflex centers of the spinal cord, located below the site of injury. Spinal shock results in arterial hypotension, lack of vascular reflexes, sphincter disorders. The duration of spinal shock is up to 2 months. According to M. Greenberg (2010), the term «spinal shock» is used in two meanings:

1. Hypotension, which develops after spinal cord injury, and temporary loss of spinal reflexes below the level of injury. Impaired sympathetic innervation due to loss of vascular tone below the level of injury, or parasympathetic effect leading to bradycardia. Loss of muscle tone due to skeletal muscle paralysis below the level of damage leading to venous stagnation and relative hypovolemia; bleeding from concomitant wounds, leading to true hypovolemia.

2. Temporary loss of spinal reflexes below the level of lesion leads to flaccid paralysis, which persists from 2 weeks up to 2 months. Spinal cord reflexes below the level of damage can also be suppressed. Spinal shock develops within hours, days, sometimes weeks after injury. The shock is caused by the trauma of the brainstem and trauma of the cervical and upper thoracic divisions of the spine. The mechanism lies in the disruption of regulatory influences from the upper autonomous centers on the cervical and upper thoracic divisions. Bradycardia, hypotension, hypothermia are main vegetative clinical manifestations of spinal shock. The peristalsis is disturbed, there is swelling of the mucous membranes. Horner syndrome occurs. The loss of vasomotor tone is manifested by hyperemia of the skin, internal organs, which exacerbates the manifestations of hypotension. With regression of shock autonomous functions, reflex activity is restored. From a pathophysiological point of view, the spinal shock is a condition of temporary inhibition of the reflex activity of the spinal cord.

Spinal shock is the result of traumatic spinal hyperafferentation, which causes a great depth of shock in the case of gross damage to the spinal cord. This leads to a significant interruption of the amplification and mitigation effects of the brain (midbrain, pons, medulla, etc.).

A complete rupture of the spinal cord is clinically manifested by loss of all motor and sensory functions below the level of injury. There are also areflexia, clarity of consciousness, anesthesia, and autonomic paralysis below the level of injury. Hypotension can occur as the result of trauma above the Th_5 vertebra.

Incomplete rupture of the spinal cord has different clinical symptoms. The Brown—Sequard syndrome occurs when the spinal cord is halved; conduction disorders in the form of central paralysis and loss of proprioceptive vibration sensitivity on the affected side are detected; on the opposite side, there is pain and temperature anesthesia.

*Central damage syndrome*often occurs as a result of a sudden flexion/extension of the cervical division of the spine or narrow spinal canal and is characterized by bilateral weakness, loss of pain and temperature sensitivity in upper and lower extremities.

Anterior-spinal-artery syndrome occurs in case of ischemia of the area, supplied by this artery, with damage to the anterior 2/3 of the spinal cord and preservation of a function of dorsal columns. Bilateral motor disorders and loss of pain and temperature sensitivity develop with partial loss of proprioceptive, vibration, and tactile sensitivity.

There are various disorders of the autonomic nervous system in case of a lesion of the spinal cord. If the damaged area is higher than C_3, spontaneous breathing is not possible, C_4—C_6 — respiratory failure leads to progressive hypoxia and CO_2 retention. Spinal cord pathology leads to intestinal obstruction and extension of the stomach. If the damaged site is higher than T5, the sympathetic effect on the tone of the vessels is disturbed. Changes in body position (elevation of the head end of the bed) can lead to a sudden decrease of the blood pressure with reflex tachycardia and vascular spasm in the periphery.

Special methods of spine examination

The diagnostic algorithm for a set of instrumental studies in the acute period of spinal trauma involves the following sequence:

1. Radiographs (spondylograms) in two (anterior-posterior and late-ral) projections.
2. Spondylograms with the use of special arrangements.
3. X-ray computed tomography (CT).
4. Myelography ascending or descending.
5. CT-myelography.
6. Magnetic resonance imaging (MRI).

Survey radiography (standard spondylography) is the basic method of radiological examination; it is carried out in a position lying in two projections. Investigations are performed with the maximum capture of the entire spine, and on the anterior-posterior radiographs wings of the iliac bones are also seen. The method allows:

— to evaluate the condition of the spine;

— to calculate the magnitude of deformation of the spine in frontal and sagittal planes, To estimate the magnitude of the torsion (pathological rotation) of the vertebrae;

— to determine the size of the spinal canal;

— to evaluate the condition of the paravertebral tissues.

Myelography is performed to determine the patency of the subarachnoid space, to detect levels of lesions and ruptures of the spinal cord. The ascending myelography is done to determine the lower boundary of the block of the subarachnoid space by the introduction of contrast agent (Omnipaque) between L3—L4 or L5—S1 vertebrae, with the lower end of the X-ray table down. The upper boundary of the block of the subarachnoid space is determined by conducting descending myelography. In this case, a contrast agent is introduced into a large cistern with the lifting of the head end of the X-ray table. Turning the patient on the table after the introduction of the contrast agent makes it possible to visualize the nature of filling the subarachnoid space with contrast: the flow of «obstacles» reveals the level of damage to the spinal cord.

Computed tomography is the most informative study to evaluate the bone structure of vertebrae in a limited number of (one to two) vertebral segments, primarily in the posterior parts of bodies, arches, and processes (transverse, articular, or spinous). It is possible to visualize the condition of the paravertebral tissues at the level of the pathological process. In combination with contrast myelography (CT-myelography), the method is used to evaluate the patency of the cerebrospinal tract, condition of the spinal canal, and the spinal cord in the site injury.

Magnetic resonance imaging is the most informative study for:

— visualization of the spinal cord, its spare spaces (subarachnoid and epidural), the spinal canal in whole and at the level of lesion;

— disk imaging;

— early detection of pathology accompanied by microcirculatory disorders in the spine;

— evaluation of the condition of the paravertebral tissues.

Functional radiographs — performance of anterior-posterior and lateral spondylograms in the maximum possible movements: in the frontal plane — at lateral slopes, in the sagittal — while bending and extending. Used to determine the natural mobility of the spine.

X-ray tomography — performing layer-by-layer X-ray sections allows clarifying the nature of pathological changes in vertebrae and paravertebral tissues, to evaluate the structure of vertebrae.

Spondylourography is a combination of spondylography with simultaneous contrasting of the urinary tract. Usually used when concomitant pathology of the urinary system is suspected in children with congenital vertebral defects. Simultaneous assessment of the condition of the spine and urinary tract helps to reduce total radiation load during the study.

Myelotomography is a study of the spinal canal with the injection of contrast substances into the subarachnoid space, which makes it possible to: a) visualize the subarachnoid space and determine its patency; b) visualize the spinal cord; c) identify extradural and extramedullary formations that impair the patency of the CSF pathways.

Echospondilography (ESG) — ultrasound examination of the spine and spinal canal. The method is always used to diagnose the malformations of the spine, as well as to assess the condition of the spinal canal.

Epidurography — a study of the spine and spinal canal with the introduction of contrast agents into the epidural space.

Venospondylography (VSG) is a study of the spine with the contrast of epidural and vertebral venous pathways. The contrast agent is introduced into the bone structures of the spine (usually in the spinous process), assessing the condition of the venous epidural plexus. The method is used for the early detection of formations of the epidural space.

Radioisotope scan of the skeleton — the study of the activity of metabolic processes in bone tissue by recording the accumulation of osteotropic radiopharmaceutical agent (RPA); allows to find out pathological bone lesions with active metabolism (inflammation, tumor).

Discography is a contrast study of the intervertebral disc. Nowadays is used in the case of polysegmental discopathies.

Thus, the value of different research methods changes significantly:

— to determine the type and magnitude of deformation of the spine, the most informative are standard spondylography and spondylourography;

— for evaluation of the structure of bone elements of the spine — CT, radiograph;

— to evaluate the condition of the disks — MRI, discography;

— for visualization of the spinal cord and its spare spaces — MRI, CT-myelography, myelography, epidurography;

— for prenatal diagnosis of spinal diseases and its approximate imaging in young children (screening) — echospondilography;

— for detection of hemodynamic disorders in vertebrae — MRI;

— for early detection of solid and systemic tumor lesions of the spine (pre-X-ray stage), evaluation of their prevalence in the spinal segments and bone system — radioisotope scanning, MRI;

— for evaluation of epidural and vertebral venous basin — venospondylography;

— for evaluation of the condition of the paravertebral tissues — MRI, CT, radiograph.

CT, MRI is the method of choice in the diagnosing of spinal injury. The CT scan allows characterizing the fracture with higher accuracy than the usual spondylography: to define its level, the number of damaged vertebrae, to detect fractures of arches, articular processes, different parts of the vertebral bodies, to determine the length of fracture lines and diastasis between bone fragments. The CT scan allows visualization of bone fragments that have shifted into the lumen of the spinal canal; on the usual radiographs, they can not be seen behind arches of the vertebrae. The use of CT and MRI scans allows determining the extent of damage — contusion, and compression of the spinal cord (or its roots) by bone fragments — and indications for surgical treatment by way of spinal cord decompression and stabilization of the spine in the site of fracture.

In case of trauma to the cervical division of the spine, the fracture line of the spine, even with a large diastasis of bone fragments on the usual and special (oblique in 1/2 and 3/4 laying) radiographs for various reasons cannot be seen. During the CT scan, a fracture of the vertebra with a sufficiently large diastasis of bone fragments can be detected.

In the case of injury to the upper and middle thoracic divisions of the spine due to the imposition of ribs, shadows of the internal organs, the scapula is not always possible to determine the amount of lesion by conventional radiographs. A CT scan reveals a complete amount of bone trauma, without the need for complicated staging of the patient, and it takes a few minutes.

Due to CT-myelography, it is possible to detect the localization and the severity of compression of the spinal cord, the patency of the subarachnoid space. In the acute period of spinal trauma, depending on the condition of a patient, it is necessary to use only some of the methods, the most informative for making the diagnosis and which are most easily tolerated by the patient. Commonly, the radiological examination is carried out in the following sequence:

1. Spondylography in required arrangements (due to the rotation of the tube of the x-ray machine, but not the rotation of the patient).
2. CT.
3. Myelography.
4. CT-myelography.

All these studies do not exclude but complement each other. Mostly, in 90—95 % of cases, such an algorithm makes the correct diagnosis and avoids diagnostic mistakes and inaccuracies. In cases of the discrepancy of the clinical picture and available radiological and CT data, it is efficient to conduct an MRI study to visualize the spinal cord.

MRI has limited use in the diagnosis of acute hematomas due to lower sensitivity. The value of MRI is significantly increased during the study of patients in subacute and chronic stages of spinal trauma due to the possibility of visualization of spine divisions (craniovertebral and cervical ones), which are unavailable for other methods.

MRI provides good anatomical imaging of the spine and spinal cord. T2-weighted images are preferred to evaluate the degree of channel narrowing. T1-weighted images show changes in vertebral body shape, fragment displacement, traumatic disc herniation, displacement, hemorrhage, and combined damage or deformation of the spinal cord. Concomitant spinal cord injuries, edema, myelomalacia, and necrosis are all better defined on T2-weighted images. Subacute epidural or intramedullary hemorrhages are seen as zones of signal enhancement on T1- and T2-weighted tomograms. MRI should be considered particularly useful in assessing the effects of spinal trauma. Without endolumbar contrast, MRI canvisualize the state of the spinal cord, changes of its size and shape, diffuse or local atrophic changes, detect hydromyelic cavities and CSF cysts. MRI greatly facilitates the diagnosis of transverse ruptures of the spinal cord.

Treatment. Indications for surgical treatment in patients with spinal cord trauma are determined after assessing the general condition of the patient and the character of the injury. Emergency surgery is indicated in case of the presence of compression of the spinal cord and its roots, the increase of neurological symptoms, and the presence of unstable spine fracture. If the patient has symptoms of the unstable fracture without neurological disorders, the operation should be performed after complete stabilization of the patient's condition. Contraindication to spine surgery is an extremely severe condition of the patient: shock, hemodynamic instability, coma (level of consciousness by a Glasgow coma scale of fewer than 10 points), multiple rib lesions with hemopneumothorax, and phenomena of respiratory failure (SpO_2 with oxygen insufflation less than 90 %) anemia (hemoglobin less than 90 g/l), sudden cardiac arrest with signs of heart failure, renal (anuria, oliguria, urea > 10 mmol/l, creatinine > 180 mmol/l) and/or hepatic failure (total protein < 45 g/l, increasing the enzyme twice or more), fat embolism, pulmonary embolism (PE), pneumonia.

Contraindication to surgery on the spine and spinal cord in the interim is the presence of purulent-septic complications and polyorganic insufficiency.

Surgical tactics in case of damage to the cervical spine (level C_3—C_7) should include anterior decompression, reposition, and fixation with the plate. If more than 2 vertebrae are damaged and there is a posterior compression, a single-step (if the patient's condition allows) or two-stage posterior decompression has to be performed with fixation of the vertebrae by contraction or transpedicular system.

In patients with injuries of the upper cervical spine (C_1—C_2), depending on the character of injuries, surgery has to be done in full — repositioning and stabilization in the Hallo-apparatus, posterior spinal fusion (spondylodesis) and internal fixation by contractions, craniocervical fixation, anterior spinal fusion (in case of damaging of C_2-vertebra) with an autobonegraftand a plate. In case of the severe condition of the patient, Hallo-fixation is recommended, with the necessary surgical treatment after stabilization of state.

In case of uncomplicated unstable injuries of the thoracic and lumbar spine divisions (anterior structures lesions), anterior spinal fusion with an autobonegraft and plate is performed. In the case of severe condition —

posterior transpedicular spinal fusion with subsequent (2—3 weeks) conducting anterior spinal fusionshould be done.

In case of complicated stable fracture of the thoracic and lumbar spine divisions, laminectomy, spinal cord revision, and posterior transpedicular fixation are indicated.

Posterior access:

1. Interlaminectomy (flavectomy) — removal of the ligamentum flavum.
2. Arcotomy (laminotomy) — partial resection of the arch, upper or lower edge.
3. Foraminotomy (facetectomy) — the opening of the intervertebral foramen by resection of the arcuate joint.
4. Hemilaminectomy — resection of half of the arch of the vertebra with retention of the spinous process.
5. Laminectomy — resection of the arch of the vertebra with the preservation of arcuate joints.

Independent work on the topic

Write the definition of the proposed terms:

1. Instability
2. Spinal shock
3. Spondylodesis
4. Laminectomy
5. Arcotomy
6. Phasetectomy
7. Interlaminectomy
8. Epidurography

Answer the question:

1. What groups are traumatic spinal and spinal cord injuries divided into?
2. Classification of traumatic injuries of the spine and spinal cord.
3. What are the clinical signs of spinal cord injury?
4. What are the clinical signs of traumatic injuries of the spine and spinal cord depending on the level of the lesion? Emergency care at the pre-hospital stage.
5. Which additional methods of examination are used in case of traumatic spinal cord injury?
6. What can cause spinal cord compression in case of traumatic injuries of the spine and spinal cord?
7. Recent principles of surgical treatment of spine and spinal cord injuries.
8. What are the early and late complications of traumatic spinal and spinal cord injuries?
9. Prevention of late complications in patients with traumatic injuries of the spine and spinal cord.
10. What types of treatment are used in the recovery period?
11. Ways to rehabilitate patients with traumatic spinal cord injury?

Tasks for self-control:

1. A 30 years-old patient, notes weakness and numbness of upper and lower extremities after an accident. Objectively, the examination revealed tetraparesis and sensitivity disorders on the upper and lower extremities. What is the level of spinal cord lesion?

a) cervical;

b) thoracic;

c) lumbar.

2. An 18 years-old patient was delivered by ambulance from a riverside zone. After diving, he was pulled out to the shore with gross movement disorders (tetraplegia). What additional methods of visualization are needed immediately to make the diagnosis?

a) radiography;

b) MRI;

c) CT;

d) myelography;

e) angiography.

3. In 28 years-old patient fracture-dislocation of Th_{12} was diagnosed. To clarify the nature of lesions, a lumbar puncture with CSF dynamics tests were performed. Whatchanges in CSF dynamics can be detected in the patient?

a) CSF-hypertension syndrome;

b) block of the subarachnoid space;

c) protein-cell dissociation;

d) cell-protein dissociation.

Topic 3

CEREBROVASCULAR DISEASES

Pathology of the cerebral blood vessels is now one of the most pressing and complex challenges of neurosurgery. Cerebrovascular disorders requiring neurosurgical intervention, include 1) cerebral aneurysms; 2) arteriovenous malformations; 3) carotid-cavernous fistulae; 4) acute cerebrovascular events: a) hemorrhagic stroke; b) ischemic stroke.

Cerebral aneurisms are local outward bulging of a thinned artery wall or a limited (possibly for a long time) enlargement of the inner lumen of the artery developing as a result of thinned or damaged artery walls.

Based on the configuration (shape) of an aneurysmal pouch, the following types of aneurysms are defined:

a) saccular («berry») aneurysms (look like small sacs), which have a neck, a sac, and a dome. The dome of an aneurysm is formed by a single layer of the intima and is therefore thinned; aneurysm rupturing occurs at the dome or sac, but never at the neck of an aneurysm; b) spherical aneurysms, with uniform or eccentric dilation of all layers of the arterial wall; c) fusiform (spindle-shaped) aneurysms, characterized with dilatation of a long portion of the artery lumen.

According to the size of an aneurysmal pouch, aneurysms are classified as:

a) miliary (linear size less than 3 mm); b) medium (4—15 mm); c) large (16—25 mm); d) giant (above 25 mm).

Clinical pattern. Certain phases can be reasonably defined in the clinical course of ruptured brain aneurysms:

1. *Pre-hemorrhagic phase* is usually symptom-free, and the first manifestations of the disease include episodes of sudden headache, dizziness, sometimes nosebleeds.

2. *Most acute hemorrhagic phase,* that starts with a sudden rupture of the dome of the wall of the aneurysmal pouch. It lasts for up to 3 days from the moment when the aneurysm ruptures and bleeding begins. At this stage, a generally healthy person suddenly experiences acute severe headache (as if someone hit his/her head), nausea, vomiting (sometimes multiple episodes), general weakness, bradycardia, and cardiovascular symptoms. Some common symptoms include fainting, generalized seizure, psychomotor agitation. Focal symptoms (develop in 50 % of cases) include anisocoria, oculomotor nerve damage, hemiparesis, hemiplegia, severe meningeal symptoms.

3. *Acute hemorrhagic phase* develops within the period from 4 days to 2—3 weeks. At this stage, the patient may develop bleeding, note an increase in the severity of focal symptoms, brainstem disorders due to cerebral edema, brainstem displacement and compression, gross vital sign abnormalities.

4. *Post-hemorrhagic phase:*

a) recovery period, this period begins in 2—3 weeks after subarachnoid hemorrhage and lasts up to 5—6 months. Fatal outcomes occur due to severe acquired disorders (pneumonia associated with abscesses, sepsis, etc.). After a period of 1 to 2—3 months recurrent hemorrhages may occur;

b) period of long-term effects, begins in 6 months after subarachnoid hemorrhage and lasts up to 5—10 years. Partial restoration of the disturbed brain functions is observed.

To evaluate the severity of a patient's clinical condition, W. Hunt and R. Hess (1968) proposed the following classification.

Classification of the severity of the clinical condition of patients in the acute phase of subarachnoid hemorrhages

Severity grade	Severity assessment criteria
I	Asymptomatic; mild headache or occipital muscle rigidity may be present
II	Moderate or mild headache. Marked meningeal syndrome. No focal neurological symptoms, except for possible oculomotor nerve disorders
III	Marked meningeal syndrome. Consciousness impairments up to obtundation. Moderate focal symptoms
IV	Marked meningeal syndrome. Stupor. Marked focal symptoms. Abnormal vital signs
V	Coma with various depth. Akinetic mutism

The main objectives of diagnosing a cerebral arterial aneurysm are to:

1) identify the aneurysm;
2) identify the aneurysm-bearing vessel and the exact location of origin of the neck of the aneurysm;
3) measure dimensions, determination of location and direction of the dome and the neck of the aneurysm;
4) determine the location of the aneurysm relative to the skull bones;
5) image the brain, brain vessels, and cerebrospinal fluid spaces;
6) choose appropriate treatment methods depending on the location and size of the arterial aneurysm;
7) perform planning and modeling of surgical intervention.

Diagnostic imaging techniques include ultrasound, spiral computed tomographic angiography (SCA), magnetic resonance imaging (MRI), and magnetic resonance angiography (MRA).

Transcranial Doppler ultrasound is one of the helpful ultrasound imaging techniques. Using this non-invasive diagnostic tool brain aneurysms can be diagnosed in 75—90 % of patients with intracranial aneurysms, and contrast media and three-dimensional scanning can increase this rate up to 97 %. However, aneurysms smaller than 6 mm cannot be detected by transcranial ultrasonography, and partially thrombosed aneurysms are also not always visualized.

Spiral computed tomographic (CT) angiography allows to visualize a non-thrombosed part of the aneurysm and determine its location and size. Small (less than 2 mm) aneurysms cannot be diagnosed with spiral CT-angiography.

Magnetic resonance imaging (MRI) is used for visualization of arterial aneurysms larger than 3—5 mm. In case of a ruptured aneurysm, an intracerebral hematoma is visualized on MR imaging and the presence of the aneurysm is determined by the MR signal.

Using ***magnetic resonance angiography (MRA)*** it is possible tomeasure dimensions, determine the location and perform differential diagnosis between a thrombus and presence of blood in the cavity of an aneurysm.

Cerebral angiography using the most state of the art using state-of-the-art angioseriographs is the most useful method providing the highest diagnostic accuracy. Timely performed cerebral angiography to detect arterial aneurysms is appropriate, given the catastrophic effects of acute cerebrovascular events that occur in patients with undiagnosed aneurysms.

Subarachnoid hemorrhage due to rupture of an aneurysm is not a contraindication for cerebral angiography. This test can be performed at any time after the occurrence of bleeding into the subarachnoid space to evaluate the need for urgent surgery. Using cerebral angiography, the neck and sac of the aneurysm can be detected using various non-standard views, especially oblique ones.

Indications for surgical treatment of arterial brain aneurysms include:

1) identified non-ruptured aneurysm larger than 10 mm;
2) history of an episode of intracranial hemorrhage caused by an arterial aneurysm;
3) oculomotor disorders caused by the presence of an aneurysm;
4) progressive enlargement of the arterial aneurysm.

The most radical treatment is by excluding the aneurysm sac from the circulation, which can only be achieved via surgery. There are currently two main methods of surgical treatment of cerebral arterial aneurysms, such as:

- intracranial intervention: a) aneurysm clipping; b) clipping of the aneurysm-bearing vessel;
- endovascular interventions: a) occlusion of the aneurysm cavity with a detachable balloon or filling the aneurysm cavity with a mixture of polymers; b) occlusion of the aneurysm cavity with microcoils; c) occlusion of the vessel containing the aneurysm.

Arteriovenous malformation (AVM) of the brain is a congenital anomaly of the blood vessels of the brain lacking capillary network, which results in a direct flow (bypass) of blood from arterial vessels into the system of superficial and deep veins. As a result, two circulatory systems are formed: 1) blood flowing through normal cerebral vessels and 2) part of the blood enters directly into the AVM bypassing the capillary network and does not participate in the blood supply to the brain. The most common localization of AVM is internal carotid arteries, less often AVM occurs in the vertebrobasilar system. AVM is responsible for 10 % of the causes of bleeding into the subarachnoid space, the overall incidence of AVM is 19 per 100.000 per year.

Classification of cerebral arteriovenous malformations

By location:

a) AVMs of deep brain structures and midline structures;

b) AVMs of the corpus callosum, subcortical ganglia, brain ventricles, thalamus, hippocampus;

c) subtentorial AVMs;

d) brainstem, cerebellar AVMs.

By size:

a) micromalformation (micro-AVMs) (with a volume of fewer than 2 cm^3);

b) small AVM (smaller than 5 cm^3);

c) medium-size AVM (5 to 20 cm^3);

d) large AVM (over 20 cm^3);

e) giant AVM (over 100 cm^3).

The proposed classifications should be used in practice.

The following stages are defined in the clinical course of AVM:

The prehemorrhage stage is characterized by the absence of clinical manifestations or the presence of epileptic seizures observed in 67 % of patients with AVM.

The hemorrhage (apoplexy) stage. Rupture of the abnormal wall of a blood vessel in an AVM leads results in subarachnoid, intracerebral, or mixed hemorrhage. At this, intracerebral hematomas form in 38 % of patients, subdural hematomas — in 15 %, and intraventricular hematomas — in 47 % of patients. The clinical pattern of AVM rupture depends on the location of the intracranial hematoma relative to the ventricular system of the brain.

Computed tomography (CT) is one of the most useful methods in AVM diagnosis, whereby AVM can be diagnosed in 91.7 % of cases. Even more useful is spiral (helical) computed tomography angiography (SCTA), which has a significant advantage over conventional CT. SCTA shows AVM in 92 % of cases.

A combination of spiral CT-angiography and transcranial Dopplerultrasound allows to diagnosis AVM in 92.9 % of cases.

At present, ***cerebral angiography*** and especially selective digital subtraction cerebral angiography is indicated for morphological characterization of AVM needed for radical surgical treatment, as none of the

modern non-invasive imaging methods can detect micromalformations, especially at the acute hemorrhagic stage. With this aim, cerebral angiography is indicated.

Methods of treatment of arteriovenous malformations

1. Surgical treatment: radical microsurgical extirpation of AVMs sized up to 100 cm^3.

2. Embolization: radical exclusion of AVM from circulation can be achieved in 10 to 40 % of patients, while in other patients (15 to 75 %) partial occlusion of the nidus can be performed.

3. Radiosurgery: 85 % of patients demonstrate a complete obliteration of the malformation with a diameter of less than 3 mm (at 2 years after radiosurgery).

Surgical treatment. The optimal treatment approach includes direct neurosurgical intervention with transcranial access. Preoperative embolization of hard-to-reach AVM afferent arteries should be considered to reduce the risk of postoperative complications. In the case of surgical treatment of AVM, especially in hard-to-reach areas, microsurgery can significantly reduce the risk of heavy bleeding due to minimally invasive removal of AVM.

Endovascular embolization of arteriovenous malformations. Total microsurgical resection of AVM is only possible in about 50 % of cases due to the large size of AVMs and/or localization of their veins in vital parts of the brain. In these cases, therefore, intravascular obliteration of AVM is often performed via the following methods:

1) permanent occlusion — balloon occlusion, occlusion of the feeding arteries of the AVM;
2) selective (superselective) embolization (filling of the nidus).

Currently, intravascular obliteration or filling of the AVM nidus via superselective embolization is performed mainly using hardening composite materials.

Carotid-cavernous fistula (CCF). The cavernous sinus has some specific topographic and anatomical features. The internal carotid artery passes through the sinus cavity surrounded by periarterial nerve plexuses. The walls of the cavernous sinus contain several branches of the trigeminal nerve (CN V): V3, V4, V6, as well as V1 and V2. Within the cavernous sinus, the internal carotid artery has two curves (called siphons) — anterior (with the convexity directed anteriorly), and posterior (with convexity directed posteriorly). Due to damages to the internal carotid artery at its cavernous segment, various carotid-cavernous fistulae (connections) are formed.

Most commonly, carotid artery damages occur as a result of traumatic brain injury. Slightly less often CCFs occur due to atherosclerosis, congenital anomalies of the internal carotid artery, and much less often — as a complication of infectious diseases. The ratio of traumatic and spontaneous CCF is 7:1. If the wall of the cavernous segment of the internal carotid artery ruptures, arterial blood enters directly into the sinus cavity leading to its significant enlargement.

The most typical clinical pattern of CCF includes a triad of symptoms:

1) ocular (aneurysmal) bruit (blood flow sounds coming from the eye);
2) pulsatile exophthalmos;
3) oculomotor nerve damage and impaired vision.

However, the gold standard for diagnosis of CCF and determination of the most appropriate treatment strategy is cerebral angiography. The angiography results provide information on the location and size of CCF,

state of cerebral hemodynamics, type of fistula (CCF formed in the internal carotid artery or as a connection between the internal and external carotid artery), and allow to exclude the presence of the internal carotid artery (ICA) deformation and arteriosclerotic plaque.

One of the challenges is to choose an appropriate method of treatment of the carotid-cavernous fistula. In the early stages of the CCF surgery, various methods were used: ligation of the carotid arteries in the neck, transcranial clipping of the internal carotid artery above the sphenoid bone, muscle embolization of the carotid-cavernous fistula (the Brooks method), or a combined method including ICA ligation, intracranial ICA clipping and then CCF embolization. Direct surgeries for CCF included fistula clipping or sinus tamponade.

Currently, the most promising method, and the method of choice, is CCF endovascular occlusion. This is performed using a detachable balloon catheter technique by F.A. Serbinenko or with microcoils. This method allows CCF to be excluded from the circulation, but at the same time maintains patency and integrity of the internal carotid artery.

Acute cerebrovascular events

Vascular pathologies of the brain and spinal cord, manifested by the development of strokes, are among the most prevalent disorders in the modern population with an incidence of 360 to 562 cases per 100.000 per year. According to the WHO, stroke is the number one cause of permanent disability and the third most common cause of death. Among women aged 30—49, the incidence of stroke is 12 per 100.000 persons, and among men aged 70—74 — 809 per 100.000 persons.

The stroke mortality rate during the first year reaches 39 %. High disability and mortality rates among patients with stroke make this type of pathology not only a medical but also a social problem. Therefore, to reduce the risk of acute cerebrovascular events, and the related disability and mortality rates, efficient prevention methods must be developed.

Ischemic stroke, or cerebral infarction, is a result of an acute cerebrovascular event with limited or completely obstructed arterial blood flow to the brain due to stenosis, thrombotic or embolic occlusion, angiospasm of the major blood vessels in the neck and arteries in the brain. The clinical pattern of ischemic stroke can be acute, subacute, chronic (pseudotumorous), and depends on the carotid artery affected, the location of the cerebral infarction, the state of collateral cerebral circulation. The clinical pattern of ischemic stroke is characterized by augmentation of focal neurological symptoms related to lesions of the cortical parts of the brain (aphasia, impairment of memory and consciousness, etc.), movement disorders (hemiparesis or hemiplegia), lesions of subcortical structures (hemianopia — in case of internal capsule lesions) and other symptoms.

The introduction of neuroimaging methods (CT, MRI) in clinical neurosurgery allowed a lifelong diagnosis of ischemic strokes, their clear differentiation from hemorrhagic strokes and other focal processes. Using ***CT*** imaging technique, a low-density area can be visualized, which is characteristic of cerebral infarction and is located in the area of the thrombosed cerebral artery. Usually, using CT, the focus of ischemic brain damage can be seen on the day 3 to 4 as a hypodense area with clear borders. In turn, ***MRI***, provides much more accurate information for the diagnosis, especially during day 1, in comparison with CT (20—30 %). Using MRI, foci of cerebral infarction can be identified, and their location, size, and shape can be determined. Also, lacunar (small) infarction areas can be

determined, as well as infarctions related to hemodynamic disorders in the areas of adjacent circulation. ***Transcranial Doppler ultrasound*** is one of the informative non-invasive methods for diagnosing ischemic strokes. This imaging technique allows to localize stenoses, occlusions of cerebral vessels and to determine the best treatment method and make the prognosis. A novel technique — digital subtraction angiography with selective injections of dye into the intracranial arteries — can also be used for diagnostic purposes.

Treatment of ischemic strokes. This condition requires a comprehensive treatment aimed at improving brain perfusion and oxygenation, microcirculation, rheological properties of blood, and brain metabolism, in addition to surgical intervention.

Surgical treatment of ischemic strokes. One of the modern approaches in the treatment of transient cerebrovascular events caused by intracranial artery stenosis includes treatment of stenosis in the pre-stroke stage to prevent ischemic strokes. One of the useful methods is the endovascular treatment of stenoses of intracranial and spinal arteries by angioplasty and placement of endoprostheses (stents) in the affected region (stenosis). Preventive measures aimed at eliminating transient cerebrovascular events include endarterectomy (removal of an atherosclerotic plaque) in patients with carotid artery stenosis. The presence of thrombosis and embolism or atheromatous plaques in the extracranial carotid artery is a direct indication for their surgical removal in the acute phase of a cerebral ischemic event (ischemic stroke).

Hemorrhagic stroke is intracerebral hemorrhage or subarachnoid hemorrhage, which occurs suddenly due to rupture of pathologically altered brain vessels in patients with hypertension and atherosclerosis. The incidence of hemorrhagic stroke in the population reaches 24.4 per 100,000. Among various diseases associated with acute cerebral circulation disorders, the proportion of hemorrhagic stroke is 30 % and its mortality reaches 50 %.

Location of the hemorrhagic stroke	**Frequency, %**
Basal ganglia	50
Brainstem	10
White matter	10
Thalamus	15
Cerebellum	15

The ***clinical pattern*** of hemorrhagic stroke often has a sudden onset, in a form of apoplexy, is accompanied by acute headache, vomiting, fainting, facial flushing, and respiratory dysfunction. Also, there are rapidly developing focal symptoms associated with general cerebral symptoms. Using modern neuroimaging techniques (CT, MRI) in clinical neurosurgery, a clear correlation was found between the development of focal symptoms and impaired consciousness and the volume of hematomas: with hematoma volume of fewer than 50 cm^3 the consciousness may remain normal, hematomas of 70—90 cm^3 are associated with stupor, with hematoma volume of more than 90 cm^3 coma occurs, and if the hematoma reaches a volume of 100—140 cm^3, displacement of the stem structures of the brain occurs with the subsequent development of severely abnormal vital signs.

Diagnosis

Using ***echoencephaloscopy*** in patients with massive intracranial hemorrhages, displacement of the stem structures of the brain (6 to 8 mm and more) can be visualized.

Computed tomography is one of the useful methods for diagnosing intracranial hemorrhages, that allows determining their location, size, position relative to the inner capsule (lateral, medial, or mixed), identify the presence of blood in the lateral ventricles (partial or complete tamponade), the degree of ventricle displacement, determine the hematoma density. Using CT scanning, the volume of hematoma can be accurately assessed to decide on surgical treatment.

To identify direct signs of a source of arterial bleeding in patients with intracerebral hemorrhage and to exclude hemorrhages caused by the presence of an arterial aneurysm or AVM, ***cerebral angiography*** is indicated.

The main indications for surgical treatment of intracerebral hemorrhages (hematomas):

1) lack of effectiveness of conservative treatment;

2) brain compression caused by intracerebral hematoma, the shift of midline cerebral structures of over 5 mm, marked perifocal edema;

3) lobar hematoma located laterally relative to the inner capsule and the volume of hematoma over 30 cm^3 (according to CT or MRI) are direct indications for surgery (first-choice treatment);

4) initial signs of displacement or edema of the brainstem according to clinical findings and CT and MRI results;

5) progressive worsening in cerebrovascular disturbances (based on results of transcranial Doppler, duplex ultrasound, MR angiography, posi-tron emission tomography);

6) a surgical procedure to remove a hematoma should be performed in the acute period (within the first hour or first 24 hours) to relieve the compression on the brain and prevent the development of displacement of the brainstem; this will help restore impaired brain function early;

7) intracerebral hematoma in the cerebellum is a direct indication for surgery.

Contraindications to surgical removal of intracerebral hematomas:

1) severe patient's condition — the stage of decompensation of cerebral blood flow, high blood pressure, deep coma;

2) marked signs of displacement of the brainstem manifested by severely abnormal vital signs (preagonal and agonal states);

3) severe concomitant pathology of different internal organs (kidneys, heart, liver, lungs) at the stage of decompensation.

Surgical treatment of intracerebral hemorrhage (hemorrhagic stroke) is primarily aimed at the total removal of the hematoma to eliminate to relieve the compression on the brain and prevent the development of displacement of the brainstem.

The main methods of surgery:

1) conventional craniotomy according to the location of the intracerebral hematoma;

2) standard local aspiration of hematoma through a burr hole;

3) minimally invasive removal of intracerebral hematoma:

a) stereotactic aspiration;

b) endoscopic hematoma evacuation.

One of the promising methods of treatment is the minimally invasive endoscopic removal of intracerebral hematomas. For this, minimal trepanation is performed, with a diameter of burr holes of 2.0 to 2.5 cm. The hematoma can be removed through one hole (monoportal surgery) or two holes (biportal surgery).

Scales for assessing the condition of patients with stroke and predicting the further course of the disease are provided below.

Scale for assessment of impairment of conscious level

(Glasgow Coma Scale)

Response	**Score (for positive response)**
1. Eye-opening response:	
eye open spontaneously	4
eyes open to verbal command	3
eye open to pain	2
no eye-opening	1
2. Motor response (applied to the less affected side):	6
obeys commands to movement	
localizes to pain (purposeful movements towards	5
painful stimuli)	4
flexion/withdrawal to pain	3
abnormal flexion to pain	2
abnormal extension to pain	1
no motor response	
3. Verbal response:	
oriented	5
confused speech	4
inappropriate words (no conversational	3
exchange)	2
incomprehensible sounds	1
no verbal response	
Glasgow Coma Scale score range: highest — 15; lowest — 3	

Levels of consciousness according to the interpretation of the Glasgow Coma Scale score

Level of consciousness	Glasgow Coma Scale score
Clear consciousness	15
Moderate obtundation	13—14
Deep obtundation	11—12
Stupor	9—10
Grade I coma (moderate)	7—8
Grade II coma (deep)	5—6
Grade III coma (severe)	3—4

Module 3 Quiz

Write the definitions for the following terms:

1. AVM.
2. Acute cerebrovascular events (stoke).
3. Hemorrhagic stroke.
4. Arterial aneurysm.
5. CCF.
6. Ischemic stroke.
7. Vascular stenosis.
8. Vascular occlusions.
9. Angioplasty.
10. Stenting (intravascular angioplasty).

Write answers to the following questions:

1. Name the major blood vessels that supply the brain, and describe their topographic anatomy.
2. What are the main pathogenic mechanisms of hemorrhagic and ischemic strokes? List the major causes of hemorrhagic and ischemic strokes.
3. Describe the etiology and pathogenesis of AVM, arterial aneurysm, CCF.
4. Name the methods of clinical and laboratory diagnosis of cerebrovascular disorders.
5. What findings of additional diagnostic testing suggest AVM, arterial aneurysm, CCF, hemorrhagic or ischemic strokes?
6. What scales are used to evaluate the severity of acute hemorrhagic cerebrovascular events?
7. What criteria are used for the differential diagnosis of different acute cerebrovascular events?
8. What are background therapy and emergency treatment of patients with acute cerebrovascular events?
9. What are the indications for emergency surgery for cerebrovascular disorders? Name the basic principles of such interventions.
10. What prognosis can be made for cerebrovascular diseases (AVM, arterial aneurysm, CCF)? Explain the development of the main complications and consequences of vascular lesions affecting the central nervous system.

11. Principles of postoperative treatment and rehabilitation of patients with vascular pathology of the CNS.

Tests for self-assessment

1. A 43-year-old female patient fainted and fell while working in the garden. Upon admission to the clinic, the patient's condition was assessed as critical. According to her relatives, she had been generally healthy so far, did not have any complaints. No traumatic injuries were found on the skull. No focal neurological symptoms were present. Impaired consciousness — stupor, stiffness of the suboccipital muscles. Blood pressure 200/110 mm Hg. Cerebrospinal fluid is intensely stained with blood, cerebrospinal fluid pressure is 200 mm H_2O. What is the most likely cause of the patient's condition?

2. A 18-year-old male student fainted during his physical education class, developed seizures, mostly in the arm and leg on his right side. The patient was delivered to the hospital's admission office. Examination revealed impaired consciousness classified as grade I coma, brisk tendon reflexes, and presence of pathological reflexes, periodic tonic spasms in the extremities («hormetonic» spasms), severe suboccipital muscle tension, positive Kernig's and Brudzinski's sign. Blood pressure 170/100 mm Hg. What is the most likely diagnosis?

3. A 63-year-old female patient has been followed-up by a primary care physician for the last 15 years for type II hyperlipidemia, aortic atherosclerosis, and coronary atherosclerosis. In the morning she could not get out of bed: she developed a deep left-sided hemiparesis (mostly in the arm) and left-sided hemihypesthesia. The examination revealed brisk tendon reflexes on the left side, and pathological reflexes on the left side, blood pressure 130/70 mm Hg. What is the most likely diagnosis?

4. A 49-year-old female patient has had a closed brain injury. One week after the injury, a protrusion of the right eyeball began. The patient complained of a constant sound in the head, similar to the sound of the train. Redness of the right half of the face is observed, dilated scleral blood vessels, conjunctival hyperemia in the right eye. What is the most likely diagnosis?

5. A 36-year-old male patient fainted and fell while working in his garden. Upon admission to the clinic, the patient's condition was assessed as critical. According to her relatives, he had been generally healthy so far, did not have any complaints. No traumatic injuries were found on the skull. No focal neurological symptoms were present. Impaired consciousness — stupor, stiffness of the suboccipital muscles. Blood pressure 200/110 mm Hg. Cerebrospinal fluid is intensely stained with blood, cerebrospinal fluid pressure is 200 mm H_2O. Preliminary diagnosis: ruptured arterial aneurysm. Which diagnostic method is the most useful in this case?

6. A 18-year-old male student fainted during his physical education class, developed seizures, mostly in the arm and leg on his right side. The patient was delivered to the hospital's admission office. Examination revealed impaired consciousness classified as grade I coma, brisk tendon reflexes, and presence of pathological reflexes, periodic tonic spasms in the extremities («hormetonic» spasms), severe suboccipital muscle tension, positive Kernig's and Brudzinski's sign. Blood pressure 170/100 mm Hg. Preliminary diagnosis — Cerebral AVM. Which diagnostic method is the most useful in this case?

7. A 43-year-old female patient has had a closed brain injury (concussion). One week after the injury, a protrusion of the right eyeball began. The patient complainedof a constant sound in the head, similar to the sound of the train. Redness of the right half of the face is observed, dilated scleral blood vessels, conjunctival

hyperemia in the right eye. The most likely diagnosis is a carotid-cavernous fistula. Which diagnostic method is the most useful in this case?

8. A 52-year old female patient presented with complaints of dizziness, shaky legs when walking, periodic double vision, nausea. Periodic elevated blood pressure up to 160/95 mm Hg. Also, the patient's complaints included constant dull pain in the cervical spine and discomfort in the neck. Dizziness worsens at turns and inclinations of the head, and everything goes dark. The patient has experienced the onset of symptoms about 4 years ago and since the symptoms have been progressing. What is the most likely diagnosis?

9. A 48-year old male driver presented with complaints for dizziness, shaky legs when walking, periodic double vision, nausea. Periodic elevated blood pressure up to 160/95 mm Hg. In addition, the patient's complaints included constant dull pain in the cervical spine, and discomfort in the neck. Dizziness worsens at turns and inclinations of the head, and everything goes dark. The patient has experienced the onset of symptoms about 4 years ago and since the symptoms have been progressing. Preliminary diagnosis: vertebrobasilar insufficiency. Which diagnostic method is the most useful in this case?

10. A 60-year-old male patient has been followed-up by a primary care physician for the last 15 years for essential hypertension. One day, the patient passed out and developed a deep left-sided hemiparesis. The examination revealed brisk tendon reflexes on the left side, and pathological reflexes on the left side, blood pressure, blood pressure 190/100 mm Hg. What is the most likely diagnosis?

Topic 4

TUMORS OF THE BRAIN, SPINAL CORD. PATHOLOGY OF INTERVERTEBRAL DISCS

Brain tumors

Early diagnosis of brain tumors, the choice of adequate treatment tactics, and improvement of the quality of the life of patients with brain tumors are some of the most relevant problems of modern neurooncology. Tumors of the area above the cerebellar tentorium reach 20—45 % in children and 64—76 % in adults. According to various authors, tumors of glial origin occur in 45.6 %, meningioma — in 15—27.9 %, pituitary adenomas in 12.2 %, and auditory nerve neurinoma in 4.9 %. At the same time, glial tumors prevail in men, while meningioma and neurinoma prevail in women. In patients older than 70 years, the predominate tumor is meningioma (up to 40 %) (V.P. Bersnev, 1990; D.S. Russel, L.J. Rubinstein, 1989).

Despite the lack of a unified theory of the development of the oncogenesis process, scientists identify several risk factors for brain tumors (D.A. Gulyaev, 1999; P. Inskip, 1995; Batra et al., 1994; M. Preston, 1996):

1. Genetic factor — mutations in chromosomes 9 (9p), 10 (10q), 13 (13q), 17 (17p), 19 (19q), 22 (22q) under the influence of adverse factors as well as congenital genetic defects.

Genetically determined diseases and brain tumors arising at their background

Syndrome	Probable concomitant brain tumor	Damaged gene
Neurofibromatosis (type 1)	Astrocytoma, peripheral neurofibroma	17q
Neurofibromatosis (type 2)	Neurinoma of the VIII nerve, meningioma, astrocytoma, ependymoma	22q
Tuberous sclerosis	Subependymal astrocytoma	9q
Li—Fraumeni syndrome	Glioblastoma, other kinds of glioma	17p
Hippel—Lindau syndrome	Hemangioblastoma of the cere-bellum, astrocytoma	3p

2. Effect of unfavourabl5e environmental factors (ionizing radiation, electromagnetic fields, high content of pesticides in the body, the presence of carcinogens within industrial conditions, such as benzene, chloroprene, lead and others, air pollution, etc.).

3. Presence of oncogenic viruses in the body (including Epstein—Barr virus, Human papillomavirus of the 16th and 18th types, Human immunodeficiency virus, etc.).

4. Unhealthy lifestyle (bad habits, including dietary factors).

5. Craniocerebral injuries in past medical history.

6. The presence of a tumor in parents or close relatives.

Classification of brain tumors

The main classifications of brain tumors are the classifications by P. Bailey, H.A. Cushing, (USA, 1926), L.I. Smirnov (USSR, 1962), B.S. Khominsky (USSR, 1969), the WHO (1993), V.V. Yarzew, D.E. Matsko, A.G. Korshunov (Russia, 1998), Y.P. Zozula, T.P. Verkhoglyadova, M.I. Shamayev (Ukraine, 2001), D.E. Matsko (Russia, 2001) and some others. Having adopted these classifications, it is possible to distinguish particular variants of brain tumors.

Classification of brain tumors by location

Relative to the cerebellar tentorium:

1) supratentorial;
2) subtentorial.

Relative to the surface of the brain:

1) convexity tumor;

2) basal tumor.

Relative to structures of the brain:

1) intracerebral (tumors of the glial origin, pineal gland, pituitary gland, cerebral vessels, etc.);

2) extracerebral (originate from the roots of the cranial nerves, meninges, adjacent soft tissues, bones, etc.).

Tumors of double localization:

a) supra-subtentorial;

b) craniospinal;

c) extra-intracranial.

By quantity:

1) single;

2) multiple (metastatic, neurinomas, meningiomas in neurofibromatosis).

By origin:

1) primary;

2) secondary (metastatic).

By etiology:

1) congenital (which manifested in clinical symptoms within the first 60 days after birth);

2) acquired.

Classification of brain tumors by histological structure:

I. Tumors of neuroectodermal origin:

— glial origin: astrocytoma; oligodendroglioma; ependymoma; mixed forms;

— low-differentiated and undifferentiated glial tumors: glioma, glioblastoma; medulloblastoma.

II. Tumors of the sheath of the cranial and spinal nerves:

— schwannoma (Schwann cell tumor, neurilemoma, neuroma);

— mixed variants: neurofibroma, neurofibrosarcoma, etc.

III. Tumors of mesenchymal origin:

— tumors of the meninges: meningioma (arachnoid-endothelioma);

— vascular tumors (angioma, angiocavernoma, hemangioma, hemangiosarcoma, hemangioreticuloma, etc.);

— primary malignant lymphoma;

— tumors of adjacent tissues (chondroma, chordoma, sarcoma, lipoma, osteoma, osteoblastoclastoma, etc.).

IV. Pituitary adenoma: acidophilic, basophilic, chromophobic, mixed, prolactinoma.

V. Craniopharyngioma.

VI. Tumors of the pineal gland (pinealoma, pineocytoma, pineoblastoma).

VII. Congenital tumors (teratoma, epidermoid, dermoid, heterotopic tumor, germinoma, embryonic cancer, etc.).

VIII. Metastatic tumors (primary source — lung cancer, breast cancer, hypernephroma, melanoma, etc.).

The clinical picture of brain tumors

1. General cerebral symptoms: hypertension syndrome: pre-morning headache, nausea, vomiting, numbness (blurred vision, double vision or loss of peripheral vision), dizziness, disturbance of consciousness, changes in the eye fundus, signs of hypertension on craniography. In children the development of hypertension-hydrocephalus syndrome due to tumor occlusion of the pathways through which cerebrospinal fluid flows is typi-cal.

2. Focal symptoms: a) primary focal; b) secondary focal — in the nearby area and distanced (dislocation syndromes).

3. Intoxication symptom (possible in the case of primary malignant and metastatic tumors).

The mechanism of formation of the hypertensive syndrome

According to the Monro—Kellie Doctrine, brain tissue that is contained in a closed craniospinal system (after the cranial sutures have healed and the temples have closed), cerebrospinal fluid and blood are non-compressible components. If the volume of one of these components changes, for example, if the volume of cerebrospinal fluid increases, then either one of the other components changes or the pressure of the cerebrospinal fluid increases.

An integrated indicator of the state of multicomponent intracranial content is intracranial pressure (ICP), which is due to the levels of intracerebral, interstitial (intermediate) pressure, and cerebrospinal fluid pressure; the ratio is normally 2 : 1 : 0.1 respectively (E.B. Syrovsky, 1984). In clinical practice, ICP is usually associated with cerebrospinal fluid pressure (the normal rate is 100—200 mm Hg; ICP is measured in the patient's supine position).

In the case of new component development, such as a tumor, this ratio is broken. In the case of supratentorial localization of the process, there are violations of the regulation of cerebral circulation, especially violation of venous outflow and a decrease in subsequent perfusion pressure and volumetric blood flow velocity. Simultaneously, edema-swelling of the brain substance is formed due to the release of the liquid part of the blood extracellularly and an increase of the amount of intracellular fluid. A sharp increase in the spinal cord fluid leads to a violation of its outflow and its normal resorption in the system of Pachyonic granulations of the arachnoid membrane of the brain — thus, hydrocephalus is formed. In the case of intraventricular location of the tumor or growth of the tumor into the ventricular system, disorders of cerebrospinal fluid outflow and the formation of occlusive hydrocephalus are the primary links in the formation of intracranial hypertension.

Enhancement of the headache in patients with brain tumors at night and in the morning can be explained by increased venous stasis due to a decrease in heart rate at night due to vagal nerve activation, mechanical impairment of cerebrospinal fluid outflow, and physiological increased secretion of cerebrospinal fluid at night. In the case of subacute and chronic development of hypertension syndrome at the background of the growth of brain tumors (mostly benign), changes in the eye fundus and skull bones can be noticed.

Ophthalmic manifestations of hypertension syndrome:

- persistent angiodystonia of the retinal vessels;
- stagnant optic discs (swelling of the optic nerve disc);

- secondary optic nerve atrophy.

Craniographic signs of hypertension syndrome:

- osteoporosis of the back of the Turkish saddle;
- enhancement of impressions of cerebral gyri in the bones of the skull;
- enhancement of an internal texture and expansion of foramina in skull bones;
- in children of the first year of life — split up of sutures, thinning of the bones of the skull, increase in the size of the skull.

The rate of growth of hypertensive syndrome depends on the location of the tumor and its histological structure.

The most typical primary focal symptoms and histological variants of neurotumors

Localization	Symptoms	Tumor type
Frontal lobe	Personality changes, cognitive impairment, apathy syndrome, epileptical syndrome, hemiparesis, hypo- and anosmia, motor aphasia, frontal ataxia, astasia-abasia	Meningioma (nasal or olfactory fossa, alae majores of the sphenoid bone, parasagittal area); astrocytoma, glioblastoma, oligodendroglioma, metastases (lung cancer, breast cancer, melanoma)
Temporal lobe	Generalized convulsions, often with auditory auras, sensory and amnestic aphasia, homonymous hemianopsia, impaired musical hearing, etc.	Meningioma alae majores of the sphenoid bone, glioblastoma, oligodendroglioma, astrocytoma, metastases (kidney cancer, stomach cancer)
Parietal lobe	Partial epileptic seizures with a sensitive component, disorders of simple and complex types of sensitivity, astereognosis, agraphia, alexia, acalculia, apraxia	Meningioma (of processus falciformis, convexital type), glioblastoma, oligodendroglioma, metastases (cancer of lungs, chest, kidney; melanoma)
Occipital lobe	Homonymous visual field disorders, visual symptoms, photopsia	Meningioma (parasagittal, tentorial), glioblastoma, metastases (lung, breast, kidney cancer)

Localization	Symptoms	Tumor type
Brain ventricles	Hypertensive crises (sharp increase in ICP); occlusive hydrocephalus	Ependymoma, vascular papilloma, epidermoid, dermoid, meningioma of tentorium, colloid cyst
Basal ganglia	Hemiparesis, thalamic syndrome	Astrocytoma, oligodendroglioma, glioblastoma, metastases (lung cancer, breast cancer)
Midbrain	Endocrine disorders (hypo-thalamus), Parinaud's syndromes, paresis of cranial nerves, occlusive hydrocephalus (increased ICP)	Glioma (in children), pinealoma
Brain stem	Dysfunction of cranial nerves (the earliest symptoms are nystagmus, diplopia), impaired sensitivity, motor function, symptoms of increased ICP (occlusive hydrocephalus)	Glioma, pilocytic astrocytoma in children, astrocytoma, metastasis (breast cancer, lung cancer, kidney cancer), sublingual nerve neurinoma, hemangioblastoma, tumors of the cerebellar slope (chordoma, meningioma, epidermoid, chondroma)
Cerebellum	Hemiataxia, falls, hypotension, dysmetria, a sharp increase in ICP (occlusive hydrocephalus)	Spongioblastoma and medulloblastoma in children, ependymoma of the IV ventricle, hemangioblastoma, metastases (stomach cancer, lung cancer, breast cancer)
Ponto-cerebellar angle	Tinnitus, deafness, compression of VII and V pairs of cranial nerves, hemiata-xia, headache, a sharp increase in ICP	Neurinoma of the auditory nerve, meningioma, dermoid, trigeminal neurinoma (rare). Differential diagnosis with AVM, giant aneurysm, etc.

Localization	Symptoms	Tumor type
Turkish saddle	Endocrine disorders, bitemporal hemianopsia, primary optic nerve atrophy	Pituitary adenoma, craniopharyngioma, Turkish saddle tubercle meningioma, epidermoid, rarely aneurysm, glioma of the optic nerve

Dislocation syndromes

In the case of progression of the hypertension syndrome, patients with brain tumors develop dislocation syndromes, which are the main cause of worsening of the patient's condition and subsequently — their death. The main mechanism of the formation of these syndromes in the case of the increase in the tumor size is the appearance of an area in the brain with a pressure gradient, resulting in a shift of some parts of the brain relative to the other parts and the processes of the dura mater. Critical areas for the likely pinching of adjacent parts of the cerebral hemispheres or cerebellum and compression of adjacent parts of the brain stem or middle structures are the gaps between the lower edge of the cerebral sickle and the corpus callosum, the gap between the edge of the cerebellar tentorium and midbrain, funnel of dura mater in the area of the occipital foramen magnum. Among the various variants of dislocation syndromes, the most prognostic are the four ones (B.S. Khominsky, 1962):

- temporal-tentorial;
- herniation of the cerebellar tonsils in the occipital-cervical-dural funnel;
- herniation of the medial parts of the frontal and parietal lobes under the crescent-shaped process;
- cerebellar-tentorial.

The first two types are the most clinically significant and life-threatening variants for patients with brain tumors and other volumetric lesions.

Temporal-tentorial herniation is most often observed in the presence of pathological processes within the temporal lobe. Morphologically, it is a protrusion of the medial parts of the para-hippocampal torsion and adjacent parts of the brain into the tentorial foramen. At the same time, the expressed changes are observed in temporal, occipital lobes and midbrain: small in size as well as drain hemorrhages, often — compression of a water supply system of a brain with the development of secondary ischemia in the basin of a posterior cerebral artery. Such changes occur both on the side of the pathological process and the opposite side — due to compression of the contralateral truncus cerebri and arteries that pass near the tent of the cerebellum.

Clinically, the temporal-tentorial herniation is characterized by symptoms of a sharp increase in intracranial pressure with signs of midbrain damage:

- a sharp increase in headache, accompanied by vomiting;
- an increase in contralateral hemiparesis, which changes to bila-teral;
- progressive disturbance of consciousness;
- diencephalic disorders (tachycardia, bradycardia, tachypnea, hyperthermia, etc.);

- development of Weber alteration syndrome, paresis of the upward gaze, positive Hertwig-Magendie symptom, weakening or absence of pupillary reaction to light;
- development of decerebration rigidity, hormetonia.

Herniation of the cerebellar tonsils into the occipital-cervical-dural funnel is more often observed in the case of subtentorial pathological processes, although it is often a symptom of the terminal stage of axial dislocation of the brain due to supratentorial volumetric lesions of the brain or occlusive hydrocephalus. The tonsils compress the medulla oblongata, causing the development of severe disorders of its functions, which leads to the death of patients.

The most characteristic clinical symptoms of this type of herniation are:

- a sharp increase in hypertension syndrome;
- progression of disorders of consciousness (up to coma II—III);
- the appearance of ponto-mesencephalic disorders (miosis, which is replaced by bilateral mydriasis, paresis of the gaze upwards, stem nystagmus, etc.) and vital disorders (tachycardia, apnea).

Diagnosis of dislocation syndromes is performed based on the evaluation of clinical symptoms and CT data, MRI of the brain.

Features of clinical manifestations of brain tumors in children

Brain tumors rank the second place in prevalence among other pediatric cancers. 81—90 % of tumors in children are intracerebral; they are located mainly along the midline. Supratentorial tumors predominate in children of the first year of life, and tumors of the posterior cranial fossa are mostly seen in children from 1 to 5 years. Tumors of the posterior cranial fossa account for up to 70—83 % of all tumors in children, the main histological type is medulloblastoma of the cerebellum (2/3 of medulloblastomas occur in males) and tumors of the brainstem (up to 8.75 %). In children, tumors of neuroectodermal origin predominate, accounting for 70 % of the total number of neoplasms (A.E. Walker, 1985; D. Schiffer, 1993; I. Pollack, 1995). Astrocytoma and medulloblastoma of the cerebellum, ependymoma, glioma of the brainstem, craniopharyngioma, tumors of the pineal body, glioma of optic nerves, etc. are most common in children. The clinical picture of brain tumors in children is characterized by the development of the hypertension-hydrocephalus syndrome.

Symptoms of brain tumors in newborns and young children: progressive macrocephaly, often asymmetry of the skull, suture split up, bulking of the fontanelle; increased excitability; vomiting; insufficient weight gain; lag in psychomotor development; swelling of the optic disc, decreased visual acuity; focal neurological symptoms; convulsive seizures.

Symptoms of brain tumors in older children: signs of hypertension: headache; visual disturbances (double vision, «blurring», decreased visual acuity); vomiting; weight loss; among the focal symptoms static, coordinating and visual disorders, dysfunction of the cranial nerves and convulsions are predominating.

Brain tumors are often located under the tent of the cerebellum in children are disguised as gastrointestinal pathology, worm infestation, and so on.

Diagnosis of brain tumors

1. General examination and neurological examination of a patient with an assessment of the functional activity of oncologic patients according to the Karnowski scale (1948), adapted for neurooncology patients by E.O. Grigoriev (1994).

2. General clinical laboratory research methods:

— general analysis of blood and urine, biochemical analysis of blood;

— general and biochemical analysis of cerebrospinal fluid, special studies of cerebrospinal fluid (according to the indications);

— determination of the content of pituitary and adrenal hormones (in the case of suspicion of a pituitary tumor);

— immunocytochemical analysis of blood (cerebrospinal fluid, saliva) to detect DNA-oncogenic viruses; viruses from the group of TORCH infections, AIDS virus or antibodies to them, etc.;

— genetic research.

3. Ophthalmologist consultation.

4. X-ray of the skull (craniography).

5. Echoencephalography.

6. Computed tomography of the brain.

7. MRI of the brain (including contrast, functional MRI, MR-spectroscopy, MR-angiography, MR-thermography, etc.). In the case of malignant tumors (especially tumors of the posterior cranial fossa in children) it is advisable to perform MRI of the spinal cord to exclude meta-stasis along the cerebrospinal canal of the spinal cord.

8. Positron emission tomography (PET).

9. Single-photon emission computed tomography (SPECT).

10. Angiography (carotid, vertebral, selective, total, superselective, etc.).

11. Electroencephalography (EEG) (analog, computerized EEG with mapping, EEG monitoring, MR electroencephalography, etc.).

12. Radioisotope methods (scintigraphy, radioisotope angiography, etc.).

13. Doppler imaging of the vessels of the neck and brain.

14. If necessary, corticography, subcorticography, determination of evoked brain potentials, electroneuromyography of peripheral nerves, etc.

15. Tumor biopsy (stereotactic, open, or using endoscopic technique under the control of PET or MRI spectroscopy, especially in the case of a tumor that is isodense to the brain substance) to determine the histological type of tumor, the stages of the process according to the WHO system. An immunohistochemical study with determination of proteins S-100, GFAP, cytokeratin, CD^+ 20, 45 (in the case of suspicion of lymphoma) is performed.

16. Genetic research (molecular-biological).

If necessary — consultations of a therapist, an otolaryngologist, an endocrinologist, an obstetrician-gynecologist, a psychiatrist, a maxillofacial surgeon, and other related specialists.

Treatment of brain tumors

I. ***Surgical treatment*** using microsurgical, navigation, laser, ultrasound, cryogenic, endoscopic, stereotactic techniques, etc.

1. Radical — aimed at maximal removal of the tumor: total (complete removal of the tumor), subtotal (with the removal of up to 80 % of the tumor), partial (with the removal of up to 50 % of the tumor), and tumor biopsy (with the removal of up to 20 % of the tumor).

Radiosurgical destruction of the tumor is a method of high-frequency irradiation of a pathological intracranial lesion using a large dose of radiation (from 20 to 100 Gy in the isocenter) at one time using stereotactic techniques through an intact skull. Gamma knife, linear accelerator, cyclotron, cyberknife, Novalis system, invasive tumor radiosurgery, etc. can be used to treat brain tumors.

Thermal destruction of the tumor (laser thermal destruction, cryodestruction, etc.). One of the conditions for the effectiveness of surgical treatment of brain tumors is adequate preoperative preparation, which includes assessment and correction of somatic pathology, reduction of cerebral edema and manifestations of hypertension (administration of corticosteroids, in some cases — osmodiuretics, correction of the convulsive syndrome, etc.).

In the treatment of meningioma, which in the case of a typical location is well vascularized by the branches of the middle carotid artery from the basin of the external carotid artery, and in the case of frontonasal location — by the branches of the ethmoidal and ocular arteries from the basin of the internal carotid artery, it is desirable to carry out preoperative embolization of the vessels which supply blood to the tumor, to reduce bleeding during surgical removal of the tumor. Preoperative embolization of vessels that supply blood to the tumor is also performed in the presence of some tumors of osteogenic, meningovascular origin, which are located mainly at the base of the skull.

2. Palliative — symptomatic treatment, aimed primarily at reducing the manifestations of hypertension:

a) improvement of cerebrospinal fluid outflow (cerebrospinal fluid bypass operations — ventriculoperitoneostomy, ventriculocysternostomy, ventriculocardiostomy, etc.);

b) reduction of cerebral edema (decompression operations — internal and external decompression of the brain).

II. **Radiation therapy** (traditional, hyperfractional, photodynamic therapy, brachytherapy, boron-neutron capture therapy, etc.). It is optimal to assign a total radiation dose of up to 60 Gy. The course of radiation therapy as an independent type of treatment or as a component of combined treatment of brain tumors is prescribed 2 weeks after partial or complete removal of the tumor for 6 weeks. with fractional daily irradiation with a dose of 180—200 mGy. If the size of the tumor is small, the total radiation dose maybe 30 Gy with 10 fractional irradiation for 2 weeks. (L.R. Cola, 1992). Regarding the application of radiation therapy in the treatment of brain tumors of metastatic origin, the tumor size up to 3 cm is considered to be optimal for its implementation. Tumors of the lung, breast, and lymphoma are most radiosensitive; melanoma, sarcoma, and papilloma are in most cases radioresistant.

One of the modern methods is intermediate brachytherapy: a radioactive implant is inserted into the tumor and during the period of decay of the radioactive element the tumor is destroyed. One of the disadvantages of this

method is significant radiation damage to the entire brain, the appearance of a zone of radiation necrosis, requiring further surgical removal of this area under the guise of steroids.

The most radiosensitive brain tumors are malignant glioma, oligodendroglioma (in the case of subtotal resection or anaplastic variant), dysgerminoma, primary CNS lymphoma, medulloblastoma, ependymoma, meningioma (malignant, non-operable, not fully removed), pituitary adenoma (after subtotal removal or after ineffective drug therapy), chordoma of the skull base (N.E. Polishchuk, S.Y. Rasskazov, 1998).

III. ***Chemotherapy*** with systemic, regional, intra-arterial (selective), intrathecal, interstitial administration of chemotherapeutics and the use of Ommaya reservoirs with mandatory prior chemosensitivity testing. The most chemosensitive tumors are malignant gliomas, primary CNS lymphomas, tumor infiltration of the meninges, and so on.

The main chemotherapeutics used in brain tumors are derivatives of alkylating agents and platinum compounds. For example, for the treatment of medulloblastoma in children, the most traditional chemotherapy regimens are: vincristine + lomustine + prednisolone + radiation therapy; cyclophosphamide + cisplatinum + vincristine; cyclophosphamide + vincristine + cisplatin + etoposide. The use of the chemotherapeutic drug temodal is promising for undifferentiated gliomas and glioblasts.

IV. ***Hormone therapy:***

- to reduce cerebral edema (usual dexamethasone): in the preoperative period 10 mg + 4 mg every 6 hours under the guise of H_2-blockers (famotidine 20 mg or ranitidine 150 mg);
- replacement, corrective therapy for hormone-dependent tumors.Í

V. ***Immunotherapy*** (specific, nonspecific, cellular, cytokine, combined, etc., with the introduction of monoclonal antibodies, antitumor vaccines, etc.).

VI. ***Gene therapy*** (introduction of genetically modified viruses).

The criterion for the effectiveness of surgical treatment of brain tumors is the maximal preservation of the patient's quality of life, which means the overall characteristics of the physical, psychological, emotional status of the patient, as well as his social adaptation, financial and spiritual well-being.

For the treatment of dislocation syndromes, it is necessary to apply a set of intensive conservative and surgical measures. Undoubtedly, the best measure is to eliminate the root cause, which led to the development of dislocation syndrome, i.e. removal of the tumor. Temporary measures should include puncture and drainage of the ventricular system; creation of external decompression of the brain by decompression trepanation of the skull, less often — tentoriotomy (in the case of temporal-tentorial herniation), reclination (in the case of herniation of the cerebellar tonsils in the occipital-cervical-dural funnel), etc.

Auditory nerve neurinoma is a primary brain tumor that grows predominantly from the upper vestibular branch of the parietal nerve in the pons-cerebellar angle. Auditory nerve neurinoma accounts for up to 10 % of all brain tumors and occurs mainly in women in their fourth decade of life and older. Bilateral auditory nerve neurinomas are most often a manifestation of neurofibromatosis type 2.

Topographic and anatomical features of auditory nerve neurinoma. The seventh and eighth pairs of cranial nerves start from the brain stem, being located close to each other. They pass laterally from the pontine and the cerebellum and enter the internal auditory canal in a certain sequence. When the neurinoma of the auditory nerve

is formed, mainly from the upper vestibular portion of the auditory nerve, the facial nerve is displaced forward or upward. Histologically, neurinomas are formed in the transition zone of the central part of the myelin sheath, which is formed by oligodendrocytes, and the area of peripheral myelin, which is formed by Schwann cells. Auditory nerve neurinomas are called schwannomas.

Main clinical symptoms:

- hearing loss (gradual, progressive);
- tinnitus (often highly frequent);
- dizziness (as a result of vestibular dysfunction);
- facial nerve dysfunction (large tumors or intracanal tumors compress the facial nerve);
- in the case of neuromas of the big sizes the hypertensive syndrome develops, appearance of other focal symptoms is possible.

Diagnosis:

- audiometry (the specific feature is the sensorineural hearing loss at the pitch);
- CT of the brain;
- MRI of the brain (including contrast).

Differential diagnosis is performed with meningioma, epidermoid, metastatic tumor, neurinoma of other cranial nerves.

Treatment. *Stereotactic radiosurgery* (Gamma knife, cyberknife, X-knife, linear accelerator, Novalis) is indicated in the treatment of small neurinoma; such a method preserves the integrity of the cranial nerves, is the method of choice in patients with contraindications to open surgery. Preference should be given to radio-surgical techniques for bilateral auditory nerve neuromas, type 2 neurofibromatosis, as the main purpose is to preserve hearing.

Surgical removal of the tumor (suboccipital, retrosigmoid access, access through the middle cranial fossa, and translabyrinthine access are applied) is indicated in the case of large neurinoma at the background of severe hypertension syndrome.

Tumors of the Turkish saddle. Tumors of the Turkish saddle include tumors that differ in histological structure and the time of onset, but common features are the proximity to the hormone-secreting pituitary and optic pathways, changes in the Turkish saddle on craniograms. The most common tumors of the Turkish saddle are pituitary adenomas; craniopharyngiomas (Rathke's pocket tumors); meningioma of the tubercle of the Turkish saddle.

Pituitary adenomas are the most common tumors of chiasmatic-sellar localization. They account for, according to various authors, from 6.7 to 18 % of all brain tumors (K.A. Lebedev, 1990; H. Jho, et al., 1997). Pituitary adenomas develop from cells of the anterior pituitary. Tumors practically do not grow from the posterior lobe. There are nowadays several classifications of pituitary adenomas. The most common classification is the division of pituitary adenomas into *acidophilic adenomas* (accompanied by hypersecretion of somatotropic hormone), *basophilic* (which is accompanied by increased ACTH secretion and manifested by Itsenko-Cushing's disease), chromophobic (without impaired hormone secretion), and *mixed*.

However, according to the most modern classifications (R. Aldman, 1980; E. Horvach, K. Kovacs, 1995), pituitary tumors can be divided into the following:

- Adenoma that secretes growth hormone (*somatotropinoma*) — up to 13—15 %. The size of the somatotropinoma reaches 13 mm or more (macroadenoma) and leads to an increase in the Turkish saddle. Increased STG secretion leads to the development of gigantism (in children and adolescents), or acromegaly (in adults). Somatotropinoma is characterized by the development of splanchnomegaly (often cardiomegaly), 25—35 % — hypertension, 15—19 % — diabetes, 70—80 % of women — dysmenorrhea and amenorrhea, 30—45 % men — sexual disorders, disorders of spermatogenesis, testicular atrophy.
- Adenoma that secretes prolactin (*prolactinoma*) — up to 25—28 %. In women microadenomas (up to 2—3 mm) are mostly found; in men macroadenomas (up to 1 cm and more) are often found. The tumor manifests by the syndrome of galactorrhea, amenorrhea (in women), sometimes hirsutism, acne due to activation of the adrenal glands.
- Adenoma that secretes ACTH (*corticotropinoma*) — up to 8—10 %. Corticotropinomas are usually microadenomas. The main mani-festations of hypercorticism are obesity of the upper torso, neck, face, the presence of pregnancy, acne, hirsutism, hypertension, systemic osteoporosis, menstrual disorders, and sexual disorders.
- Adenoma that secretes thyrotropic hormone (*thyrotropinoma*) — up to 1 %. It is usually manifested by symptoms of thyrotoxicosis with diffuse enlargement of the thyroid gland, less often eu- or hypothyroidism. It is often combined with symptoms of dysmenorrhea, amenorrhea, galactorrhea, sexual disorders, less often — acromegaly.
- Adenoma, which secretes follicle-stimulating and luteinizing hormones (*gonadotropinoma*) — up to 7—9 %. In adults, the symptoms are vague. In children, it is manifested by premature puberty (the appearance of secondary sexual characteristics before 9 years in boys and before 8 years in girls), increased growth and differentiation of bone tissue, and closure of growth plates. It is necessary to conduct a differential diagnosis with tumors of the ovaries, testicles, adrenal glands, and congenital hyperplasia of the adrenal cortex.
- Adenomas that secrete more than one hormone (*mixed forms*) — 3—5 %.
- Endocrinologically inactive adenomas (oncocytoma, adenocarcinoma), etc.

Pituitary adenomas can grow relative to the Turkish saddle: infrasellar (under the Turkish saddle); suprasellar (above the Turkish saddle); parasellar (near the Turkish saddle); retrosellar (behind the Turkish saddle); antesellar (in front of the Turkish saddle); combined options are also possible.

The main clinical symptoms:

- Endocrine disorders are caused by increased rarely decreased secretion of hormones.
- Visual disorders caused by compression of the chiasm (decrease in visual acuity, change of visual fields, more often the development of bitemporal hemianopsia; formation of primary optic nerve atrophy, dysfunction of II, III, IV, VI pairs of cranial nerves).
- Changes in the Turkish saddle (increase in size, its deformation, destruction, etc).
- Formation of the hypertensive syndrome, focal symptoms due to the formation of a mass effect, impaired cerebrospinal fluid circulation with the development of hydrocephalus, etc.

- Hemorrhage in pituitary adenoma (apoplexy) with a clinical picture of acute cerebral circulatory disorders, visual disorders.

Diagnosis

- consultation of an endocrinologist, determination of the content of pituitary hormones in the blood;
- ophthalmologist consultation;
- craniography (aiming at areas of the Turkish saddle);
- MRI of the brain.

Treatment. The main goal of treatment of pituitary adenoma is the correction of endocrine disorders, reduction of tumor size and/or masseffect.

Drug treatment: in the case of prolactinoma, such drugs as bromocriptine (Parlodel) or cabergoline (Dostinex) are used; their advantage is applied once a week. The mechanism of action of such drugs is to increase the release of dopamine from the hypothalamus, which inhibits the secretion of prolactin. Octreotide (somatostatin) is prescribed in the early stages of acidophilic pituitary adenoma.

Surgical treatment: the absolute indications for surgical treatment of pituitary adenoma, despite the severity of hormonal activity, are a progressive decrease in visual acuity, narrowing of the visual fields due to compression of the chiasm, optic and oculomotor nerves, disturbance of spinal liquid outflow followed by the formation of hydrocephalus, increase in intracranial pressure, hemorrhage into the tumor, nasal cerebrospinal fluid. Relative indications are determined individually; they are aimed at restoring the hormonal function of the pituitary gland in the case of ineffectiveness or severe side effects of drug therapy.

The main accesses to the pituitary gland are:

- transcranial subfrontal (indications: pronounced parasellar growth with invasion into the cavernous sinus, pronounced supra-retroantesellar growth of the tumor with growth into the III and lateral ventricles, the presence of a thin membrane between the suprasellar and infrasellar nodes of the tumor);
- transnasal transsphenoidal (it is used in most cases);
- sublabial transsphenoidal (rare).

Transnasal transsphenoidal access to the pituitary adenoma is performed under X-ray control (CT, MRI navigation) using microsurgical, endoscopic instruments. In some centers, endoscopic cryo-, electro-, and laser destruction of pituitary adenomas are performed.

The most common postoperative complications include the decreased secretory activity of the adenohypophysis, nasal cerebrospinal fluid, infectious complications, damage to the carotid artery or cranial nerves, the formation of hydrocephalus, and more.

Radiation therapy is used as an adjunct to the surgical treatment or in the case of severe contraindications to the surgical treatment. The most radiosensitive tumors are somatotropinomas, the total dose of course irradiation is 45—50 Gy.

Craniopharyngioma is a dysembryogenetic tumor that mainly occur in children and adolescents. The first peak of development of this tumor occurs within the period between the 5th and 10th years of life, the second peak — in the 5th decade of life. The appearance of such tumors is associated with incomplete reverse development of the so-called Rathke's pouch — the growth of embryonic pharyngeal epithelium, which is

involved in the formation of the anterior pituitary gland. These tumors can develop in the Turkish saddle and beyond, in the infundibulum and bottom of the III ventricle. Tumors consist of dense tissue, which often contains calcified areas (petrifications), and a cystic cavity. Cysts are mostly multiple, often giant, containing xanthochromic or brown fluid. The following dysembryogenetic tumors also often develop in this area: cholesteatoma, dermoid cyst, teratoma, germinoma, etc.

Clinical picture:

- endocrine disorders: stunted growth (dwarfism), obesity, menstrual irregularities, diabetes mellitus, lethargy, rarely — cachexia;
- visual disorders: decreased visual acuity, primary atrophy of the optic nerves;
- development of the hypertensive syndrome: mainly in the case of growth of the tumor into in the third ventricle and compression of interventricular foramina;
- the presence of petrifications over the Turkish saddle on the craniogram or CT.

Diagnosis of craniopharyngioma is similar to the diagnosis of pituitary adenoma.

Surgical treatment of craniopharyngioma:

- temporary interventions: puncture and emptying of cysts, ventricu-lar drainage;
- tumors of small size, which are located mainly in the cavity of the Turkish saddle, are removed using transsphenoidal or subfrontal accesses;
- in the presence of tumors of gigantic size and those that grow through the bottom of the third ventricle, are removed via combined accesses.

In the chiasmatic-sellar area (the area of the intersection and the Turkish saddle) there also could happen meningioma of the tubercle of the Turkish saddle, glioma of the optic nerves. The main and almost the only manifestation of meningioma of the tubercle of the Turkish saddle is a progressive decrease in visual acuity.

Diagnosis: CT, better MRI of the brain.

Surgicaltreatment. The tumor tissue often includes carotid, anterior cerebral arteries, chiasm, optic nerves, pituitary gland. The tumor should be removed gradually, using only microsurgical instruments and an ope-rating microscope. Optic glioma is usually seen in childhood and is characterized by a progressive decrease in visual acuity. The main method of treatment is radiation therapy. Surgical treatment is rarely used.

Metastatic tumors. Up to 20—40 % of malignant tumors metastasize to the brain. In the United States alone, they are diagnosed at 170.000 cases annually, which is almost 10 times the rate of primary brain tumors. Metastases to the brain account for 10—23.7 % of all brain tumors (I. Wallon, 1977). Among the primary foci, the predominant tumors are lung cancer (up to 50 %), breast cancer (15 %), melanoma (10.5 %), hypernephroma (4.4 %), etc. (J.B. Posner, 1978).

In individuals before the age of 20, the primary foci are osteosarcoma, rhabdomyosarcoma, and embryonic cancer. Among patients diagnosed with brain metastases, 47 % have one metastasis (mostly in the case of breast cancer, hypernephroma), and 53 % of patients have two or more metastases, which is characteristic for melanoma, lung cancer. The main way of metastasis to the brain is hematogenous.

Most often metastases are formed on the border of grey and white matter in the area of blood supply to the middle cerebral artery. 80 % of brain metastases are formed in the hemispheres of the brain, 15 % — in the cerebellum, 5 % — in the brain stem (J. Cairncross, 1983; J. Dellattre, 1988).

According to CT and MRI data, metastases are mostly spheric, with fairly clear outlines (melanoma is characterized by fuzzy edges of metastasis, there is a small node with large vasogenic cerebral edema) and moderate perifocal edema, which allows their removal or the use of radio-surgical techniques (T. Hwang, 1996).

Hypertension syndrome (up to 40 %) is one of the most common clinical symptoms; the predominant complaints include headache, weakness, and memory impairment. Frequent focal symptoms are disorders of sensitivity, ataxia, cognitive and mental disorders (up to 2 %). Congestion of the optic disc is observed in 25 % of patients. Hemorrhage in tumors develops in 5 % of patients. Convulsive seizures are observed in 15—20 % of patients with metastatic brain tumors, and in 30—40 % of them, this symptom is the only manifestation of the process (J.B. Posner, 1977; B.S. Borgelt, 1980). The metastasis of brain tumors to other organs and systems is not typical. Medulloblastoma, anaplastic ependymoma, glioblastoma, pineoblastoma, and some other tumors can metastasize to the cerebrospinal fluid and sometimes to the spinal cord.

Diagnosis of metastatic tumors

- General clinical research.
- Contrast-enhanced CT.
- Contrast-enhanced MRI.
- Stereotactic (rarely open) tumor biopsy.

In the case of absence of data regarding the primary source:

- Chest radiography (to exclude lung cancer);
- CT scan of the chest, abdomen, pelvis;
- scintigraphy (radiography of the spine, pelvis, and bones of the extremities);
- mammography in women.

Differential diagnosis of metastatic tumors is performed with the primary brain tumor, abscess, radiation necrosis, inflammatory reaction, etc.

Treatment strategy. When determining treatment strategy for metastatic brain tumors, the following considerations should be taken into account: the general condition of the patient, the presence of systemic cancer signs, the size of the tumor, the number of metastatic foci, the severity of perifocal edema, etc.

In the case of the presence of one metastatic focus:

- lateral location of metastasis: removal and the course of radiation therapy;
- medial location: application of one of the radio-surgical techniques and the course of radiation therapy.

In the case of the presence of several metastatic foci:

- if one of the foci is large and gives clear focal symptoms and is located laterally, it is possible to remove it, followed by the course of radiation therapy;
- in the presence of three or fewer foci: application of one of the radio-surgical techniques followed by the course of radiation therapy to the entire head (in the case of lateral location of foci, their surgical removal is possible);

- in the presence of three or more foci: a course of radiation therapy.

It is possible to finally determine treatment strategy in patients with me-tastatic brain tumors after stereotactic (rarely open) tumor biopsy. Taking into account the violation of the regeneration process in this category of patients, it should not be made a horseshoe-shaped incision, but rather linear or other options providing careful hemostasis during the removal of metastases. Removal of metastases is preferably performed using microsurgical instruments, an operating microscope, and, if possible, using navigational techniques or intraoperative neurosonography. If the metastases are located in the precentral gyrus, access should be made through the pre-or central sulcus, and if manipulations are provided in the speech areas, surgery is performed according to the «wake up» method (in full consciousness).

Independent study on the topic

Write the definition of the following terms:

1. Hypertension syndrome.
2. Dislocation syndromes.
3. Radical and palliative surgical treatment of brain tumors.
4. Combined treatment of brain tumors.
5. Radiosurgical treatment.
6. Quality of the life of the patients.

Please give a written answer to the following questions:

1. Classification of brain tumors by location.
2. Classification of brain tumors by histological structure.
3. Name the main clinical symptoms of brain tumors.
4. What pathophysiological mechanisms underlie the occurrence of hypertension syndrome?
5. What underlies the occurrence of primary and secondary focal symptoms in the case of brain tumors?
6. What additional research methods should be used in the case of a brain tumor is suspected?
7. What changes in the eye fundus can be observed in the presence of brain tumors?
8. Name the craniographic signs of brain tumors.
9. What modern treatment methods do you know?
10. What is the basis of palliative surgery for brain tumors?
11. Name the principles of combined treatment of brain tumors.

Tasks for self-control

1. A 63-year-old patient was hospitalized in the neurological department with complaints of severe headache, speech disorders, weakness in the right extremities. It is known that during the last two months the patient was suffering from a moderate headache in the pre-morning period with nausea and vomiting. A year ago, the patient was operated on for a tumor of the right lung. Objectively: consciousness is clear, right-sided hemiparesis, symptoms of damage of VII and XII pairs of cranial nerves on the right of the central type, motor

aphasia. Stagnant optic discs are expressed. What process can be suspected in the patient? What diagnostic algorithm can be used? What treatment should be prescribed to the patient?

2. A 45-year-old female patient applied to a neurosurgeon about the lack of hearing in her right ear, severe headache in the morning, at the peak of which there was nausea, vomiting, staggering while walking. From the anamnesis it is known that 5 years ago there was a noise in the right ear, hearing in the right ear gradually decreased. For the last two years, there has been no hearing in the right ear, the symptoms described above appeared soon. Objectively: the patient is lethargic, adynamic, there is spontaneous horizontal nystagmus, the right nasolabial fold is smoothed, instability in the Romberg position is noticed. Optic discs are stagnant. Which process can be suspected in the patient? What stages of symptoms are typical for this pathology?

3. The mother of an 8-year-old girl went to the doctor with complaints of severe headache in the child, general weakness, decreased visual acuity, premature sexual development, weight gain. Consciousness is clear, there is a decrease in the direct and concomitant reaction of the pupils to light with a preserved response to convergence, paresis of the upward gaze, a slight increase in tendon and periosteal reflexes D = S. Stagnant optic discs are expressed. What pathological process can be suspected in a child? With what pathology should a differential diagnosis be made?

4. A 32-year-old woman developed dysmenorrhea; a few months after she gained weight; there was sharply decreased visual acuity in one eye; a few weeks ago there was a moderate headache, general weakness. What pathology can be suspected in a patient? What additional research methods can be informative?

5. A 7-year-old girl has been complaining of severe headache, nausea, vomiting, staggering while walking, dizziness, double vision when looking up. Consciousness is clear, there is a violation of the function of the III pair of cranial nerves, vertical nystagmus, instability in Romberg position, finger-nose test performed with intention. Meningeal signs are not present. Where is the pathological focus?

6. A 50-year-old patient had two generalized epileptic seizures at night during the month. It is known that for the last two years the patient has been suffering from moderate headache, which was relieved by analgesics. In the neurological status, the left pyramidal insufficiency in the form of positive upper and lower Barre symptom on the left increased tendon and periosteal reflexes in the left extremities. On funduscopy, the initial signs of stagnant optic discs were observed. What pathological process can be suspected?

Tumors of the spinal cord

Tumors of the spinal cord include neoplasms that develop in the substance of the spinal cord and from its roots and membranes. Tumors that develop from the elements of the spine do not belong to the tumors of the spinal cord.

Classification of spinal cord tumors is based on three main features:

1) histological structure of the tumor;
2) localization of the tumor relative to the spinal cord, membranes, and spine;
3) the level of the tumor along the spinal cord.

According to the histological structure, tumors are distinguished depending on their origin: from the spinal cord — astrocytoma, ependymoma; from the roots of the spinal cord — neurinomas; from the membranes — meningioma; from adipose tissue — lipoma; from connective tissue elements — sarcoma.

Depending on the location relative to the spinal cord, tumors are divided into two groups:

1. Extramedullary tumors that grow outside the spinal cord, but cause compression of the spinal cord and its roots: a) extradural (epidural); b) intradural; c) intra-extradural; d) intra-extravertebral — of hourglass type.

2. Intramedullary tumors, localized inside the spinal cord.

Taking into account the level of tumor localization along the spine axis, the following types are distinguished: a) tumors of the upper cervical localization (C_1—C_4); b) tumors of the lower cervical localization (C_5—Th_1); c) tumors of the thoracic localization (Th_2—Th_{10}); d) tumors of the lumbar localization (Th_1—Th_{10}); e) tumors of the ponytail — of the roots of the nerves in the lower back (L_2—S_5).

Clinical picture. The clinical picture of spinal cord tumors is characterized by the presence of three typical stages: 1) radicular; 2) stages of half-spinal cord injury — Brown—Sequard syndrome (Brown—Sequard paralysis); 3) paralytic.

1. *The radicular stage* is characterized by the appearance of paresthesias or pain at the initial stage of the disease. This pain is localized according to the level of the tumor, which causes compression of the spinal root located nearby. Initially, the pain occurs, on the one hand, exacerbates with physical exertion, sneezing, or coughing. Sensitivity disorders manifest in hyperesthesia, which becomes permanent; as the root compression increases, hypoesthesia appears; complete anesthesia then develops. Quite often radicular pain occurs in the case of localization of the tumor in the cervical area of the spinal cord or the area of the ponytail.

2. *Stage of Brown—Sequard syndrome.* In extremities of the same side, where the tumor is localized, motor disorders develop; sensitive disorders of the conductive type develop in the extremities on the side opposite to the tumor. The presence of Brown—Sequard syndrome together with root pain is a feature of extramedullary tumors (meningioma and neurinoma).

3. *The paralytic stage* is the final one in the clinical development of spinal cord tumors of different localization and histological structure. It appears after Brown—Sequard syndrome in the form of symmetrical spastic paraparesis in the lower extremities with increased tendon reflexes, the presence of pathological reflexes, decreased sensitivity at the area below the location of the tumor, pelvic dysfunction.

In the clinical course of spinal cord tumors, there are two types of paralysis: ascending and descending.

Ascending paralysis manifests in the initial development of motor and sensory impairments distally from the tumor (back, perineum) with the gradual development of pathological signs from the bottom up (foot, shin, thigh, etc.). This type of spastic paralysis and impaired sensitivity is a feature of extramedullary tumors of the spinal cord. At the same time, the tumor of the spinal cord firstly causes compression of superficial long nerves, and then — of the deep short nerves.

Descending paralysis manifests in the development of conductive disorders of the spinal cord, which spread from top to the bottom. This type of disorder is typical for intramedullary tumors, due to the compression of short nerves of the spinal cord leading to the central segments (upper extremities). In intramedullary tumors, in

contrast to extramedullary tumors, sensitivity disorders are detected by the type of anesthesia in the perineum, external genitalia, which has diagnostic value.

Tumors of upper cervical localization (at the level of segments C_1—C_4). The tumors are characterized by the early appearance of radicular pain in the occipital region, limited mobility of the cervical spine. Motor and sensory disturbances arise from C_1 downwards, central spastic tetraparesis, and hypoesthesia of the whole body gradually develop. The lesion of the C_4 segment results in paralysis of the diaphragm, respiratory and pelvic organs dysfunction (the central type of paralysis). Sometimes due to compression of the medulla oblongata there are bulbar disorders.

Tumors of the cervical enlargement (at the level of segments from C_5 to Th_1). Upper extremity lesions appear as peripheral paralysis; in lower extremities central spastic paraparesis first develops, leading to lower extremity paraplegia. There is a loss of all types of the sensitivity of the conductive type below the level of the lesion — from C_5 downwards (hypoesthesia or anesthesia). Loss of tendon reflexes from the biceps (segments C_6—C_7) then develops. Horner's syndrome (segments C_8—Th_2), narrowing of the pupils and palpebral fissure, enophthalmos, pelvic dysfunction of the central type (urinary retention, periodic urinary incontinence) are observed.

Tumors of the thoracic region (at the level of segments Th_{11}—Th_{12}). Initially, there is radicular pain, which often has banding character and is localized along the intercostal nerves, in the area of the abdomen, lower back. The upper extremities are not affected. In the lower extremities, motor disorders of the central type — paraparesis, and later paraplegia — are seen. Disturbance of all types of the sensitivity of the conductive type in the lower half of the body (below the level of the lesion — hypoesthesia or anesthesia) develops. The function of pelvic organs is impaired by the central type in the lower half of the body. Abdominal reflexes — upper (Th_7—Th_8 segments), middle (Th_8—Th_{10} segments) and lower (Th_{11}—Th_{12} segments) are decreased or absent.

Tumors of lumbosacral enlargement (at the level of segments L_1—S_2). The tumors manifest in the slow development of flaccid paralysis in the lower extremities. In the case of tumor localization in the upper half of the enlargement, the reflexes of the testicular suspension muscle (L_1—L_2) and knee reflex (L_2—L_4) are absent; Achilles reflexes are increased (L_5—S_1), pathological reflexes are present. If the tumor is located in the lower lumbar region, the tendon reflexes are preserved, the Achilles and plantar reflexes are not present. In the distal parts of the lower extremities paralysis of the foot, up to the knee and above, as well as anesthesia in the foot develop. The function of the pelvic organs is impaired by the central type.

Tumors of the medullarcone (at the level of segments S_3—S_5). Isolated flaccid paralysis (peripheral type) of the pelvic organs — incontinence, feces, sexual weakness. Sensitivity disorders in the area of both buttocks, perineum, and the area of the vagina and genitals. In the case of isolated lesions of the cerebral cone, the motor function of the lower extremities is not impaired, and in the case of stable lesions of the cone and the lower lumbar region, paralysis and anesthesia are manifested in the distal extremities.

Cauda equina tumors (at the level of L_3 and below). These tumors are characterized by prolonged persistent and intensifying pain in the lumbar region, sacrum, anal hole, and lower extremities. Gradually, motor and sensory disturbances increase of the root type, first in one leg and then in both legs. Then disorders of the pelvic

organs of the peripheral type (flaccid paralysis) — complete incontinence of urine and feces develops. The symptoms develop not symmetrically in contrast to the tumors of cone medullaris as well as upper lesions.

Diagnosis. The modern method of neuroimaging — MRI is of particular importance in the diagnosis of spinal cord tumors. MRI data can determine the location, size, and spread of the tumor process. The possibility of research in three projections makes it possible to visualize the upper and lower poles of the tumor, and in the axial plane to determine the spread of the tumor in the anterior-posterior direction. Characteristic features of extramedullary spinal cord tumors on MRI are symptoms of spinal cord compression with the expansion of the subarachnoid space above or below the tumor. In the case of intramedullary tumors clear expansion of a spinal cord is defined on MRI.

Surgical treatment. Diagnosis of the tumor is the direct indication for surgery. The use of new methods of neuroanesthesiology, surgical optics, microsurgical and endoscopic techniques makes it possible to remove spinal cord tumors low-traumatically and radically. Contraindications may include the decompensated condition of the patient with severe disorders of vital functions — respiratory and cardiovascular disorders. According to the location of the spinal cord tumor, laminectomy is performed — the vertebral arches are removed (opening of the spinal canal).

The technique of laminectomy and tumor isolation. Under general endotracheal anesthesia in the position of the patient on the side by the location of the tumor, a linear incision of the skin along the midline over the spinous processes is made. The length of the incision should be one vertebra above and below the removal of the planned number of spinous processes and vertebral arches. The fascia is dissected along the midline, and then the spinous processes are isolated (skeletonized) from the adjacent soft tissues. Skeletonization of spinous processes can be performed using a monopolar electric knife. During the skeletonization of the spinous processes and arches, they must be carefully inspected, as they are often sharply thinned or destroyed by the tumor. Skeletonized processes are cut at the base with the help of Liston bone forceps. The cut width of the arches is 2—3 cm, while it is necessary to avoid pressure with forceps on the dura mater and spinal cord to prevent their trauma.

Hemostasis is performed by bipolar coagulation at all stages of surgery and especially in the epidural space, wherein the presence of spinal cord tumors epidural veins are sharply dilated, their walls are fixed to the epidural tissue, the veins have no valves and collapse poorly. Damage to the epidural veins can cause profuse bleeding, which can be stopped by pieces of gauze or a piece of muscle.

The epidural tissue is dissected along the midline and bluntly removed to the sides. In the presence of an extradural tumor, it is removed by isolation from the surrounding tissues, starting with the tumor pole. The tumor is isolated with microsurgical instruments. If the tumor is large (hourglass-type neurinoma), it is removed by opening the capsule, and then via encapsulation using an ultrasound disintegrator. Microsurgical and endoscopic methods, as well as ultrasound disintegrator, are used to remove spinal cord tumors intramedullary, which allows removing the tumor with minimal spinal cord injury. The wound is sutured tightly after surgery. The results of surgical treatment of spinal cord tumors depend on the radicality of the operation and the histological structure of the tumor.

The results of the operation are most favorable after removal of neuroma (60 % of patients recover in the immediate postoperative period and 30 % — a few months after surgery). After meningioma removal, recovery occurs in 40 % of patients. Good results were observed after the removal of intramedullary cystic ependymoma. Unsatisfactory results were obtained after removal of diffuse glioma, malignant intramedullary tumors, sarcoma. After surgical subtotal removal of ependymoma, lymphoma, angioma, and other neoplasms sensitive to radiation, radiation therapy is indicated.

Independent work on the topic

1. Describe the process of Wallerian degeneration?
2. Name the indications and contraindications to the revision of peripheral nerve trunks.
3. Describe the concept of «neurolysis»?
4. List the types of closed injures of the nerve trunks.
5. Describe the anatomical structure of nerves.
6. Name the types of injuries of the brachial plexus and characterize them.

Tasks for self-control

1. A 20-year-old male patient complained of the absence of movement in the left shoulder and elbow joints with the preservation of movements in the fingers and hands decreased sensitivity on the outer surface of the shoulder, forearm, hand. A month ago he had a fracture of the transverse processes of vertebra C_5—C_6. What is the preliminary diagnosis?

a) post-traumatic upper-left shoulder plexitis;
b) post-traumatic neuritis of the left median nerve;
c) post-traumatic neuritis of the left radial nerve;
d) post-traumatic neuritis of the left ulnar nerve;
e) post-traumatic neuritis of the left median and radial nerves.

2. The patient complained of limited bend in the arm, the inability to bend the end phalanges of the IV and V fingers, the contraposition of the V and I fingers. During the examination, the hand is similar to a bone: hypoesthesia in the area of IV and V fingers and hands on the palmar and posterior surfaces, a scar in the upper third of the medial surface of the left forearm. What is the previous diagnosis?

a) post-traumatic lower-left plexitis;
b) post-traumatic neuritis of the left median nerve;
c) post-traumatic neuritis of the left radial nerve;
d) post-traumatic neuritis of the left ulnar nerve;
e) post-traumatic neuritis of the left median and radial nerves.

3. The patient underwent a gastrectomy. During the operation, the patient's left upper limb was extended from the body and fixed on the operating table for anesthesia. In the postoperative period, the patient developed dysfunction of the upper limb in the form of a dropping hand. Damage to which anatomical structure led to this symptom?

a) radial nerve;
b) axillary nerve;
c) ulnar nerve;
d) the median nerve;
e) musculoskeletal nerve.

4. The newborn after abnormal birth from the first day of life had no active movements in the right upper extremity. The general condition is disturbed. Moreau's reflex could not be caused. The tendon-periosteal reflexes on the affected arm are sharply reduced. What is the most likely diagnosis?

a) traumatic plexitis, total type;
b) intracranial birth trauma;
c) traumatic fracture of the right humerus;
d) osteomyelitis of the right humerus;
e) traumatic plexitis, distal type.

5. A patient with an injury to the lower third of the palmar surface of the forearm applied to the trauma center. Objectively: the flexion of the IV and V fingers is impaired, the sensitivity of the inner dorsal and palmar surface of the hand and the IV finger is reduced. Which nerve is injured?

a) ulnar;
b) middle;
c) radial;
d) musculoskeletal;
e) axillary.

Pathology of intervertebral discs

Intervertebral discs in the form of fibrocartilage are located between the vertebral bodies and consist of a pulp nucleus, a fibrous ring, and cartilaginous hyaline plates. They act as buffers that significantly reduce the axial load on the spine. The pulp nucleus is well hydrated, has constant pressure within the fibrous ring that surrounds the pulp, is elastic, and in the case of pressure can expand. The pulp nucleus acts as an axis around which the movements of the spine are made. Depending on the direction of pressure, the axis can change its position and the movements of the spine are carried out in different directions. When compressed, the pulp nucleus is not compressed, but only changes its shape and the shape of the fibrous ring.

Under the load, the pulp nucleus flattens and stretches the fibers of the fibrous ring, in the posterior parts a protrusion is formed. After the pressure ceases, the fibrous ring acquires its initial shape. However, degeneration of the intervertebral discs gradually occurs, the pulp nucleus is dehydrated, becomes fibrous, and its turgor decreases. The fibrous ring becomes brittle; cracks are formed in it. The pulp nucleus penetrates the cracks, enters the vertebral body, and gradually turns into a small cartilaginous nodule (Schmorl nodule). In the case of the development of degenerative processes in the discs, there are reactive changes in the structure of the vertebral bodies, which leads to the development of **osteochondrosis**. As the disease progresses, all elements of the disc are destroyed with the subsequent development of fibrous ankylosis in the vertebral bodies.

In the case of degenerative changes only in the area of the fibrous ring, there is a protrusion and separation of its fibers from the vertebral bodies with the subsequent development of calcification of the longitudinal ligaments and the formation of osteophytes, which leads to the deve-lopment of **spondylosis**. Marginal bone growths (osteophytes) develop only in the presence of degenerative changes in the intervertebral discs. At the same time, there is also an expansion of edges of articular surfaces of a vertebra in places of the greatest load that manifest in the development of intervertebral arthrosis — **spondyloarthrosis**.

Due to the development of degenerative changes in the area of the disc, its displacement can occur without rupture of the fibrous ring, which is known as «**protrusion» (protrusion of the disc)**. In the case of the rupture of the fibrous ring, the degeneratively altered pulp nucleus extends beyond it and prolapse of the intervertebral disc occurs or a **herniated disc** is formed, which leads to compression of nerve structures and instability in the spinal motor segment.

Depending on the direction of disc prolapse or hernia, there are three types of this pathology:

1. Lateral disc herniation — foraminal, located in the intervertebral foramen, causing significant compression of the spinal cord roots.

2. Paramedian hernias — penetrate the spinal canal and cause compression of the spinal cord and its roots.

3. Middle (central) hernias — penetrate degeneratively altered hyaline plate into the spongy mass of the vertebral body and in the case of their small size have an asymptomatic course (Schmorl nodule). Large prolapses (hernias) of the disc lead to the compression of ventral parts of the spinal cord and its roots. Marginal bone growths (osteophytes), protrusions, and disc herniations located in the lumen of the spinal canal leading to the development of compression of the roots and spinal cord.

Classification of osteochondrosis of the spine

1. Intervertebral osteochondrosis:

a) internal degenerative-dystrophic changes of intervertebral discs with preservation or loss of stability of the motor vertebral segment;

b) displacement of intervertebral discs with the occurrence of their protrusion or prolapse into the lumen of the spinal canal;

c) anterior and lateral protrusions or disc prolapses;

d) paramedian and central disc prolapses;

e) scarring changes of discs and adjacent tissues with the presence of ankylosis of the motor spinal segment.

2. Reactive changes of the spine:

a) deforming spondylosis — marginal bone growths of vertebral bodies;

b) spondyloarthrosis — changes in the joints of the spine, characterized by narrowing of the joint space, the formation of marginal osteophytes.

All these clinical forms are different manifestations of osteochondrosis in the form of degenerative processes in the intervertebral discs and the reaction of the adjacent vertebrae. The static load on different parts of the spine is different, so neurological complications are most pronounced in the cervical and lumbar spine.

In the case of osteochondrosis in *thecervical spine*, there are three types of syndromes.

1. Reflex syndromes: a) cervicalgia; b) cervicocranialgia; c) cervicobrachialgia with muscular-tonic, vegetative-vascular manifestations.

2. Root syndromes (indicating which roots are affected).

3. Vascular radicular-spinal syndromes: a) radiculoischemia;

b) radiculomyeloischemia — transient, acute (indicating the affected basin of the spinal arteries), chronic, indicating the predominant localization of ischemic lesions of the spinal cord.

In the case of osteochondrosis in *the thoracic spine*, there are two types of syndromes.

1. Reflex syndromes: thoracalgia with muscular-tonic, vegetative-visceral, or neurodystrophic manifestations.

2. Root syndromes (indicating which roots are affected).

In the case of osteochondrosis of *the lumbosacral spine* three types of syndromes could be seen:

1. Reflex syndromes: a) lumbago (acute lower back pain); b) lower back pain; c) lumboischialgia with muscular-tonic, vegetative-vascular, or neurodystrophic manifestations.

2. Root syndromes (indicating which roots are affected, including the cauda equina).

3. Vascular radicular-spinal syndromes: a) radiculoischemia; b) radiculomyeloischemia; c) myeloischemia — transient, acute (stroke), chronic.

Clinical syndromes of lumbosacral osteochondrosis. The clinical picture of lumbar osteochondrosis consists of vertebral symptoms (changes in the statics and dynamics of the lumbar spine) and symptoms of dysfunction of neurological structures (motor, sensory, autonomic, and trophic nerves) and adjacent formations (arteries, veins, etc.). The main complaint is pain.

Lumbago and lumbalgia — pain in the lumbar spine, which occurs acutely. The disease develops suddenly, after a sudden movement or in the case of lifting heavy objects (especially if it is combined with hypothermia). There is stiffness, which is accompanied by pain of expansive, burning, compressive nature. Any movement, even conversation, increases the pain. At first, the pain radiates widely, spreading to the chest, buttocks, and even the abdomen. Patients take a forced position. After a few hours or days, the pain decreases. New recurrences of the disease also occur under the influence of mentioned unfavorable factors.

Lumboischalgia syndrome is observed in more than half of people engaged in heavy physical labor. The duration of the disease (with periods of exacerbation and remission) ranges from several months to many years.

Muscle-tonic (neuromuscular) forms of lumboischalgia are diagnosed in 62 % of patients. The disease is caused by sharp lifting of heavy objects, prolonged physical activity, tonic muscle tension, combined loads.

The pain syndrome is characterized by lumbar pain that extends to one or both lower extremities.

X-ray diagnosis of degenerative-dystrophic lesions of the spine is based on the assessment of both early and late signs, as well as changes in spinal function. For this purpose, functional radiological examinations (flexion, extension) are performed together with the radiographs of the spine, necessarily in two projections. It is recommended to combine the functional radiological examinations in two opposite directions, as this is accompanied by the effect of strengthening or weakening of the hidden displacements of the vertebrae (disc instability). A large group of radiological symptoms of osteochondrosis is associated with an impaired static function of the spine. On radiographs, this manifests in a change in the axis of the spine — straightening of the cervical and lumbar lordosis or increased thoracic kyphosis. When the C_5—C_7 discs are affected, complete straightening of the lordosis is usually observed, and changes in the C_3—C_5 discs lead to only a partial straightening of the lordosis. The formation of kyphosis is possible. The distance between the vertebral processes at the level of kyphosis increases. Scoliosis is often observed in the lumbar spine. On radiographs with functional examinations, it is possible to detect a shift of one, two, or more vertebrae in one direction, which is referred to as a stair shift. These changes indicate a loss of fixation of the disc — the initial manifestations of osteochondrosis.

Instability — the appearance of displacements of the vertebrae forward, back, and forth, which is not characteristic of the unchanged motor segment. In the case of instability, there is an angular deformation at the level of the discs due to the displacement of the vertebrae. Vertebral displacement up to 2 mm is within the normal ranges. Children are characterized by the increased mobility of the C_2—C_3 segment, but the instability of the disc within 2 mm should be assessed taking into account clinical symptoms. Degenerative-atrophic changes reflect the disintegration of the disc, the destruction of the marginal plates of the vertebral bodies, and the protrusion of the disc into the body in the form of small nodules. Degenerative-reactive changes in the vertebral bodies manifest in the form of marginal bone growths (osteophytes) and subchondral sclerosis. Marginal bone growths are a manifestation of the compensatory adaptive response in the vertebrae to the load to the altered disc.

Another radiological feature that reflects the functional overload of the spine is reactive sclerosis of the vertebral bodies (subchondral sclerosis), which sometimes extends to a third of its height. Radiological signs associated with changes in vertebral bodies include chamfer angles of vertebral bodies, which may be due to a degenerative process in the marginal surface of the vertebral body.

Osteochondrosis of the intervertebral discs and spondyloarthritis are complicated by vertebral displacement (spondylolisthesis), which is found in the neutral position of the patient, but maybe exacerbated during flexion or extension.

Cartilaginous nodes of vertebral bodies (Schmorl nodule) can be one of the manifestations of dystrophic lesions in the spine. Dystrophic changes of the hyaline plate lead to its rupture and are accompanied by protrusion of the disc tissue into the spongy substance of the vertebral body. Along with dystrophic genesis, the cause of cartilaginous nodes of vertebral bodies can be trauma, as well as several features of development that create a functional defect of the disc.

X-ray diagnosis of lumbar disc herniation is based on indirect symptoms: straightening of the lordosis; scoliosis, more often expressed in the affected side of the spine, the symptom of the strut — wedge-shaped disc, with an apex directed forward, osteoporosis of the posteroinferior corner of the vertebra.

In the presence of the clinical picture of compression of a root, the early signs of an osteochondrosis are estimated on the X-ray — both moderate decrease in height of a disk and its instability.

A direct radiological sign of a disc herniation is the detection on the lateral radiograph of a shadow protruding into the spinal canal, the morphological substrate of which is the compaction, calcification, or ossification of the posterior cartilaginous node. In difficult diagnosis cases, in the presence of clinical data indicating a herniated disc, it is advisable to use myelography, epidurography, venospondylography, CT, MRI.

Pneumomyelography (PMG) in the normal state of the intervertebral disc and subarachnoid space shows the particular characteristic picture. The gas column is usually well expressed at the PMG, and a light wavy contour 1—2 mm high is formed at the level of the intercostal discs. In the presence of posterior hernias of the intervertebral disc, an arcuate protrusion at its level is detected. In the anterior hernia on the background of tissue edema and narrowing of the dural sac, detection of intervertebral disc herniation is difficult. However, PMG detects intervertebral disc herniation in 40 % of cases. The main disadvantage of PMG is that it does not detect changes in the intervertebral disc itself.

Myelography, which is performed with a water-soluble contrast agent, is a more accurate method, it allows detecting the presence of a herniated disc.

At present, ***computed tomography*** is of great importance in the diagnosis of intervertebral disc herniation. Due to this method of research, disc herniations are well detected, their structure is specified, secondary reactive-inflammatory changes in the subarachnoid space are found, and the narrowing of the vertebral canal due to bone growth is determined.

Vascular spiral computed tomography can detect the presence of spinal artery stenosis caused by cervical spondylosis.

Magnetic resonance imaging can determine the severity of osteochondrosis of the spine, the condition of the pulp nucleus, the presence of anatomical integrity or rupture of the fibrous ring, protrusion or prolapse of the disc, the condition of the posterior longitudinal ligament, the degree of compression of the intervertebral foramen.

Treatment of osteochondrosis

In the early stages of osteochondrosis of the spine, when there are initial signs of disc degeneration (stage I osteochondrosis) and the predominant symptoms are spinal cord roots irritation in the form of lumbago and lumbalgia and the presence of disc protrusion up to 6 mm, conservative medical treatment is indicated. The occurrence of acute pain in the presence of protrusion up to 6 mm (stage II osteochondrosis) is an indication for the use of various types of blockades with the application of analgesics and steroids, which significantly reduce inflammation and promote regression of clinical manifestations of the disease.

The lack of stable positive results from conservative therapy for 5—6 months, despite the use of analgesic blockades or the application of hormones, it is indicated to carry out an additional examination using neuroimaging methods (CT, CT-spiral tomography, NMR, electromyography, etc.) with the further determination of indications for surgical treatment.

The main types of surgery in the treatment of osteochondrosis

I. Extraforaminal.

1) Percutaneous laser nucleotomy.

2) Endoscopic nucleotomy and discectomy.

II. Intraforaminal.

1) Interlaminar access to intervertebral disc herniation.

2) Hemilaminectomy or hemilaminectomy with facetectomy, microdiscectomy, video-endoscopy, neurotransplantation in the presence of root and spinal cord pathology in the thoracic and lumbar spine.

Interlaminar access to intervertebral disc herniation with video-endoscopic technique.

A linear incision of the skin along the spinous processes is made, usually along with the level of two vertebrae followed by the unilateral exposure of the lateral surfaces of the spinous processes and the arches of the two vertebrae. The yellow ligament is dissected and the adjacent vertebral arches are partially removed. The intervertebral disc herniation is exposed, and its capsule is cut crosswise. This access is mostly used in the case of the removal of lateral hernias of the intervertebral disc with video-endoscopic assistance.

Hemilaminectomy or hemilaminectomy with facetectomy, foraminotomy, microdiscectomy, video-endoscopic assistance, neurotransplantation in the presence of root atrophy, and severe myelopathy.

Interarchal access allows the removal of only lateral hernias. In the case of paramedian and medial disc herniations, hemilaminectomy or hemilaminectomy with facetectomy should be used.

To perform hemilaminectomy a linear incision of the skin and subcutaneous tissue along the apexes of the spinous processes at the level of two vertebrae is made. One-sided exposure of the lateral surfaces of the spinous processes and arches is carried out. In the case of the location of osteophytes in the intervertebral foramen and compression of the root, the foramen is expanded craniotomy to decompress the root (foraminotomy). Foraminotomy is one of the stages of hemilaminectomy.

After the skeletonization of the spinous process, the arch is cut together with the adjacent sections of the yellow ligament. The dura mater is removed medially with the help of a microsurgical instrument. With the use of microsurgery and assistive video-equipment, the root is isolated, the hernia capsule is cut crosswise and the disc herniation is removed. Access to disc herniation can be extradural and transdural. Transdural access is used for herniated discs of large size to reduce trauma to the roots and the spinal cord. At the same time, hemilaminectomy with a partial facetectomy is carried out. Disc herniation is removed using microsurgical techniques assisted by video-endoscopy. Minimal vertebral resections through interlaminar access, hemilaminectomy, or in combination with facetectomy generally do not require orthopedic correction. Wide laminectomy in the case of disc herniation is not advisable. After the operation of a herniated disc removing in the presence of segmental instability, intracorporeal fixation systems are introduced, which provide reliable stabilization of the affected motor segment.

For intracorporeal fixation, cages of various designs are most often used, which have the shape of a hollow cylinder with an external screw thread, which facilitates their introduction into the intervertebral space.

About 10—20 % of patients with osteochondrosis of the spine with the presence of segmental instability require stabilization operations.

Independent work on the topic

Write the definition of the proposed terms:

1. Tumors of the spinal cord.
2. Extramedullary tumors.
3. Intramedullary tumors.
4. Ascending type of sensitivity disorder.
5. Descending type of sensitivity disorder.
6. Laminectomy.
7. Protrusion of the intervertebral disc.
8. Prolapse of the intervertebral disc.

Give a written answer to the following questions:

1. What are the first clinical manifestations of spinal cord tumors?
2. What are the first clinical manifestations of osteochondrosis of the spine?
3. What are the types of intervertebral disc herniation?
4. Classification of spinal cord tumors.
5. What are the peculiarities of providing first aid to patients with radicular syndrome?
6. What are the clinical signs of spinal cord injury?
7. What are the clinical symptoms of spinal cord tumors depending on the level of damage?
8. What is the most common location of intervertebral disc herniation and why?
9. What is the difference between the clinical picture of extra- and intramedullary tumors?
10. What additional research methods are used in the case of suspicion of spinal cord tumor and osteochondrosis of the spine?
11. What are the changes in the cerebrospinal fluid test and cerebrospinal fluid dynamic tests in patients with spinal cord tumors and intervertebral disc herniation?
12. What should be the treatment strategy for sequestered intervertebral disc herniation?
13. Name the clinical stages of extramedullary tumor development.
14. Principles of conservative treatment of discogenic radiculitis.
15. Modern principles of surgical treatment of spinal cord tumors.
16. Modern principles of surgical treatment of intervertebral disc herniation.
17. Laminectomy technique.
18. Name the possible complications of spinal cord tumors.
19. Name possible complications at hernias of intervertebral discs.
20. What types of treatment are used in the recovery period?
21. What rehabilitation do patients with spinal cord tumors and intervertebral disc herniation require?

Tasks for self-control

1. A 34-year-old male patient, while lifting a load of about 30 kg, felt a sharp pain in the lower back, buttocks, and on the back of the right leg. Objectively, the examination revealed an antalgic posture, suppression of tendon reflexes in the right leg, a positive Lasegue symptom on the right. What is the probable diagnosis?

2. A 24-year-old female patient has diagnosed a tumor via magnetic resonance imaging: a tumor of the cone of the spinal cord. A tumor removal operation was performed. The lower paraplegia, sphincter disturbances remain in the neurological status of the patient. Which complications should be prevented?

3. A 45-year-old female patient gradually developed symptoms of spinal cord compression at the level of the Th10. To clarify the nature of the process, the patient underwent lumbar puncture with cerebrospinal fluid tests. What cerebrospinal fluid changes including dynamic changes can be detected in the patient?

4. A 43-year-old male patient after a neurological examination was diagnosed with L5—S1 intervertebral disc herniation. Which research method is the most informative?

5. A 44-year-old male patient after a chiropractic session developed acute low back pain with irradiation to the left leg along the lateral surface of the thigh and lower leg. Objectively during the examination: sharply positive Lasegue symptom, hypoesthesia in the area of L5-root innervation on the right, moderate paresis of the dorsiflexion of the right foot. What preliminary diagnosis can be established?

6. A female patient has complained of moderate pain in the left half of the chest for the past 3 years. Over the past six months, the left foot and then the thigh began to weaken, the woman began to walk poorly. Objectively: monoparesis of the left leg, decrease in pain and temperature sensitivity on the right from the level of the D5-segment, segmental loss of sensitivity in the Th3 dermatome on the left. The clinical picture of which disease was described?

7. The female patient is 65 years old and suffers from urinary incontinence, perineal numbness, weakness in the legs for 2 months. She suffers from insulin-dependent diabetes mellitus and coronary heart disease. The examination revealed a tumor of the cauda equina. What treatment is indicated for the patient?

8. In a patient who had been treated for discogenic radiculitis, protein cell dissociation was detected after lumbar puncture. Magnetic resonance imaging revealed a zone of increased pathological density at the level of the S1-root. What is the correct diagnosis?

9. A patient was diagnosed with sequestered disc herniation L4—L5, instability of the lumbar spine at the same level. What should be the treatment strategy?

10. A patient was diagnosed with an intramedullary tumor at the level of the Th4-segment of the spinal cord. On the second day after hospitalization, the patient's condition deteriorated sharply: the movements in the legs disappeared, there were numbness below the dermatome Th_6, urinary retention. What complication has developed?

Topic 5

HYDROCEPHALUS

Hydrocephalus is an abnormal increase in the amount of cerebrospinal fluid (CSF) within the cranial cavity that is accompanied by an expansion of the cerebral ventricles, enlargement of the skull and especially the forehead and atrophy of the brain.

Hydrocephalus was first described by Hippocrates. Hydrocephalus was not treated effectively until the mid 20th century when the development of appropriate shunting materials and techniques occurred.

Because hydrocephalus has many different causes, it is difficult to classify specific varieties. As a result, many different classification systems have been developed. Hydrocephalus can be grouped based on two broad criteria: 1) pathology and 2) etiology.

Pathology can be grouped as either obstructive (non-communicating) or non-obstructive(communicating). Etiology can be grouped as congeni-tal or acquired.

Congenital hydrocephalus is caused by a birth defect or genetic disorder; acquired hydrocephalus has other causes, such as a hemorrhage, infection, or tumor.

Obstructive hydrocephalus is caused by an obstruction of CSF drainage by cysts, tumors, hemorrhages, infections,congenital malformations, and most commonly, aqueductal stenosis or cerebral aqueductblockage. Most cases of hydrocephalus are obstructive.

Communicating hydrocephalus is caused by an overproduction of CSF that allows insufficient drainage time, or occurs when CSF is not absorbed in a normal rate. Examples include cranial hemorrhage and meningitis.

By localization of the liquid hydrocephalus is divided into external (the liquid accumulates in ventricles of the brain); by the character of the functioning of liquor system — into open (circulation of the CSF is not disturbed) and occlusive, or closed (disorders of circulation at different levels of liquor system). In its turn, the open hydrocephalus can be antiresorptive (slow absorption of the CSF), hypersecretory intensification of secretion in constant resorption, and mixed. The occlusive hydrocephaly can be caused by impairment of liquid outflow at the level of interventricular apertures (Monroe), III ventricles, an aqueduct of the brain, IV ventricles, median and lateral apertures of IV ventricles, cerebellar — medulla oblongata tanks. By course hydrocephalus is divided into acute and chronic, by stages — progressing and stabilized.

Describing hydrocephalus based on the type of CSF flow (i.e. communicating/non-obstructiveor non-communicating/obstructive) is preferred because of the implications for treatment.

There are two other forms of hydrocephalus that do not fit distinctly into the categories mentioned above and primarily affect adults: hydrocephalus ex-vacuo and normal pressure hydrocephalus.

Hydrocephalus ex-vacuo occurs when there is damage to the brain caused by stroke or traumatic injury. In these cases, there may be actual shrinkage (atrophy or wasting) of brain tissue. Normal-pressure hydrocephalus can occur in people of any age, but it is most common in the elderly population. It may result from a subarachnoid hemorrhage, head trauma, infection, tumor, or complications of surgery. However, many people

develop normal pressure hydrocephalus even when none of these factors are present. In these cases the cause of the disorder is unknown.

The rate of the congenital forms of hydrocephalus reaches 2—5 on 1000 newborns. It is a disease of mainly early children's age, from birth till the first year of life. It is necessary to distinguish precisely hydrocephalus as the disease having the clinical picture, course, the prognosis, and hydrocephalus as a syndrome in various neurosurgical diseases and traumatic affection of the brain.

About 80 % of hydrocephalus patients are born with other defects. Other medical conditionsusually associated with hydrocephalus include arachnoid cysts; brain injury; Dandy-Walker Syndrome; meningitis; porencephaly; tumors;spina bifida (hydrocephalus occurs in 70 to 90 % of children with the most severe form of spina bifida).

It is considered, that in the development of hydrocephalus the birth trauma, asphyxia, and hypoxia during delivery have essential value. However the major importance in the development of hydrocephalus has infectious affection of the fetus, it is more often the virus, during the pregnancy of the mother. Probably there may be intranatal infection of the fetus during delivery, as a result of contact with causative agents in the area of the generic duct, most often with a virus of genital herpes and chlamydia. Premature infants have an increased risk of intraventricular hemorrhage in which severe bleeding within the ventricles of the brain can lead to hydrocephalus.

Pathological findings

CSF is the fluid that acts to serve as a cushion for the brain and plays a role inhaemostasis and metabolism of the brain. It is produced by the choroid plexus, found inthe body and inferior horn of the lateral ventricle, the foramen of Monroe, the roof of thethird ventricle and the inferior roof of the fourth ventricle. The flow of CSF through theventricles is as follows: begins in the left and right lateral ventricles — interventricularforamen of Monroe — 3rd ventricle — cerebral aqueduct — 4th ventricle and out throughthe two lateral apertures of Lushka or the one medial aperture of Magendi into thecisterna magna. From there, CSF will flow into the cortico-subarachnoid space and thespinal subarachnoid space. CSF is continuously being produced by the choroid plexus at a rate of 400—500 ml/day andcontinuously reabsorbed by the arachnoids granulations into the dural sinuses, andeventually into the venous system. At any given time, there is approximately 140 ml of CSFin adult system, of which 25—40 ml is in the ventricles. The rate of absorption isproportional to the difference in intracranial pressure and dural sinus pressure. An equilibrium between CSF production and CSF reabsorption maintains mean CSF pressure at 7—15 mm Hg in normal adults.

So pathophysiological classification of hydrocephalus present a subdivision into the following 3 forms:

1. Disorders of CSF production: This is the rarest form of hydrocephalus. Choroid plexus papillomas and choroid plexus carcinomas can secrete CSF over its absorption.

2. Disorders of CSF circulation: This form of hydrocephalus results from obstruction of the pathways of CSF circulation. This can occur at the ventricles or arachnoid villi. Tumors, hemorrhages, congenital malformations (such as aqueductal stenosis), and infections obstructat either point in the pathways.

3. Disorders of CSF absorption: Conditions, such as the superior vena cava syndrome and sinus thrombosis, can interfere with CSF absorption. Some forms of hydrocephalus cannot be classified clearly. This group includes normal pressure hydrocephalus and pseudotumor cerebri.

Clinical features

Symptoms of hydrocephalus vary with age, disease progression, and individual differences intolerance to CSF. For example, an infant's ability to tolerate CSF pressure differs from an adult's. Theinfant skull can expand to accommodate the buildup of CSF because the sutures (the fibrous jointsthat connect the bones of the skull) have not yet closed.

In infancy (0—2 years), the most obvious indication of hydrocephalus is often the rapid increase in headcircumference or an unusually large head size, bulging fontanelles, and bulging scalp veins, which occurs especially whenthe infant cries. Other symptoms may include vomiting, sleepinessand irritability, seizures, and developmentaldelays. As hydrocephalus worsens, the infant may suffer from «sunsetting eyes». This symptom ischaracterized by the child's inability to look upward, as the eyes are displaced downwarddue to the pressure on the cranial nerves controlling eye movement. As a result, the infantappears as though it is looking at the bottom lid of its eye. Vision may also be affected inadvanced hydrocephalus due to compression of the optic chiasma as a result of a dilated 3rd ventricle. Stretching of periventricular structures can cause abducent nerve paresis,presenting in nystagmus and random eye movement.

Infants with advanced hydrocephalus may also present with increased deep tendon reflexesand muscle tone in lower extremities, growth failure, delayed neurological development, and limited control in the head and trunk regions. Left untreated, this can progress and canresult in seizures and/or coma.

Older children and adults may experience different symptoms because their skulls cannot expand toaccommodate the buildup of CSF. In older children or adults, symptoms may include headachefollowed by vomiting, nausea, papilledema (swelling of the optic disk which is part of the optic nerve), blurred vision, diplopia (double vision), sunsetting of the eyes, problems with balance, poorcoordination, gait disturbance, urinary incontinence, slowing or loss of development, lethargy,drowsiness, irritability, or other changes in personality or cognition including memory loss. Affections of the cranial nerves, motor and cerebellar systems, spasms, mental disorders can be observed.

Symptoms of normal pressure hydrocephalus include progressive mental impairment and dementia, problems with walking, and impaired bladder control leading to urinary frequency and/or incontinence.

Disorders of the movement have various characters; there may be different pareses, sometimes in a combination with hyperkinesias. Cerebellar dysfunctions are manifested by disturbance of statics and coordination of movements. Children often cannot go, stand, and even sometimes sit and hold the head. There can be significant retardation in intellectual development, hypererethism and irritability or weakness, and adynamia, indifference to everything.

The person also may have a general slowing of movements or may complain that his or her feet feel «stuck». Because these symptoms are similar to those of other disorders such as Alzheimer's disease, Parkinson's disease, and Creutzfeldt — Jakob disease, the disorder is often misdiagnosed. Many casesgo unrecognized and are never properly treated. Doctors may use a variety of tests, including brainscans (CT and/or MRI), a spinal tap or

lumbar catheter, intracranial pressure monitoring, andneuropsychological tests, to help them diagnose normal pressure hydrocephalus and rule out otherconditions.

Diagnostic evaluation

Head circumference should be routinely measured in infants. Any excessive growth in serialmeasurements is a risk factor for hydrocephalus and should be followed up with aphysician.

Hydrocephalus is diagnosed through clinical neurological evaluation and by using cranialimagingtechniques such as ultrasonography, CT, MRI, or pressure-monitoring techniques.Hydrocephalus can be diagnosed before birth with the use of ultrasound. Also, inpremature infants and very young infants with open fontanelles, ultrasound can be used toimage the size of ventricles.For adequate treatment, it is necessary to specify hydrocephalus and a level of occlusion of the liquor ways.

Treatment

Conservative methods of treatment in hydrocephalus are ineffective and can be used during the limited time at the early stages of its development as a course of dehydration therapy.

Patients with communicating hydrocephalus, including adult NPH, are primarily treatedwith shunt surgery.Surgical methods of treatment are widely used directed at formation of collateral ways of outflow of CSF beyond the central nervous system and normalization of intracranial pressure. The main indication for operation is progression of hydrocephaly in absence of inflammation of the brain membranes.

Shunt systemdiverts the flow of CSF from a site within the central nervous system (CNS) to another area of thebody where it can be absorbed as part of the circulatory process.

A shunt is a flexible but sturdy elastic tube. A shunt system consists of the shunt, a catheter, and avalve. One end of the catheter is placed in the CNS — most usually within a ventricle inside the brain,but also potentially within a cyst or in a site close to the spinal cord. The other end of the catheter iscommonly placed within the abdominal cavity, but may also be placed at other sites within the bodysuch as a chamber of the heart or a cavity in the lung where the CSF can drain and be absorbed. Avalve located along the catheter maintains one-way flow and regulates the rate of CSF flow.

Shunt systems are not perfect devices. Complications may include mechanical failure, infections, obstructions, and the need to lengthen or replace the catheter. Generally, shunt systems requiremonitoring and regular medical follow-up. When complications do occur, usually the shunt system willrequire some type of revision.

Some complications can lead to other problems such as over-draining or under-draining. Overdrainingoccurs when the shunt allows CSF to drain from the ventricles more quickly than it isproduced. This over-draining can cause the ventricles to collapse, tearing blood vessels and causingheadache, hemorrhage (subdural hematoma), or slit-like ventricles (slit ventricle syndrome). Underdrainingoccurs when CSF is not removed quickly enough and the symptoms of hydrocephalus recur.

In the case of occlusive hydrocephalus in children over 3 years and adults when it is impossible to remove the direct cause of the occlusion by the surgical way the operation of ventriculocysternostomy was suggested by

Torkildsen in 1993. The essence of the operation is that with the help of a catheter the connection between a lateral ventricle and cerebellar — medulla oblongata tank is formed.

In case of occlusion at the level of the IV ventricle and its apertures, and also aqueduct of the brain it is possible to make unilateral ventriculocysternostomy. However, taking into account the possibility of displacement of a catheter and stop of the functioning of the shunt, it is worth making bilateral shunting at such levels of occlusion. If there is occlusion at the level of the interventricular aperture or III ventricle, bilateral shunting is obligatory as lateral ventricles can be divided.

Universal operations can be used in case of both open, and occlusive hydrocephalus. The most widespread are two kinds of such operations — valvular ventriculo-diostomy and ventriculoperitoneostomy. During the first operation excess CSF is removed from the ventricles for the regulation of outflow of liquid to avoid the sharp decrease in the intracranial pressure. Each type of valve is designed for a certain pressure of CSF, below which the valve is closed and ceases to function.

The success of surgery in hydrocephalus depends on the approach to a choice of a kind of the operation (it should be individual), skills to define the precisely optimum term of intervention, and to provide such system with dosed removal of CSF which would form the best conditions for its circulation. Some surgeons make subarachnoidal-sinus shunting when one end of the shunt is introduced in the subarachnoid space, and another — in the nearest sinus.

Topic 6

FUNCTIONAL AND STEREOTACTIC NEUROSURGERY

Functional neurosurgery — is a set of methods of surgical influence on the root, leading paths, and neuronal structures of the central nervous system, based on the anatomical and physiological patterns of pathological processes in the central nervous system, and aims to change the pathogenesis of these processes to obtain the therapeutic effect.

The stereotactic method — is a collection of techniques and calculations that allow using special devices and methods of X-ray and functional control with great accuracy to enter the electrode into a predetermined depth structure of the brain or spinal cord.

The essence of such interventions is to provide spot therapeutic and diagnostic effects in strictly defined areas of the brain. Stereotaxis makes it possible in case of various diseases of the central nervous system to perform low-traumatic interventions on the deep parts of the brain, inaccessible under normal neurosurgical. Extremely important is the statementby M. Burdenko, which, in our opinion, has a direct relation to stereotaxis: «Stereotaxis should provide anatomical availability, technical ability and physiological permissibility of stereotactic manipulations». Only through the stereotactic method, surgical interventions on deep subcortical and stem structures of the human brain were possible.

Modern stereotaxis provides an opportunity of:

— low traumatic surgical access to deep structures or areas of the brain;

— obtaining information from deep structures;

— aiming local influence on selected brain areas for the diagnosis and treatment of various diseases of the central nervous system. Currently, the stereotactic method is used both in the typical neurosurgical pathology and in the treatment of certain diseases, which are usually referred as the scope of neurology and psychiatry. Depending on the diseases that are indications for the operations, non-functional and functional stereotaxis are determined.

Unfunctional stereotaxis includes, as a rule, different types of neurosurgical pathology:

— brain tumors (using a stereotaxic method, diagnostic biopsy, therapeutic destruction of tumor tissue, the targeted introduction of chemotherapeutic drugs into tumors, stereotactic craniotomy — use of stereotactic equipment for navigation support microsurgery for the removal of tumors);

— intracerebral hematomas (stereotactic evacuation of the hematoma);

— abscesses of the brain (evacuation of abscesses of the brain, insertion of a catheter for rinsing of the abscess with solutions of antiseptics, antibiotics into the cavity);

— alien bodies (removal of alien bodies).

Various types of neurological and psychiatric pathology refer to functional stereotaxis:

— motor disorders (stereotactic interventions are used to eliminate tremor, hyperkinesis, for normalization of muscle tone). Among the nosological forms that are indications for functional stereotactic interventions that is, Parkinson's disease and other variants of parkinsonism, essential tremor, deforming muscular dystonia, including spastic cervix play a leading role;

— unbridled pain (operations on the deep structures of the brain contribute to eliminating or reducing the severity of the pain syndrome). As a rule, stereotactic operations can be performed with phantom pain, but are sometimes used in some other variants of non-curable pain syndromes;

— epilepsy (the stereotactic method is used both for the diagnosis of localization of intracerebral epileptic lesions and for therapeutic impact on the brain to reduce the frequency or eliminate attacks);

— mental disorders (stereotactic effects on the deep structures of the brain, usually associated with the limbic system, may contribute to the improvement of the state in many cases of pharmacokinetic resistance of mental disorders). Stereotactic neurosurgical treatment can be used with diseases such as obsessive-compulsive disorder (including narcotic and alcoholic obsessions), depression, anxiety, aggressiveness, and several other conditions.

Basic stages of stereotactic interventions

1. *Anesthesia.* Methods of pain relief depending on the nature of the disease and the tasks set. For example, local anesthesia with a light premedication is the method of choice in case of parkinsonism and pain syndromes, to control the reduction or complete disappearance of the tremor and rigidity during the operation. Naturally, for these observations, the patient should be in complete consciousness, answer the questions, and follow the instructions of the surgeon. With evident hyperkinesis, local anesthesia is not used, since it does not allow you to get correct tomograms of the head. In these cases, intravenous or endotracheal anesthesia is required.

2. *Determination of the place of trepanation.* When choosing a place of trepanation three items should be taken into consideration:

a) the place of the insertion of the probe must be located at a safe distance from functionally important areas of the brain;

b) the probe on the way to the specified structure should not damage other important structures of the deep zones of the brain;

c) the path of the probe should be as short as possible.

1. The technique of stereotactic cryodestruction of the ventrolateral nucleus of the visual hump in patients with parkinsonism under X-ray control

In the position of the patient on the back in the parietal region, retreating from the nasion of 12.5—13 cm, from the middle line to 3.5 cm, linear incision length of up to 4 cm is performed. Hemostasis. Skeletalization of the parietal bone is conducted. Bone fragment using a crown cutter with a diameter of 25 mm, is cut out and is placed into the physiological solution. Hemostasis. The dura mater usually has a normal color, clearly transmits the pulsation of the brain. It is opened crosswise. Hemostasis. The substance of the brain has normal color, gyrus and furrows are clearly expressed. The platform of the stereotactic apparatus and then the coordinator is installed in the bone hole. A brain cannula is introduced in the projection of the anterior horn of the lateral ventricle. Transparent liquor is received at a depth of 50—55 mm.Liquor flows under normal pressure. 5.0 ml of liquor is extracted and is mixed with 5.0 ml of contrast agent (Conrey or Omnipack), the resulting mixture is injected intraventricularly, then X-rays is performed. Intraoperative calculations of the surgical target for cryodestruction (VL TH) are performed according to the data of the stereotactic atlas of the stereotactic coordinates G. Shaltenbrand, P. Bailey after the ventriculography, inthe direct and lateral shots (Fig. 6.1). Depending on the predominance of a clinical picture of tremor or rigidity one or another group of nuclei Th (VOa and VOp — to normalize muscle tone, Vim — to eliminate the tremor) is chosenas a «target». The technique of stereotactic calculations is described in detail in the famous monographs.

Fig. 6.1. **Ventriculograms with water-soluble substance and stereotactic calculations of the ventrolateral nucleus of the thalamus:**
1 — cryoprobe; *2* — anterior commissure; *3* — posterior commissure; *4* — surgical target; *5* — the third ventricle

The obligatory stage of the stereotactic operation is functional control, the elementary example of which is the effect in the form of complete or partial cessation of the tremor and normalization of muscle tone in the limbs opposite to the area of the performed operation, as a result of slight mechanical damage of the nuclei of the subcortical structures (in our case, this ventrolateral group of nuclei of the thalamus). However, to objectivize the accuracy of the cannula, a diagnostic electrostimulation (EC) is performed. By the corrections made, the data is set on the coordinator. Platinoridium electrode diameter of 300 micronscovered with Bakelite varnish with an active working end surface of 5 mm is installedinto stereotactic apparatus, which is injected intracerebrally to the

appropriate depth. After that repeated photographsand calculations are performed. In the case,if the tip of the electrode is at the point of the target, electrical stimulation is performed with an anode current of 5 V and a frequency of 50 Hz and pulses of rectangular shape and duration of 1 ms. In opposite extremities, as a rule, the acceleration of the rhythm of tremor is demonstrated, indicating that the working end of the electrode is outside the inner capsule, and in the VL Th. The electrode is extracted from the substance of the brain and autonomous neurosurgicalcryozond with an external diameter of 2 mm and internal vacuum isolation is set up into the stereotactic apparatus (Fig. 6.2)

Fig. 6.2. **Autonomous neurosurgical cryozond**

Cryozond is injected into the substance of the brain. After the introduction, a sharp loss of the rhythm of the tremor and the reduction of hypertonicity in the opposite limbs is usually noted. Cryodestruction VL Th for 1—2 minutes under the control of muscle tone and strength in the limbs (Fig. 6.2). Usually, as a result of cryodestruction, complete or significant regress of the tremor in the limbs and reduction of rigidity in them are noted. After 10 minutes, the cryozond is extracted from the substance of the brain. Hemostasisis conducted, the plastic coveringof the defect of dura mater is carried out by fluoroplast film, the bone fragment is set and fixed by nodular sutures to the periosteum; layered sutures and an aseptic bandageare imposed on the wound.

During the first day the patient is under the supervision in conditions of a resuscitation department, where hemostatic, dehydration, anti-inflammatory and symptomatic therapy are performed.

2. The technique of stereotactic cryodestruction of the ventrolateral nucleus of the visual hump in patients with parkinsonism under the control of a computed tomograph

In the position of the patient on the back in the parietal region, retreating from the nasion of 12.5—13 cm, from the middle line to 3.5 cm, linear incision length of up to 4 cm is performed. Hemostasis. Skeletalization of the parietal bone is conducted. Bone fragment using a crown cutter with a diameter of 25 mm, is cut out and is placed into the physiological solution. Hemostasis. The dura mater usually has a normal color, clearly transmits the pulsation of the brain. It is opened crosswise. Hemostasis. The substance of the brain has normal color, gyrus and furrows are clearly expressed. The platform of the stereotactic apparatus and then the coordinator is installed in the bone hole. CT control is conducted. Computer-tomographic examination of the subtalamic region during the stereotactic operations shows the main orienting points of stereotactic calculations — cisterna ambiens and the bottom of the III ventricle (Fig. 6.3).

Fig. 6.3. **Computed tomography landmarks of the subthalamic area during stereotactic operations:**

1 — thalamus; *2* — biconvex nucleus; *3* — black substance; *4* — hypothalamus; *5* — caudate nucleus; *6* — nuclei of the thalamus

The end of the cryozond is located near the edge of the left horn of the cisterna ambiens and the bottom of the III ventricle, which corresponds to the point of the target in which the cryozond is introduced. Cryodestruction for 1—2 minutes under the control of muscle tone and force in the opposite limbs is conducted. The following steps are the same as in the previous technique.

Stereotaxic biopsy

Several variants of the stereotactic cannula design for biopsy are known. The most suitable for biopsy of intracerebral tumors is Sedan-Nashold cannula, consisting of two tubes, concentrically inserted into each other. Each of these tubes has a sealed end and a small aperture in the side surface near the tip. The outer diameter of the cannula is 1.5—2.5 mm. When you enter the cannula into the brain, its limiter is set so that the hole on its active end corresponds to the position of the target point. When reaching the target point, the rotation of the inner tube about the external, providesthe connection of the holes in both tubes, thereby opening the «window» to take a biopsy. Then a syringe is attached to the proximal end of the inner tube, throughpulling the piston, discharge in it is created, so that the area of the brain tissue in the region of the target point is sucked into the tube. At the same time, the rotation of the inner tube on its axis is produced, thereby cutting the edge of the «window» fromthe extracted piece of tissue. After that, the inner tube is taken out along with the material taken for the research. The material is placedinto a test tube, then the inner tube is inserted back into the external one. Usually, a few samples of the material at the target point and at points located in the stereotaxic cannula are taking. The volume of the tissue sample obtained is several cubic millimeters, depend on the inner diameter of the cannula and the size of the window at its end.

Impactusing intracerebral electrodes

There are several variants of the design of the electrodes, allowing tocarryout diagnostic and therapeutic impacts on the brain tissue. Bands of bonded electrodes are suitable for long-term implantation (for a period ofseveral weeks to several months). The technique of stereotaxic access and methods of impact at the target points with six wired electrodes with a nichrome or gold wire with a diameter of 0.1 mm, covered with fluoroplastic isolation is given below. The area of the contact surface of each electrode is 0.1—0.2 mm, the distance between the contact surfaces of the adjacent electrodes in the beam is about 2 mm. Such electrodes can be used to record electrosubcorticogram, for electrolysis of brain tissue with direct current, and diagnostic electrostimulation and electrolysis. Recording of the electrobascorticogram is usually used as an invasive diagnostics in patients with epilepsy. Due to the possibility of implantation for a long period, the records are made repeatedly and at different times of the day, allowing to register the epileptic foci more objectively. For local destruction by direct current (DC) (anode electrolysis), a stabilized DC source is used. The destruction is made bipolar, that is, through two adjacent contact surfaces of the electrode beam: on one of the surfaces positive electrical charge is fed, and the negative charge is fed on the other. The destruction

zone of 4—5 mm in diameter is received by 5mA current and an exposure of 300 ms. (Fig. 6.4). The method is used for local destruction of the brain regions both independently and in combination with other methods of local stereotaxic effect in the treatment of patients with parkinsonism, epilepsy, psychiatric disorders, etc. Only the electrode bundles made of gold wire are suitable for electrolysis.

Fig. 6.4. **CT stereotactic calculations of the subthalamic area: horizontal (*a*) and axial (*b*) CT:**
1 — forehead; *2* — nape; *3* — right; *4* — on the left; *5* — ambient cystern;
6 — cannula; *7* — III ventricle

Electropolarization is a reversal shutdown of the brain tissue that is performed before conducting anode electrolysis using long-term electrodes. These actions are used with a diagnostic purpose, and allowto avoid complications after electrolysis. Electropolarization is carried out by constantly increasing DC from 0 to 1 mA for 10 s, and then after 10 seconds, the current is gradually reduced to zero. Diagnostic electrostimulation through implanted long-term electrodes is performed by bipolar, rectangular current pulses for 1 msec, by series from 4 to 50 pulses per second, current strength from 0.1 to 2 mA, depending on the threshold of excitability of the investigated brain structures. Electrostimulation of deep intracerebral targets can be performed both for the physiological identification of the structures in which the implants are incerted and for therapeutic purposes with epilepsy, parkinsonism, and other motor disorders. Curative electrostimulation through long-term electrode bundles is carried out by series of pulses for 5—10 s with an interval of 1 min, from 10 to 30 times.

Methods of guidance in modern stereotactic neurosurgery

Electrodes of cylindrical shape with a diameter of 0.8—1.2 mm with 4—6 ring-shaped contact surfaces located near the active end can be used both for short-term and for life implantation in the brain of the patient. The material for contact surfaces is stainless steel or platinum-iridium alloy. Such electrodes are made in the conditions of industrial production (firms DIXI, Medtronic, etc.). With the help of electrodes of this typechronic therapeutic electrostimulation (DBS-deepbrainstimulation) of deep subcortical structures in patients with motor disorders, psychiatric disorders, pain syndromes, epilepsy, and Alzheimer’s disease are performed. Opposite to the courses of therapeutic stimulation performed by longitudinal electrode beams, the implantation of the electrodes and electrostimulation of the brain structures of the patient (neuromodulation) are made forever.

The peripheral ends of the implanted electrodes are input through the milling hole underneath the skin and are connected to the pulse generator implanted in the subclavian area. Electrical stimulation is carried out by alternating current with the frequency of 80—185 Hz. The clinical effect of electrostimulation is similar to the effect of destruction in the corresponding intracerebral structures. However, unlike destructions, the effect of electrostimulation is the reversal and does not cause damage to the brain tissue. The termination of stimulation

causes the repeated appearance of symptoms in the patient. The optimum amplitude and frequency for suppression of pathological symptoms are chosen individually in the postoperative period.

Electrodes of this type are also suitable for intraoperative recording of electrosubcorticograms (diagnosis of epileptic activity in the deep structures of the brain). The ability to perform diathermocoagulation of deep structures of the brain in parkinsonism is described (M.Y. Oh et al., 2001). Coagulation is carried out by bipolar current of 38 mA and the frequency of 250 kHz for 60 s through adjacent contact surfaces, with a preliminary mono- and bipolar diagnostic electrostimulation with a frequency of 150 Hz and a voltage from 0 to 10 V.

Stereotactic thermal destruction

The most frequently performed stereotactic destructive impact on the deep structures of the brain in functional neurosurgery is diathermocoagulation with high-frequency alternating current (radiofrequency thermal destruction). The tissue destruction area of the brain, obtained this way, is delimited from the surrounding tissue. Also, the destruction zone can be correlated, changing the temperature and the duration of exposure.

Apparatus for stereotactic thermodestruction with alternating current consists of multiple electrodes with a diameter of 1.1—1.6 mm for stereotaxic injection into the target points of the brain and of agenerator. The monopole electrode is insulated all over its length, except the active end of 3—10 mm. COSMAN RFG-1A and COSMAN G4 radiofrequency generators have become the most commonly used sources of AC. The devices allow regulating the frequency and amplitude of current, the temperature of the active end of the electrode, and the exposure time of high temperature, which affects the final zone of the brain tissue destruction. With the help of the devices described it is also possible to conduct diagnostic procedureswhere the electrode is used — impedance measurement, test electrical stimulation, and registration of electrical potential of the brain.

Destructionis carried out by a monopolar alternating current of 500 kHz at the temperature of 60—85 °C and the exposure of 60—120 s. Diagnostic examination before destruction includes impedance measurement (which allows distinguish grey, white substance, and liquor), test electrostimulation (the current up to 10 mA, the frequency of 60 Hz, and the current of 5 mA and the frequency of 120 Hz) and short-term reversal shutdown of brain tissue by heating it to a temperature of 42—44 °C. In some clinics, microelectrode registration is additionally conducted at the target points to better recognize the subcortical nuclei. In the absence of negative effects, the final exposure is carried out at the target points. The diameter of the destruction area in brain structures, depending on the size of the electrode and the parameters of impact, can range from 3—4 to 12 mm.

Another method of stereotaxic thermal destruction is the effect of high-energy laser irradiation. This method of destruction can be used for stereotactic destruction of intracerebral tumors. As a source of radiation, carbon dioxide (CO_2) and neodymium-yttrium-alumina-pomegranate (Nd: YAG) lasers with a wavelength of 10.6, 1.064, and 1.318 microns are commonly used. The laser radiation is fed into the tumor tissue by stereotactic light guides introduced into the target point. In some cases, a preliminary systemic injection of photosensitizers to increase tumor tissue energy absorption (stereotaxic photodynamic therapy) is carried out. The disadvantage of laser stereotactic effects is the impossibility to predict the volume and shape of the resulting destruction due to the inhomogeneous optical density of the tissue, which makes it impossible to plan the position of the destruction

centers carefully. Another significant drawback of laser thermal destruction, which limits its application, is the impossibility of temporal shutdown at the target points.

Other methods of stereotactic effects

In some clinics, patients with intracerebral neoplasms of small size, are subjected to stereotactic craniotomy — the method of open microsurgical removal of the tumor, in which access to deeply arranged formation is performed using stereotactic technique and is low traumatic. Local intra-tumor chemotherapy is performed by stereotaxic implantation of a catheter into the tumor cyst, then it is connected subcutaneously with the implanted reservoir Ommaya, in which injections of medicinal substances are made in the postoperative period.

Stereotactic evacuation of intracerebral hematomas and abscess content is carried out with the help of a two-channel cannula with a screw, which operates like the «screw by Archimedes». One of the channels is used for the removal of pathological fluid, the second can be fed with the physiological solution for washing clots that are extracted with a screw. With a predominance of clots in thecavity with the pathological fluid adrainage is inserted for several days. Through the drainage drugs with fibrinolytic activity can be injected to gradually dissolve clots. Non-functional stereotactic surgery can be successfully used in the surgical treatment of patients with benign intracranial hypertension. Since the ventricles in such patients are not usually dilated, the stereotactic method in such cases allows less traumatic implantation of the ventricular catheter and ventriculoperitoneal bypass surgery.

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