MINISTRY OF HEALTH OF UKRAINE

ODESA NATIONAL MEDICAL UNIVERSITY

Faculty of Medicine №2

Department of Neurology and Neurosurgery

APPROVED BY
Vice-Rector for Scientific and Educational Work
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TEACHING MATERIAL
FOR LECTURES ON THE ACADEMIC SUBJECT

Faculty, Course: Stomatological, 4th year
Academic Discipline: **Neurosurgery**

Approved by:
Meeting of the Department of Neurology and Neurosurgery
Odesa National Medical University
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LECTURES

Lecture No. 1 .

Topic: Traumatic lesions of the nervous system. Closed traumatic brain injury. Open traumatic brain injury, clinic, diagnosis. Spinal cord injury. Traumatic injuries of the peripheral nervous system.

Actuality of theme: Traumatic lesions of the central and peripheral nervous system remain an urgent problem in the modern world with numerous medical, social and economic consequences.

Entire lectures

Educational:

- To acquaint students with the etiology and pathogenesis of traumatic lesions of the nervous system.
- During the lecture, provide students with knowledge of the classification of traumatic lesions of the neovascular system, the main clinical symptoms.
- On the basis of the lecture material, provide students with the opportunity to master the skills of diagnosis and treatment of traumatic lesions of the nervous system.

Educational:

- Aimed at the development of a professionally significant personality substructure ;
- Education of modern professional thinking in students ;
- Ensuring that students master the leading value of domestic clinical, scientific and pedagogical schools.
- Acquisition of deontology and medical ethics skills by students.

Basic concepts: closed brain injury, Glasgow coma scale, dislocation syndrome, open brain injury, fracture of the bones of the skull, compression -ischemic (tunnel) neuropathies, basic principles and methods of surgical interventions for peripheral nerve injuries, depending on the type, level and mechanism of damage, principles of restorative treatment of patients in the postoperative period".

Equipment: laptop, multimedia projector.

Plan and organizational structure of the lecture

No	The main stages of the lecture and their content	Goals in levels of abstraction	Type of lecture, methods and means of activation of acquirers, equipment	Time allocation
I 1. 2.	Preparatory stage Setting an educational goal Providing positive motivation	 I I	According to the publication "Methodical recommendations for planning, preparation and analysis of a lecture"	5% (5 min)
II 3.	The main stage Presentation of lecture material according to the plan:		Slide presentation of lecture material	85% (75 min)

	1. Actuality of theme 2. Definition 3. Classification 4. Etiology and main links of pathogenesis 5. Clinical picture 6. Diagnostics 7. Leading syndromes and differential diagnosis 8. Evaluation of the severity of the course 9. Treatment 10. Prevention	II II II II II II II II II II	Extracts from the medical histories of patients. Excerpts from clinical protocols of the Ministry of Health of Ukraine on providing medical care to patients.	
III 4. 5. 6.	The final stage Summary of the lecture, general conclusions Answers to possible questions Tasks for independent preparation	 III III III	List of references, questions, assignments	10% (10 min)

Content of the lecture material.

- Classification of brain injury:
- Closed cerebellar injury
- Brain concussion
- Cerebral contusion
- Compression of the brain
- Diffuse axonal damage
- is open
- Penetrating
- Impenetrable

By degree of severity

Mild SKG 14 OR 15 + OR brief loss of consciousness (<5 min) OR impaired concentration of attention.

1. Moderate SKG 9-13 OR loss of consciousness ≥ 5 min OR focal neurological deficit.
2. Severe SHKG 3-8.

A brain concussion is a complex of pathophysiological processes that affect the brain and result in a change in brain functions; induced by nonpenetrating biomechanical forces with no detectable abnormalities on standard structural imaging. Neurological symptoms (do not) include loss of consciousness.

The onset of symptoms is usually acute, they are short-term and disappear spontaneously. Manifestations may include transient disturbances in balance, coordination, memory/cognition, strength, or concentration.

Neuropathological changes may occur, but acute clinical symptoms mostly reflect functional changes rather than structural pathology.

Diagnosis is based on history and clinical picture.

Cerebral infarction is characterized by the presence of areas of high density on CT images. It most often occurs when the head slows down sharply, which leads to the impact of the brain on bony protrusions (temporal, frontal, occipital bones) by the "ku-contraku" mechanism. Surrounding areas of low density on CT images suggest cerebral edema.

The focus of the beating is an area of brain tissue necrosis with a perifocal zone in which

inflammatory reactions, secondary damage, and edema develop. Depending on the severity of the injury, small focal hemorrhages, small areas of brain tissue homogenization, as well as the formation of massive necrosis centers filled with brain detritus, rupture of blood vessels, hemorrhages in the destroyed tissue, pronounced edema-swelling of the perifocal zone with a tendency to spread can be found in the area of the injury, depending on the severity of the injury. to adjacent areas, sometimes to the entire brain.

With a **mild stroke** , loss of consciousness is observed (the duration is usually described as minutes), intense headache, dizziness, weakness, tinnitus, often amnesia, severe nausea, repeated vomiting. Violation of vital functions is not characteristic, sometimes moderate tachycardia or (less often) bradycardia, flushing of blood to the face, sleep disturbances, etc. are noted. Spontaneous nystagmus, mild anisocoria, signs of pyramidal insufficiency, and mild meningeal symptoms are also characteristic of neurological symptoms. Usually, the described neurological symptoms completely regress within 2-3 weeks.

A stroke **of moderate** severity is accompanied by a loss of consciousness that lasts from several tens of minutes to several hours (the duration is usually described as hours). Amnesia is almost always detected, the characteristic presence of a headache, intense and long-lasting, repeated vomiting, possible mental disorders. This form of craniocerebral trauma is also characterized by temporary disturbances of vital functions (bradycardia or tachycardia, increased blood pressure, tachypnea without respiratory rhythm disturbance, subfebrile); trunk disorders are sometimes observed. Meningeal symptoms are pronounced; show a clear focal symptomatology, which is determined by the localization of the focus of the beating (oculomotor disorders, limb paresis, sensitivity disorders, etc.). Within 2-5 weeks, the neurological symptoms gradually regress, but residual symptoms are often found.

Severe homicide is accompanied by loss of consciousness (the duration is usually described as days, sometimes weeks), psychomotor excitement, pronounced, often threatening disorders of vital functions, gross neurological symptoms, which are dominated by signs of damage to the brain stem.

Meningeal symptoms are sharply expressed, generalized or focal epinaives often occur. Focal symptoms regress slowly and not completely, gross residual phenomena are characteristic primarily from the motor and mental sphere.

The basis of diagnosis is brain CT.

Diffuse axonal damage

Primary lesion as a result of TBI with rotational acceleration/deceleration. In severe forms, hemorrhagic foci are observed in the corpus callosum and the dorsolateral rostral part of the brainstem with microscopic signs of diffuse axonal damage (axonal retraction balls, microglial stars, and white matter degeneration). It is often the cause of long-term loss of consciousness in patients who instantly fell into a coma after a traumatic brain injury , in the absence of a volume formation on CT (although DAU can exist with/without a subdural or epidural hematoma). It can be diagnosed clinically when loss of consciousness (coma) lasts more than 6 hours in the absence of an intracranial mass or ischemia.

DAU step	Description
light	coma > 6–24 h followed by mild/moderate memory impairment and functional impairment.
moderate	coma > 24 h followed by stupor and long-term amnesia. Mild/moderate impairment of memory, behavior and mental function.
difficult	Coma lasting months, flexor and extensor motor responses. Violation of functions: cognitive, memory, speech, sensorimotor. Personality disorders. Dysautonomia.

Epidural hematoma

A fracture of the temporal and parietal bones leads to a rupture of the middle meningeal artery at the point of its exit from the bone groove and entry into the skull cavity (pterion). As a result, arterial bleeding occurs, which gradually peels off the dura mater. .

Clinic

-short post-traumatic loss of consciousness

-"light interval" for several hours

-stunning, contralateral hemiparesis, ipsilateral pupil dilation (result of mass effect)

-headache, vomiting, convulsions (can be unilateral), hemihyperreflexia, unilateral Babinski symptom, increased heart rate pressure, bradycardia.

Displacement of the brain stem in the opposite direction from the hematoma can cause compression of the brain peduncle in the tentorial groove and ipsilateral hemiparesis (Kernogan's phenomenon).

Diagnosis:

CT scan of the brain

Highly dense biconvex (lenticular) hematoma adjacent to the skull. In 11% it is moon-shaped. May cross the sickle, but is usually limited to the sutures of the skull. Usually has a uniform density, sharply delineated edges on several sections, high absorption (undiluted blood), adjacent to the inner surface of the cranial vault, and usually limited to a small segment of the skull.

Treatment

Conservative

- Subacute or chronic epidural hematomas (maximum thickness ≤ 1 spinal cord) with minimal neurological manifestations (mild lethargy, headache) without signs of impingement.

Indications for surgical intervention:

1. The volume of EDH > 30 of the spinal cord3 regardless of SHKG.

2. EDH with the following characteristics can be managed conservatively with repeated CT scans and intensive supervision in the neurosurgical department.

a) volume < 30 spinal cord3

b) thickness < 15 mm

c) dislocation of midline structures (DSS) < 5 mm

d) SHKG > 8

e) absence of focal neurological deficit.

Acute subdural hematoma

Reasons

1. Accumulation of blood around the lesion of the parenchyma (usually the frontal or temporal lobe).

As a rule, there is no "light gap".

Focal symptoms are observed later and are less pronounced than with EDH.

2. Rupture of surface and bridge vessels from sudden acceleration/deceleration of head movement. More frequent "clear gap".

Diagnostics

CT is a moon-shaped formation of increased density

Localization

Above the dorsal surface of the brain.

● Interhemispheric

● Lines the tent

● in the posterior cranial fossa

Indications for surgical intervention

1. thickness > 10 mm or dislocation of midline structures > 5 mm (on CT scan) regardless

of SKG.

2. thickness < 10 mm and dislocation of middle structures < 5 mm, if:

a) reduction of SKG by ≥ 2 points from injury to hospitalization

b) and/or the pupils are asymmetrical or fixed and dilated

c) and/or ICP > 20 mm Hg. Art.

3. monitor ICP in all patients with acute SHD and GFR < 9.

Chronic subdural hematoma

Occurs in the elderly (at an average of 63 years)

Risk factors: alcohol abuse, convulsions, heart rate shunt, coagulopathy, patients at risk of falls.

Traditionally, it contains a dark, "machine oil"-colored liquid that does not coagulate.

It is formed from sharp. Blood within the subdural space causes an inflammatory response. Within days, fibroblasts penetrate the blood clot and form neomembranes on the inner (cortical) and outer (dural) surface. This is followed by the growth of neocapillaries, enzymatic fibrinolysis and liquefaction of the blood clot. Fibrin breakdown products are incorporated into new clots and inhibit hemostasis.

Clinic:

-headache

-stun

-difficulties in speech

- TIA-like symptoms

Treatment

1. Prevention of seizures
2. Reversal of coagulopathy
3. Surgical treatment

Indication:

- Presence of symptoms (thickness of the hematoma > 1 spinal cord): focal deficit, changes in mental state, convulsions.
- Progressive enlargement of the hematoma on CT or MRI.

Methods:

- Hematoma evacuation
- Endovascular embolization of the middle meningeal artery.

Clinical signs of an open craniocerebral injury :

a) visualization of brain tissue or dura mater in the wound;

b) damage to the soft tissues of the vault of the skull, which are accompanied by damage to the aponeurotic helmet (galea aponeurotica), while between the edges of the wound, a diastasis is arbitrarily maintained - the wound is gaping, in the depth you can see the surface of the bone; damage to the aponeurotic helmet is sufficient for infection of the skull cavity due to the existence of a system of outlet veins (venae emissariae: v. emissaria parietalis, v. emissaria mastoidea, v. emissaria condylaris, v. emissaria occipitalis) and spongy veins (venae diploicae: v. diploica frontalis, v. diploica temporalis anterior, v. diploica temporalis posterior, v. diploica occipitalis), which are connected to each other and to the venous sinuses; thus, there are anastomoses between the pool of the internal and external jugular veins; this means that the direction of venous blood movement depends on the difference in pressure in the external jugular vein and inside the skull, therefore, the direction of blood movement in the specified anastomoses can change many times, which causes the possibility of the sleeping brain to occlude infected liquid components from the depth of the wound into the system of cerebral sinuses, i.e. inside skulls;

c) fractures of the bones of the base of the skull containing air sinuses; at the same time, nasal (fracture of the body of the sphenoid bone, ethmoid bone) or ear (fracture of the stony part of the temporal bone) cerebrospinal fluid is detected. In the case of a fracture of the base of the skull, hemorrhage often occurs in the periorbital tissue, under the skin of the eyelids, and subconjunctivally, which is manifested by the characteristic "glasses symptom". Bilateral bruises (bruises) in the medial parts of the orbit ("raccoon eyes") are a sign of a fracture in the anterior cranial dimples A bruise or hematoma over the mastoid process is a sign of a possible fracture of the pyramid of the temporal bone, or Bethel's symptom. In the presence of nasorrhea (flow of liquid from the nose), it is necessary to determine whether this liquid is cerebrospinal fluid. For this, the symptom of a nasal handkerchief is used: if the leaking liquid is a nasal secretion, the handkerchief moistened with it becomes stiff after drying, if the liquid is liquor - the tissue density of the handkerchief practically does not change after drying. The nasal secretion contains significantly more proteins and proteoglycans than the cerebrospinal fluid.

Depending on the damage to the dura, open TBI is divided into penetrating (dura is damaged) and non-penetrating (integrity of the dura is preserved).

In the case of a fracture of the bones of the base of the skull with liquefaction, the injury is automatically considered open penetrating, since the dura mater at the base of the skull is tightly fused with the bones; therefore, a bone fracture means a rupture of the dura mater.

Diagnostics

CT scan of the brain.

Surgical treatment of a depressed skull fracture:

Indications for surgery

1. Open (complex) fractures:

a) surgery is recommended for fractures with a depression that exceeds the thickness of the roof of the skull (calvaria) and those that do not meet the criteria for non-surgical treatment listed below;

b) conservative treatment is considered in cases of:

● lack of evidence (clinical or CT) of penetrating craniocerebral trauma (CNS leak, intradural pneumocephalus on CT...);

● and the absence of significant intracranial hematoma

● and compression of less than 1 spinal cord

● and the absence of damage to the sinuses of the frontal bone;

● and absence of wound infection or marked contamination

● and the absence of pronounced spinal deformity

2. Closed (simple) fractures with depression can be treated both surgically and conservatively.

Technique of craniotomy

- Position: (depends on the location of the fracture).

- Postoperative period: in the intensive care unit.

- Procedure: surgical intervention in the area of the skull fracture, restoration of the "cover" of the brain, removal of foreign material and damaged brain tissue (i.e. dead brain tissue), removal of any hemorrhage and stopping of any identified bleeding, possible installation of intracranial pressure monitoring. Position: (depends on the location of the skull fracture).

Spinal cord injury with spinal cord injury is considered one of the most severe injuries of the human body, as it causes a large spinal cord stiffness, which in some forms of damage is 60-70% and leads to severe disability of the victims (most of them become disabled of the I and II groups). The cost of treatment and maintenance of one patient with a spine and spinal cord injury in the USA is estimated at two million dollars.

Every year in Ukraine, more than 2,000 people receive a spinal cord injury (SCI). They are mostly young people. Fractures of the spine with damage to the spinal cord and/or its roots

occur with direct application of mechanical force (direct injuries), falling from a height on the feet or head, as well as with excessive bending or extension of the spine (indirect injuries).

There are open injuries of the spine and spinal cord with a violation of the integrity of the skin and closed (without a violation of the integrity of the skin). In case of damage to the dura mater, open penetrating injuries occur. Closed injuries of the spinal cord are divided into concussion, contusion and compression of the spinal cord.

Concussions of the spinal cord are characterized by reversible disorders of the functions of the spinal cord , which after treatment completely disappear within the first five to seven days. Clinically, it manifests itself as segmental (in the form of muscle weakness) or sensory disorders in the area of the injured spinal cord segments. Sometimes there may be conductive disorders in the form of delayed urination, a decrease in strength in the departments distal to the level of injury, or conductive sensory disorders in the form of hypoesthesia, etc. Thus, a concussion can manifest as a partial disruption of the functions of the spinal cord .

Spinal cord injury is characterized by both reversible functional and irreversible morphological changes in the form of ischemic or hemorrhagic contusion zones. Morphological changes in the case of spinal cord injury can be primary and secondary as a result of impaired blood and liquid circulation, which often leads to a morphological interruption of the spinal cord. Clinically, this manifests as a syndrome of partial or complete disruption of spinal cord conduction. In the case of slaughter, the neurological symptoms are stable and, as a rule, do not fully regress. The dynamics of neurological symptoms is very important.

Compression of the spinal cord can be caused by bone fragments of vertebrae, damage to ligaments and discs, hemorrhages (hematomas), edema, and combinations of these causes. It can be posterior, due to a fracture of the bracket, damage to the articular processes, anterior, due to a fracture of the body, fragments of the damaged disc, thickened by the posterior longitudinal ligament), and internal (intracerebral hematoma), detritus, edema. After a traumatic hematoma, these and subdural hematomas (hydromas) can have any localization. Spinal cord compression is often caused by several reasons, clinically it is characterized by a syndrome of partial or complete disruption of spinal cord conduction.

Open injuries of the spine and spinal cord , as well as closed injuries, are divided by the level of injury into injuries of the cervical, thoracic and lumbosacral divisions of the spinal cord , as well as the roots of the horse's tail.

In addition, fire and non-fire injuries are distinguished. In relation to the wound channel to the spine and spinal cord, the following injuries are distinguished:

- Through (crossing the spinal canals);
- Blind (end in the spinal canal);
- Tangential (the wound canal passes touching one of the walls of the spinal canal, destroys them, but does not penetrate into the canal);
- Non-penetrating (the wound channel passes through the bone structures of the spine without damaging the walls of the spinal canal);
- Paravertebral (the wound canal passes near the spine without damaging it).

In case of damage to the dura mater, penetrating injuries occur, without damage to the dura mater, non-penetrating injuries. The nature of the injury is established during clinical and instrumental examinations, as well as surgical intervention.

Damage to the spine is divided into stable and unstable.

Unstable injuries are due to the rupture of the posterior ligaments, the presence of fractures and dislocations of the vertebrae, in which repeated displacements of the vertebrae with additional damage to the spinal cord and its roots are possible.

Unstable injuries of the spine include all displacements (dislocations) of vertebrae, fractures and dislocations of articular processes, ruptures of intervertebral discs and their connections with damage to vertebral bodies. All patients with spinal instability need medical stabilization, splints, stitches, surgical methods).

Stable injuries of the spine most often occur with wedge-shaped compression fractures of the bodies and with fractures of the brackets of the proximal 4th lumbar vertebra, as well as with fractures of the transverse and spinous processes. Finally, the instability of the fracture is established using additional examination methods: functional spondylography, computerized tomography (CT) and (or) magnetic resonance imaging (MRI).

The greatest role in the pathogenesis of spinal disorders is played by vascular post-traumatic disorders, which further develop according to the ischemic, less often - hemorrhagic type. Even a slight compression of the spinal cord causes disruption of cerebral blood circulation. Hypoxic-ischemic phenomena increase in adjacent segments under these conditions.

It is worth remembering that nerve cells located in the immediate vicinity of the focus of damage are in a state of inhibition, that is, in a functionally depressed state. Long-term functional impairment of the neural apparatus of the spinal cord leads to the development of organic changes.

Damage to the spine and spinal cord can be multiple (injuries in two different parts of the spinal column) and combined (in the case of damage to other organs and systems). With a fracture of the body, arch and (or) spinous processes, vertebral fractures are interpreted as multiple.

Closed injuries of the spine include:

1. Damage to the spine without impairment of the functions of the spinal cord or its roots (not complicated).
2. Damage to the spine with impaired functions of the spinal cord and its roots (complicated).
3. Damage to the spinal cord can be without traumatic changes on the part of the vertebral column (concussion, contusion, hemorrhage).

Nature of spinal cord injury :

1. Damage to the ligamentous apparatus.
2. Damage to the bodies of the vertebrae (cracks, compression, fracture, transverse, longitudinal, explosive, separation of the locking plates).
3. Fractures of the posterior semiring of vertebrae (braces, spinous, transverse, articular processes).
4. Fractures and dislocations with damage to the ligamentous-articular complex.
5. Fractures of bodies and brackets with or without displacement.

According to the localization of damage:

- cervical
- chest
- lumbar
- lumbar-sacral sections of the spine
- damage to the roots of the horse's tail.

The frequency of spine injuries depends on the anatomical and physiological characteristics of the spine, ligaments and its mobility. V, VI and VII vertebrae in the cervical, XI and XII in the thoracic and I and V in the lumbar regions are most often damaged. Accordingly, the spinal cord is also damaged at these levels . Children often (18-20%) have spinal cord injuries without X-ray changes in the bone apparatus.

Depending on the degree of spinal cord conduction disturbance , a distinction is made between complete and partial damage to the spinal cord .

Signs of a spinal cord injury are: local pain and deformation, reduced or absent sensitivity below the level of injury, movement disorders in the upper and lower limbs (in case of injury to the lower part), in the lower limbs (in case of injury to the thoracic and lumbar region), disorders of the functions of the pelvic organs. Pathological changes in spinal cord injury are caused by mechanical (immediately after the injury) causes. Circulatory disorders occur immediately after

the injury and often progress, ischemia is more pronounced than hemorrhage and secondary compression injuries.

The assessment of the functional state of patients with spinal cord injury should be carried out according to Frankel:

Group A - patients with anesthesia and plegia below the level of injury.

Group B – patients with incomplete sensory impairment below the level of injury, no movement.

Group C - patients with complete sensory impairment, there are weak movements, but the muscle strength is insufficient for walking.

Group D - patients with incomplete sensory impairment below the level of injury, there is movement, muscle strength is sufficient for walking with external assistance.

Group E - patients without sensory and motor disorders below the level of injury.

Today, the problem of providing emergency and specialized medical care to victims with spinal cord injury in Ukraine has not been organized.

A large number of victims are detained in district and district hospitals, where soon after the injury they develop trophic disorders, infectious and inflammatory complications. Because of this, operations must be postponed, which leads to negative consequences.

The main task of personnel providing assistance in the event of an injury is to prevent further injuries, prevent secondary changes caused by compression and ischemia of the SPINAL BRAIN, and maintain optimal conditions for its functioning.

Treatment of patients with spinal cord injury begins at the pre-hospital stage.

The main task before the hospital stage is not to worsen the patient's condition during his transportation to the hospital. Prehospital care includes preservation or normalization of vital functions (breathing, hemodynamics); fixation of the spine, introduction of neuroprotectors methylprednisolone. Patients with spinal cord injury are recommended to be transported directly to specialized neurosurgical departments or trauma departments of the Central Hospital.

The patient must be examined by a neurosurgeon. They conduct: spondylography, CT or MRI of the spine.

All patients with spinal cord injury who have not undergone CT or MRI are prescribed a spinal puncture, during which cerebrospinal fluid pressure is measured, as well as fluid dynamics tests are performed to detect the patency of the subarachnoid spaces. Violations of the patency of the latter indicate compression of the spinal cord . In this case, it is necessary to eliminate the compression immediately. With an injury of the cervical spinal cord, fluid-dynamic tests have relative values, because the patency of the subarachnoid spaces is often preserved when the brain is compressed posteriorly or anteriorly. If possible, a CT scan or MRI is performed.

Treatment of a patient with a spinal cord injury is incredibly complex, long-term, and is carried out directly during the examination of the patient. In the case of spinal shock, atropine, dopamine, saline solutions (3-7% sodium chloride solution), reopoliglukin, hemodeze are administered, and the lower limbs are bandaged.

The conducted examinations indicate the need to introduce large doses (30 mg/kg per body weight)

Methylprednisolone (solumedrol) intravenously in the first 8 hours - another 5.0 mg/kg every 4 hours for 48 hours. Methylprednisolone acts on the damaged spinal cord as follows:

- Inhibits lipid peroxidation caused by free radicals;
- Inhibits lipid hydrolysis;
- Supports blood supply to spinal cord tissues and aerobic energy metabolism;
- Improves the introduction of calcium from cells (prolongs the activation of neutral proteases – supports the integrity of neurofilaments;
- Strengthens the excitability of neurons and conduction of impulses.

Methylprednisolone as an inhibitor of lipid peroxidation is much more effective than conventional prednisolone or dexamethasone. Saluretics are used along with hypertonic NaCl

solution to relieve cerebral edema. Vitamin E is used as an antioxidant (5 ml IV 2-3 times a day). Diphenin, seduxen, relanium are prescribed to increase the brain's resistance to hypoxia.

In the case of an injury to the cervical spine, a bone extraction is immediately carried out behind the parietal tubercles or halloapparatus are applied, which (in up to 80% of cases) reduce compression of the spinal cord , or a closed fracture-dislocation exercise is carried out.

Data from the literature and our observations indicate that in the case of compression of the spinal cord , its elimination should be carried out as early as possible. Early decompression of the brain is a prerequisite for the successful treatment of patients with spinal cord injury .

Surgical interventions for spinal cord injury are indicated in 68% of cases. The operation of decompression of the brain ends with stabilization of the spine with anterior or posterior spondylodesis. In the future, if necessary, a reconstructive stabilizing operation can be performed in the appropriate centers.

Contraindications to the operation are shock, combined with injuries that must be energetically and quickly eliminated.

The following indications for operations should be followed:

1. Deformation of the spinal canal , established during an X-ray examination, CT scan or MRI, which indicate compression of the spinal cord or narrowing of the spinal canal by 30% or more.

2. The presence of bone or muscle-tissue fragments in the spinal canal .

3. Partial or complete blockage of the CSF channels.

4. Progression of secondary respiratory failure as a result of ascending linear swelling of the spinal cord .

5. Clinical and angiographic data indicating compression of the main vessels of the brain (anterior artery syndrome, complications of venous outflow).

6. Instability of the vertebral-motor segment, which threatens the growth of neurological symptoms.

spinal cord decompression method depends on the nature of the spinal cord injury and the level of brain compression. Early decompression of the spinal cord is mandatory. It can be achieved by repositioning, corpectomy, laminectomy (lateral, ventral-lateral, ventral), depending on the nature of the spinal cord injury. It should be noted that decompression must be complete. The operation must be completed with revision of the spinal cord and duraplasty.

An interbody, interspinous, or interarch spondylodesis must be performed.

Refusal of early operations in the case of brain compression is harmful, unacceptable for the patient, because it leads to multiple organ failure: bedsores, infectious-inflammatory processes on the part of the genitourinary and respiratory systems appear early. Because of this, surgical intervention is postponed, the duration of the patient's treatment increases, and the rehabilitation period is delayed.

In the case of an injury to the cervical spinal cord due to its compression, ischemia and swelling of the brain increases, which leads to the most widespread swelling of the spinal cord and medulla oblongata, as well as breathing disorders.

Adequate elimination of compression of the spinal cord and deformation of the spine in the early stages after the injury, reliable internal fixation of the damaged spinal segment, effective prevention of bedsores and urological complications ensure a favorable course of the disease. In the future, comprehensive rehabilitation of such victims should be carried out in a timely manner.

Examination algorithm and sequence of assistance in the acute period of spinal cord injury.

I. 1) hospitalization in the neurosurgical department;

2) assessment of the condition according to Frankel;

3) general analysis of blood and urine, biochemical analysis of blood, determination of blood group and Rh factor;

4) spondylography;

5) lumbar puncture for examination of fluid dynamics, myelography;

6) if possible, perform MRI or CT. Consultations of a therapist, urologist.

II. normalization of breathing and hemodynamics, catheterization of the urinary bladder, central vein. In the case of spinal shock, lower limbs are bandaged, atropine is administered, and hypertonic (3-7%) NaCl solution is administered. In the presence of spinal cord compression, early decompressive and stabilizing surgery is indicated (in the first 6-12 years). in the first 8 h - methylprednisolone at a dose of 30 mg/kg/body weight once, after 2-4 h - 15 mg/kg, in the future 5 mg/kg every 4 h not for 2 days. Vitamin E - 5 ml IV, difenin - 500 (relanium, seduxen, sodium thiopental, sibazone), nimotop, broad-spectrum antibiotics, returning the patient every 30-40 minutes, analgesics, neuroprotectors, magnesium sulfate, piracetam 10 -12 g/day, cerebralisin - 15-25 ml IV in 200 ml isotonic NaCl solution, symptomatic treatment. After 8-12 weeks, he is transferred to the neurological department, rehabilitation centers for rehabilitation.

Combined spinal cord injury.

Hospitalization in the neurosurgical department, evaluation of the state according to Frankel. General analysis of blood and urine, biochemical analysis of blood, determination of its group, rhesus factor, spondylography, lumbar puncture for examination of fluid dynamics, myelography, if possible, MRI or CT. Consultations of a therapist, urologist. Clarification of the nature and localization of the extravertebral component.

In case of hypovolemic shock – anti-shock therapy. Emergency operations for hemopneumothorax, damage to internal organs. Normalization of breathing, hemodynamics, catheterization of the urinary bladder, central vein. In case of spinal shock, bandage the lower limbs, administer atropine, hypertonic (3-7%) NaCl solution. In the presence of compression of the spinal cord - early decompression-stabilizing surgery. In the first 8 years - methylprednisolone at a dose of 30 mg/kg once, after 2-4 years - 15 mg/kg, then 5 mg/kg every 4 years for 2 days. Vitamin E - 5 ml intravenously, difenin - 500 (relanium, seduxen, sodium thiopental, sibazone), nimotop, broad-spectrum antibiotics, returning the patient every 30-40 minutes, analgesics, neuroprotectors, magnesium sulfate, piracetam, Yumex Cerebralizin - 15-25 ml IV in 200 ml isotonic sodium chloride solution, protecting the spinal cord and brain from secondary post-traumatic changes, symptomatic treatment. In case of shock – anti-shock therapy, then decompressive-stabilizing surgery. As early as possible - decompression of the spinal cord and stabilization of the spine. Early rehabilitation in the rehabilitation center. Patients with spinal cord injury have a significant risk (up to 50%) of thromboembolic complications (deep leg vein thrombosis and pulmonary embolism). The prevention of these complications is bandaging of the lower limbs, massage, activation of the victims, injection of 0.3 ml of fraxiporin per day into the subcutaneous tissue of the anterolateral surface of the abdomen for 7 days, from the 6th day it is necessary to switch to tiklid - 1 tablet 2 times a day during meals for 2-3 months. With prolonged bed rest, prevention of thromboembolism is carried out at all stages of traumatic spinal cord disease .

Surgical tactics in the treatment of chronic complicated spine injury in the long term after the injury.

After a severe spinal cord injury, about 80% of victims remain alive. These patients at all stages of traumatic spinal cord disease need medical assistance. In the remote period of a spinal cord injury, changes are observed not only at the site of the injury, but also at a distance from it. Contrast x-ray, radionuclide, and immunological examinations, as well as MRI, are used to diagnose the level, degree, and length of damage to the spine and spinal cord in the long term after the injury. These examinations should be carried out in the dynamics of the course of a traumatic disease of the spinal cord .

After the diagnostic examination, the stages of treatment of spinal patients are developed. Thus, a visible violation of the cardiovascular, respiratory, immune and urinary systems, the digestive tract, requires appropriate correction, this can be identified as the 1st stage of treatment

methods. At this stage, it is necessary to prepare patients for reconstructive operations on the spine and spinal cord, if they are indicated. The indication for the operation is the deformation of the spinal canal and compression of the spinal cord , which interfere with the implementation of further rehabilitation methods. As the 2nd stage of appropriate treatment in the remote period of the injury, according to the indicators, it is worth carrying out types of operative interventions decompressive-stabilizing, on the spine and, accordingly, reconstructive on the spinal cord . Repeated trauma, which necessarily occurs during surgery, will lead to edema, hypoxia and impaired blood circulation of nervous structures. All methods aimed at reducing and eliminating these negative phenomena make up 3 stages of treatment. Not all patients recover their impaired functions after the 3rd stage of treatment, so such patients require long-term treatment (pharmacological, functional and physiotherapeutic, prosthetic and orthopedic care). All these methods make up 4 stages of treatment.

At the 1st stage, the general condition of spinal patients is assessed (function of the cardiovascular, respiratory, urinary, and other systems). Patients with inflammatory processes in the respiratory tract are prescribed detoxification and anti-inflammatory therapy, broad-spectrum antibiotics. In the inflammatory process of the urinary system, the species composition and dynamics of urine microflora, the sensitivity of microorganisms to antibiotics are determined. Taking into account the antibiotic profile, appropriate antibiotics are used. Patients need to wash the bladder with disinfecting solutions (bifuran, furacilin) 1-2 times a day, at the end of the second wash for 30-40 minutes. a solution of colargol (protargol) is injected into the bladder.

At this stage, prevention and treatment of trophic disorders of the skin (bedsores, etc.) are carried out. patients require constant care (changing the position of the body every 2-3 hours, wiping the skin with camphor alcohol, performing a light massage). Widely used special beds with rotating frames, special mattresses, soft pads, the best of millet).

Complex treatment includes the use of detoxification, dehydration, anti-inflammatory, general strengthening therapy, as well as drugs that improve blood and lymph circulation. They carry out ultraviolet irradiation of bedsores with suberythemic doses (1-3 biodoses) and UHF in a weak thermal dose. Local and general laser therapy and electrical stimulation of bedsores are widely used in order to accelerate the preparation of wound surfaces for the next surgical treatment.

Para-articular ossification in the area of pelvic and knee joints significantly hinders further rehabilitation of spinal patients. In the rehabilitation process, the following goal is set: if the patient has a partial impairment of the functions of the spinal cord and there is still hope for the restoration of movements in the lower limbs, then joint mobilization is used so that the patient has the opportunity to move independently, move around in a wheelchair (that is, sit). The system of treatment of paraarticular ossification includes resection of heterotopic ossification and arthroplasty.

The state of the immune system is important for further rehabilitation. Cellular and humoral immunity is most reduced in the 2-3rd month after the injury. Because of this, when plastic surgery is performed on the spine and spinal cord , during this period, spinal patients find themselves in rather difficult conditions. It is not advisable to perform the operation 2-3 months after the injury, because bad results are possible.

In the presence of purulent complications of a toxic-septic state, T-activin (1 ml each; 0.1% solution subcutaneously or intramuscularly every other day, total dose - 500 μg) is used to eliminate secondary immunodeficiency in combination with immunoglobulin (25 ml each drip with with an interval of 24 and 48 h.). only 75 ml per course of treatment.

The use of laser irradiation of blood and cerebrospinal fluid has a positive effect on the course of the disease. Already after 3-4 treatment sessions, there is an improvement in the general condition, appetite, regeneration and cleaning of bedsores, and a decrease in body temperature.

In the case of stabilization of the general condition of the patient (that is, when correcting disorders of the respiratory, cardiovascular, urinary and immune systems, digestive tract), elimination of trophic disorders of the skin and mobilization of large joints of the lower extremities, it is necessary to proceed to the 2nd stage of treatment - reconstruction of the spinal canal.

In the case of compression of the spinal cord by anterior structures (body of vertebrae and discs) in the cervical spine and blockage of the subarachnoid space, which is determined by X-ray radionuclide studies on MRI, perform anterior decompression of the spinal cord followed by corporedesis with a bone graft.

In the case of compression of the spinal cord in the cervical region, a decompressive laminectomy is performed with the posterior elements.

Depending on the nature of spinal cord compression, anterior, posterior, or total decompression is performed in the thoracic region.

In the early postoperative period, spinal patients are corrected for disorders of the cardiovascular, respiratory, immune, and urinary systems, digestive tract, metabolic disorders, and water-electrolyte composition.

of the spinal cord is an essential need during this period . It is important to preserve the functioning of those structures that have been damaged, but can still restore their function. It is necessary to use antispasmodics, anticoagulants, anti-edema therapy, nootropin, cerebrolysin 15-25 ml IV for 20-25 days, antihypoxants, antioxidants.

Not all patients recover lost functions after 3 stages of treatment. Therefore, in the future, long-term medical and physiotherapeutic treatment is carried out, as well as prosthetic and orthopedic assistance is provided. All these methods make up the 4th stage. Prescribe drugs and GABA-ergic substances (nootropil, cerebrolysin - 15-25 ml IV each), anticholinesterase drugs (galantamine, proserin, etc.), activators of bioenergetic metabolism (ATP riboxin, cocarboxylase, group B vitamins), anabolic hormones (retabolil, nerobol), tissue therapy (aloe extract, plasmol, vitreous body).

Cavinton, listenone, actovegin, cinnarizine, eufilin, papaverine, ticlid, trental, etc. are prescribed from vascular drugs. To reduce painful and spastic syndromes, ganomeoblockers, prostaglandin inhibitors, midokalm, and sodium oxybutyrate are used.

Normalization of functions is important in the rehabilitation of spinal patients

pelvic organs, establishment of controlled acts of urination and defecation.

In the remote period of a spinal cord injury, it is mandatory to take measures to prevent thromboembolic complications (massage of the patient's activation, fraxiparin, tiklid, etc.).

Medical and social examination.

With a concussion of the spinal cord, the treatment lasts, on average, up to 3 weeks. The total duration of temporary disability is up to 2-3 months, taking into account the fact that the ligamentous apparatus may have been damaged during a spinal cord concussion at the time of injury.

Light work (up to 1 year) is recommended for people engaged in physical labor. In some cases, they are transferred to 3 gr. disability (up to 1 year).

A contusion of the spinal cord is often accompanied by a fracture of the bony skeleton (body, arch, articular processes). Expert assessment depends on the degree and nature of bone damage and spinal cord injury. Vertebral body fractures combined with articular process fractures are unstable. In this case, the term of temporary incapacity is up to 7 months, which is necessary for the consolidation of the cancellous bone.

Mild contusion of the spinal cord: duration of inpatient treatment - up to 1 month, after discharge - outpatient treatment of spinal cord functions. Sometimes the patient is transferred to 3 gr. disability (up to 1 year). The terms of restoration of lost functions - 3-4 weeks.

Contusion of the spinal cord of medium degree: the duration of inpatient treatment is 1.5-2 months. In the case of a favorable clinical course, outpatient treatment according to a letter

of incapacity for work for up to 8 months. Then the appointment of 3 gr. disability, less than 2 gr. The term of restoration of lost functions: the beginning - from 3-4 weeks, the duration - a month, a year.

Severe contusion of the spinal cord: duration of inpatient treatment up to 3-4 months. The start of restoration of the broken functions - from 3-4 months, but continues for years, full restoration does not always occur. After inpatient treatment, 1 gr. disability, initially for 1 year before clarifying the prognosis of the course of the traumatic disease. In the future, the disability of these patients can be extended up to 3 years, and only after 5 years of observation and establishment of a hopeless recovery of the impaired functions, permanent disability can be recommended.

With hematomyelia, the clinical and labor prognosis is unfavorable, because of this temporary disability is extended up to 4 months. These patients are often prescribed 1 and 2 gr. disability for up to 1 year. Then, after clarifying the clinical prognosis, these terms are extended to the disability group or assigned indefinitely.

With hemorrhages in the spinal canal with adhesions in the horse's tail, the temporary incapacity is extended to 4-6 months, in the future, 3 gr. is often prescribed. disability due to residual effects, less often - 2 gr. (with arachnoiditis, cauditis or deep paresis with pain syndrome). Dynamic observation and treatment are carried out for 3-5 years, after which the disability group can be established indefinitely.

All victims with a spinal cord injury need medical and social rehabilitation. Early medical rehabilitation begins in the acute period of the injury in a hospital, where urgent surgery and treatment are carried out, aimed mainly at complications (bedsores, contractures, urinary fistulas, etc.). In the acute stage of a traumatic disease, patients are transferred to a special rehabilitation department - the 2nd stage lasts 1-2 months.

After that, the victims are sent for sanatorium-resort treatment in specialized sanatoriums (Saki, Slovyansk, Donetsk region, Solony liman, Dnipropetrovsk region, etc.).

The 4th stage of rehabilitation is employment. Patients are given the opportunity to work in specially created conditions.

Peripheral nerve injuries are divided into open and closed. The first include: cut, chopped, punctured, torn, bruised, crushed wounds; to closed ones - concussion, contusion, compression, stretching, rupture and dislocation. 3 morphological points of view, distinguish between complete and partial anatomical rupture of the peripheral nerve.

Damage to the nerve is manifested by a complete or partial block of conduction, which leads to varying degrees of impaired motor, sensory, and autonomic function of the nerve.

With partial nerve damage, symptoms of irritation in the area of sensitivity and vegetative reactions (hyperpathy, causalgia, hyperkeratosis) appear.

Neuropraxia (praxis - work, apraxia - inability, inactivity) - temporary loss of physiological function - nerve conduction after a slight injury **.** Anatomical changes mainly from the side of myelin sheaths. Clinically, mainly movement disorders are observed. 3 sides of sensitivity are primarily marked by paresthesias. Vegetative disorders are absent or not pronounced. Recovery occurs within a few days. This form corresponds to a concussion (according to Doynikov).

Axonotmesis is a more complex form of injury due to compression or stretching. Anatomical continuity of the nerve is preserved, but morphologically Wallerian degeneration occurs in more detail at the site of injury.

Neuropraxia and axonotmesis are treated conservatively.

Neurotmesis means a complete interruption of a nerve or a severe injury with the rupture of some of its nerve trunks, as a result of which regeneration is impossible without surgical intervention.

The process of breakdown of nerve fibers, described in 1850 by the French scientist Waller, is now denoted by the term - Wallerian degeneration. The reverse process - regeneration of the nerve takes place under the condition of exact alignment of bundles (respectively - sensitive and motor) of both segments of the nerve, proceeds rather slowly (at a rate of approximately 1 mm per day).

The clinical and electrophysiological picture of peripheral nerve damage depends significantly on the time that has passed since the injury. 3 considering the peculiarities of the course of the Wallerian degeneration process, it is advisable to divide this interval into two periods: acute and remote.

The acute period of trauma is a period in which the decisive importance in the clinical picture is not so much the manifestations of nerve damage as all the factors of the trauma as a whole: shock reaction to pain, blood loss, the presence of secondary infection, mental trauma, etc. The acute period lasts 15-20 days, during this time, even after a complete rupture, the distal segment retains the ability to conduct, therefore, the results of most electrophysiological examination methods in the acute period are uninformative.

The remote period of the injury is characterized by the formation of the main pathomorphological changes in the nerve fibers caused by Wallerian degeneration, starting from the third to fourth week after the injury.

In the acute period of the injury, the most informative sign of nerve damage is: impaired sensitivity in the innervation zone.

The best treatment results are achieved when adequate surgical treatment is performed on the day of the injury. However, the operation is possible only if certain conditions are met: the presence of trained specialists, the necessary equipment, including microsurgical instruments, suture material and magnifying optics, adequate anesthetic support and the absence of complications from the wound and the patient's somatic condition.

Conducting operations on the nerve in the absence of the listed conditions mostly leads to unsatisfactory results.

Therefore, in institutions of a general surgical profile, for peripheral nerve injuries, it is enough to stop the bleeding, carry out anti-infective measures and apply stitches to the wound, followed by the patient's referral to the microsurgery department.

The diagnosis of nerve damage is based on general clinical data and the results of an electrophysiological study.

The location of the injury to the limb in the presence of neurological symptoms makes it possible to suspect damage to the peripheral nerve.

Diagnostics.

Anamnesis to a large extent makes it possible to specify the nature and mechanism of nerve damage. Examination of the injured limb, localization of the wound allow us to conclude which of the nerves is damaged and specify the extent of this damage.

The main function of the nerve is conduction. Nerve damage is manifested by a syndrome of complete or partial disruption of its functions. The degree of its loss is determined by the symptoms of loss of movement, sensitivity and vegetative function of the nerve.

Movement disorders with complete damage to trunk nerves and limbs are manifested by a picture of peripheral muscle paralysis (atonia, areflexia, atrophy. However, when evaluating motor disorders, one should take into account the possibility of compensatory strengthening of synergist muscle function, masking manifestations of lost motor functions.

The study of sensitivity is often decisive in the diagnosis of damage to one or another nerve. Anesthesia in the zone of innervation is characteristic for anatomical rupture of the nerve trunk or complete crushing.

Trophic disorders with nerve damage are manifested by sweating disorders (anhidrosis, hypo- or hyperhidrosis), immediately after the injury by hyperthermia in the innervation zone with a subsequent decrease in temperature, changes in hair growth in the form of partial baldness

(hypotrichosis) or increased growth (hypertrichosis), thinning of the skin, the disappearance of folds on it .

Palpation and percussion along the course of the nerve trunk help to clarify the level and type of damage. In the acute period of injury, when nerve fibers are torn, tapping at the level of the injury causes projection pain. In the longer term, palpation can reveal a neuroma of the central segment of the damaged nerve. The appearance of tenderness during palpation and percussion along the course of the peripheral segment of the injured nerve is a characteristic sign of nerve regeneration after its suturing (Tabel's symptom).

Treatment.

The type and degree of nerve damage determines the further tactics of treatment: conservative or surgical.

The main method of treatment of traumatic lesions of peripheral nerves is surgical. 3 purposes of surgical treatment are used:

Neurolysis - release of the nerve from the tissues that surround and cause its compression (hematoma, scars, bone fragments, calluses). The operation is performed by carefully separating the nerve from the surrounding scar tissue, which is then removed, avoiding damage to the epineurium if possible.

Internal neurolysis, or endoneurolysis - the separation of bundles of the nerve trunk from intraneural scars after dissection of the epineurium, performed with the aim of decompressing the bundles and clarifying the nature of nerve fiber damage. To prevent the formation of new joints and scars, the nerve is placed in a new bed prepared from intact tissues, and thorough hemostasis is performed.

Nerve suturing - The indication for nerve suturing is a complete or partial rupture of the nerve with a significant degree of conduction disturbance. There is a distinction between primary nerve suturing, which is performed simultaneously with primary surgical treatment of the wound, and delayed, which is performed 2-4 weeks after wound treatment. An operating microscope, microsurgical instruments, and suture material 6/0-10/0 are required to perform an operation on peripheral nerves at the modern level. When performing epineural suturing, it is necessary to achieve an exact coincidence of the cross sections of the central and peripheral segments of the transected nerve trunk.

In recent decades, with the development of microsurgery, perineural (interfascicular) suturing is also used to connect nerve ends. A combination of these two stitching techniques is possible. Comparison of bundles and application of seams is carried out exclusively under a microscope. The operation is completed by immobilizing the limb with the help of a plaster bandage in a state in which the nerve is subjected to the least tension and pressure. Immobilization is maintained for two to three weeks.

Tasks for self-control :

1. Define neurosurgical tactics and specify the method of treatment for each type of skull fractures:

a) linear fracture;

b) depressed fracture:

- closed
- open

c) multilobed:

- penetrating
- impenetrable

Correct tactics:

a) conservative treatment.

b) - possible conservative tactics depending on the zone;

- operative tactics.

c) operational tactics in all cases.

Materials for the classroom independent preparation:

List of educational practicals tasks:

1) Psychoneurological examination of patients with TBI
2) Plan of examination of a patient with TBI
3) Reading radiographs skull
4) Indications and technique of carrying out lumbar puncture
5) Fill out the appointment letter for a patient with a brain concussion and cerebral infarction of various degrees severity

Instructional material for mastering professional skills

Professional algorithm for examination of a patient with TBI:

1. Examination of the patient.
2. Anamnesis (history of injury, heredity).
3. Complaints (headache, vomiting, visual disturbances, epileptic seizures).
4. Objective examination of internal organs (obligatory blood pressure and pulse on both hands).
5. Study of the degree of consciousness disturbance (Glasgow scale from 3 to 15).
6. Study of 12 cranial pairs nerves
7. Study of the motor-sensitive sphere (reflexes, definition of disorders sensitivity
8. Definition of statics and coordination
9. Definition of meningeal syndrome
10. Local research heads
11. Detection of leakage of liquid, blood from the nose, ears
12. Detection of external soft injuries fabrics
13. Craniogram in two projections.
14. Additional research methods at necessity

Professional algorithm for performing spinal cord Punctures :

1. Position of the patient on the right sides
2. The legs are bent at the knees and pulled up to stomach
3. Treatment of hands with an antiseptic, sterile gloves.
4. Treatment of the field with 5% iodine, then with alcohol at the level of 3-5 lumbar vertebrae
5. Local anesthesia with 0.5% novocaine solution at the level of the intervertebral space between the 4th and 5th lumbar vertebrae
6. Needle control (presence of mandrel, sharpness needles).
7. Lumbar puncture. During the puncture, the feeling of two obstacles (yellow ligament and dura mater) and failure
8. Slowly pulling the mandrel from the needle, checking availability liquor
9. With an extended mandren, evacuation of 1-2 ml of liquor for analysis
10. Removal of the needle is aseptic sticker

Situational tasks:

1) The patient was found unconscious in the yard, smelling of alcohol from his mouth. Locally - swelling of soft tissues in the right temporal region, anisocoria on the right, areflexia. Determine the examination plan, treatment tactics. ANSWER: hospitalization in the NHV, CT scan, trepanation of the skull, removal of intracranial hematomas

2) The patient was being treated for a closed craniocerebral injury - a temporal

fracture bones to the left. 10 hours after receiving the injury, there was a sharp deterioration, coma 4-5 on the Glasgow coma scale developed, respiratory function disorders, periodic tonic convulsions, wide pupils, sluggish photoreaction, the left pupil is slightly wider than the right.

QUESTION: What can cause a change in the clinical picture? Define tactics doctor

ANSWER: With the growth of an epidural hematoma on the left, dislocation of the middle structures of the brain. The doctor's tactics are urgent surgical intervention based on vital signs (trepanation of the skull).

3) The 18-year-old defendant suffered a closed craniocerebral injury at the age of 12. In the following years, periodic headaches, there were two bouts of loss of consciousness. Finished 11th grade, studies in college.

QUESTION: Draw up an examination and treatment plan.

ANSWER: Psycho-neurological examination, EEG CT scan of the brain.

Clinical tasks.

1. During the game, the boy threw a bottle that hit his 16-year-old friend in the side of the head. The victim seemed deaf for 30 seconds, but then recovered completely. But he suddenly fell into a coma. Decreased sensitivity on the opposite side. In 25 minutes, he was taken to the hospital without a reaction to pain. Pulse - 40 beats/min, without arrhythmia. Blood pressure on both arms is 170/110 mm Hg, there is no swelling of the optic nerve discs, but there is venous congestion at the bottom of the fundus. What is the possible diagnosis:

Options:

a) Epileptic attack.

b) Violation of thermal conductivity.

c) Increased intracranial pressure.

d) Weakness syndrome.

e) Formation of hydrocephalus.

2. During the game, the boy threw a bottle that hit his 16-year-old friend in the side of the head. The victim seemed deaf for 30 seconds, but then recovered completely. But he suddenly fell into a coma. Decreased sensitivity on the opposite side. In 25 minutes, he was taken to the hospital without a reaction to pain. Pulse - 40 beats/min, without arrhythmia. Blood pressure on both arms is 170/110 mm Hg, there is no swelling of the optic nerve discs, but there is venous congestion at the bottom of the fundus. What is the fastest treatment for a young person in the next 4 hours:

Options:

a) Craniotomy.

b) Antihypertensive therapy.

c) Setting the rhythm driver.

d) Ventriculoperitoneal shunt.

e) Lack of treatment of epileptic attacks.

3. The patient received a severe brain injury 3 months ago - an open fracture of the right frontal bone. After the initial surgical treatment, the patient's condition improved. In the right frontal area there was defective skin and bone, covered with granulations with a purulent process. What is the best treatment for this patient:

Options:

a) Conservative treatment.

b) Plastic surgery of a skull defect.

c) Lumbar puncture.

d) Dressings with ointments.
e) Partial removal of parts of the brain.

4. The patient was brought to the trauma department in a state of alcohol intoxication and psychomotor agitation. A subcutaneous hemorrhage was detected in the left temporal region with constant leakage of fluid from the left external auditory meatus. A diagnosis of alcohol poisoning was established. Treatment was started 6 hours after hospitalization. There were attacks of tonic spasms, mydriasis of the left pupil and a state of deep coma. What is the possible diagnosis?

Answers:
a) Alcohol poisoning.
b) Concussion.
c) Basal skull fracture with epidural hematoma.
d) Acute disturbance of blood circulation in the brain.
e) Meningoencephalitis.

Test tasks for self-control.

1. Which cranial nerve nuclei are damaged in Weber's alternating syndrome?
A. Abductor
B. Additional
C. Okorukhovoi
D. Trichastogo
E. Glossopharyngeal

2. What is characteristic of central paralysis of the facial nerve?
A. Loss of pupillary reflex
B. Trophic disorders of the tongue muscles
C. Damage to the masticatory group of muscles
D. Damage to facial muscles of the lower half of the face
E. Damage to facial muscles of the entire half of the face

3. A 42-year-old patient was diagnosed with a helical fracture of the middle third of the diaphysis of the right humerus. In addition to the "classic" picture of a fracture of this localization, the following are noted: the inability to actively extend the hand, active abduction of 1 finger, and loss of sensitivity on I-II and partially III fingers. Determine the appropriate treatment tactics.

A. Open reposition, revision of the nerve, stabilization of fragments
B. Closed one-moment reposition with subsequent external immobilization
C. Closed repositioning and fixation of fragments using external fixation devices
D. Skeletal traction

4. After a knife wound in the front area of the forearm, there is atrophy of the thenar muscles of the thumb with flattening of the palm ("monkey's hand"), hypalgesia and hyperpathy in the area of the palmar surface of the I-III fingers and the corresponding part of the palm. What nerve is damaged?

A. Elbow
B. Radial
C. Medial cutaneous nerve of the shoulder
D. Musculocutaneous
E. Average

5. Patient M., 27 years old, who had been in the cold for a long time, after sleep drew attention to the inability to close the left eye, lacrimation from it, hyperacusis, a spinal cord disorder on the front 2/3 of the tongue. The examination revealed flattening of the left nasolabial fold, drooping of the left corner of the mouth, absence of the left corneal reflex, and the symptom of "sail" on the left. Your diagnosis:

A. Neuralgia of the left trigeminal nerve

B. Neuritis of the left facial nerve

C. Neuritis of the right facial nerve

D. Tumor of the bridge-cerebellar angle on the left

E. Tumor of the bridge-cerebellar angle on the right

6. When opening the radial tenobursitis, the surgeon carelessly extended the incision into the proximal third of the tenor, as a result of which the patient lost the ability to oppose the first finger. Branch of which nerve did the surgeon cross?

A. Posterior interosseous

B. Lyktyovoi

C. Front interosseous

D. Promenevoi

E. Middle

7. The victim has a fracture of the body of the humerus. What nerve can be damaged?

A. Elbow

B. Radial

C. Medial cutaneous nerve of the shoulder

D. Musculocutaneous

E. Average

8. After a forearm injury, the patient has impaired function of the flexor muscles of the lateral group. What nerve is damaged?

AN cutaneus antebrachii

BN medianus

CN musculocutneus

DN radialis

EN ulnaris

9. Which one muscle not accepts participation in formation of foramen quadrilaterum?

AM biceps brachii

BM subscapularis

CM teres major

DM teres minor

E. M. triceps brachii

10. The patient has a sensitivity disorder on the lateral surface of the forearm. Which nerve damaged ?

AN Axillaris

BN medianus

CN musculocutaneus

DN radialis

EN ulnaris

11. The patient has a dysfunction of the parotid salivary gland. Which nerve strengthens her secretion ?

AN auricularis major
BN auricularis minor
CN re trosus major
DN re trosus minor
EN re trosus profundus

12. Patient A., delivered by ambulance to the hospital with a cut wound on the forearm. The examination revealed the absence of all types of sensitivity on the surface of the palm 1, 2, 3 and the radial half of the 4th finger. Active bending 2 and 3 fingers is impossible. Enter the correct diagnosis.

A. Damage to the ulnar nerve and flexor tendons of the 2nd and 3rd fingers
B. Damage to the radial nerve
C. Damage to the radial nerve and flexor tendons of the 2nd and 3rd fingers
D. Damage to the median nerve and flexor tendons of the 2nd and 3rd fingers
E. Damage to the flexor tendons of the 2nd and 3rd fingers

13. The patient has a cut wound in the lower third of the left shoulder. After the injury, the left hand was hanging down. The patient cannot unbend the hand, withdraw the thumb, unbend the fingers. What nerve is damaged near the painful one?

A. ulnar nerve
B. axillary nerve
C. brachial plexus
D. radial nerve
E. median nerve
4. To conclude

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2. Clinical guidelines in neurology. (Order of the Ministry of Health of Ukraine No. 487 dated August 17, 2007)
https://zakon.rada.gov.ua/rada/show/v0487282-07#Text

Lecture #2

Topic: Vascular diseases of the brain and spinal cord

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

Entire lectures

Educational:

- To acquaint students with the etiology and pathogenesis of HPMK (1st degree of abstraction)
- During the lecture, provide students with knowledge of the classification of HPMK, the main clinical symptoms of strokes (2nd degree of abstraction)
- On the basis of the lecture material, provide students with the opportunity to master the skills of diagnosis and treatment of hemorrhagic and ischemic strokes and transient disorders of cerebral circulation (3rd degree of abstraction)
- Aimed at the development of a professionally significant personality substructure ;
- Education of modern professional thinking in students ;
- Ensuring students learn the leading importance of domestic clinical, scientific and pedagogical schools, including Odesa, which made a significant contribution to the study of cerebro-vascular pathology;
- Acquisition of deontology and medical ethics skills by students.

Plan and organizational structure of the lecture

No	The main stages of the lecture and their content	Goals in levels of abstraction	Type of lecture, methods and means of activation of acquirers, equipment	Time allocation

I 1. 2.	Preparatory stage Setting an educational goal Providing positive motivation	 I I	According to the publication "Methodical recommendations for planning, preparation and analysis of a lecture"	5% (5 min)
II 3.	The main stage Presentation of lecture material according to the plan: 9. Actuality of theme 10. Definition 11. Classification 12. Etiology and main links of pathogenesis 13. Clinical picture 14. Diagnostics 15. Leading syndromes and differential diagnosis 16. Evaluation of the severity of the course 9. Treatment 10. Prevention	 II II II II II II II II II II	Slide presentation of lecture material Extracts from the medical histories of patients. Excerpts from clinical protocols of the Ministry of Health of Ukraine on providing medical care to patients.	85% (75 min)
III 4. 5. 6.	The final stage Summary of the lecture, general conclusions Answers to possible questions Tasks for independent preparation	 III III III	List of references, questions, assignments	10% (10 min)

Content of the lecture material

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

Classification of vascular diseases of the brain

1. **Diseases and pathological conditions leading to stroke**
2. **Main clinical forms of stroke**

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

B. Acute stroke

1. Transient disorders of cerebral circulation

a) transient ischemic attacks

b) cerebral hypertensive crises

2. Acute hypertensive encephalopathy
3. Membranous hemorrhages
a) subarachnoid
b) Epi - and subdural
4. Hemorrhage in the brain
a) parenchymatous
b) parenchymatous-subarachnoid
c) ventricular
5. Brain infarction (not embolic)
a) in the case of pathology of the main arteries of the head
b) in case of pathology of intracerebral vessels
c) other etiology
6. Brain infarction (embolic)
a) cardiogenic
b) other etiology

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

Sh. Localization of the lesion
1. Hemispheres of the brain
2. Brain stem
3. Ventricles of the brain
4. Multiple foci
5. The localization is not determined
IV . Nature and localization of vascular changes
A. Nature of vascular pathology
B. Localization of pathology

V. Characteristics of clinical syndromes

VI . State of working capacity

Brain strokes

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

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

In this section, parenchymal and subarachnoid hemorrhages are considered.

Parenchymal hemorrhage

Cerebral hemorrhage is characterized by a history of hypertension, hemorrhagic diatheses, post-traumatic and congenital aneurysms, septic endocarditis, alcoholism; rapid development of clinical symptoms against the background of acute psychoemotional stress or physical overload, nowadays; significant increase in blood pressure; rare tense pulse; significant hyperthermia (especially when blood breaks into the ventricles of the brain), pronounced violation of vegetative and vital functions (cardiac activity and breathing); very strong headache; vomiting (sometimes "coffee grounds"); meningeal symptoms, floating movements of the

eyeballs; hormetonia; convulsive attacks; hyperglycemia; changes in the blood characteristic of the "alarm reaction" of Selye's stress syndrome (aneosyphilia, lymphopenia, an increase in rod- and segmented-nuclear neutrophils, general hyperleukocytosis); compensatory significantly pronounced hypercoagulation (if the cause of hemorrhage is not hemophilia and other coagulopathy); facial hyperemia; congestive discs of the optic nerves against the background of hypertensive angioretinopathy; complication of venous outflow from the cranial cavity; dislocation of the middle structures according to ECHO-EG by 4-8 mm or more, blood in the cerebrospinal fluid; unconscious comatose state; cerebral edema clinic. Complications such as bedsores, pulmonary edema, thromboembolism of the pulmonary artery, etc. occur early. A fairly frequent complication of hemorrhage into the brain substance is the breakthrough of blood into the ventricles, which are accompanied by a sharp deterioration of the patient's condition, hyperthermia, (40-41 ^{0} C) breathing, deepening of other vegetative disorders, the development of hormonal syndrome, which is manifested by a constant change in the tone of the limbs, when they are hypertensive states with a sharp dissolution pass into a hypertensive state. Paroxysmal increase in muscle tone, especially if it prevails in the extensors, is similar to defibrillation rigidity, which is observed not only in the case of blood breakthrough in the ventricle meningeal symptoms of leukocytosis in peripheral blood, smooth or pendulum-like movements of the eyeballs also appear.

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

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

Diagnostics

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

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

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

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

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

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

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

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

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

This standard should take up to 30 minutes to complete.

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

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

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

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

After the measures taken, the patient is urgently hospitalized.

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

Correction of respiratory disorders is carried out (tracheal intubation, inhalation of an oxygen-air mixture, early activation of the patient, respiratory gymnastics, APC). Measures to stabilize the function of the cardiovascular system and blood pressure continue, for which the same drugs as at the pre-hospital stage continue to be administered. Normalize the water-

electrolyte balance. In the presence of hydrocephalus, 300-350 ml of fluid is injected; in case of diarrhea, vomiting, hyperhidrosis and high temperature - the amount of liquid is increased.

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

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

Prevention of complications is carried out - brain hydration (mannitol 25-50 g every 3-6 hours, 10% sodium chloride solution 50-100 ml, furosemide); pulmonary complications (antibiotics, correction of blood gas composition); urinary tract infections (catheterization as indicated, general urinalysis and bacteriological examination of urine, if necessary, antibiotics are prescribed), bedsores, deep vein thrombosis (compression stockings, fraxiparin, fragmin 7-10 days, warfarin per os). They monitor the improvement of the function of the gastrointestinal tract (metoclopramide).

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

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

A) basic therapy, prevention and treatment of complications

B) improvement of brain perfusion

C) prescription of nootropic drugs

D) use of surgical treatment methods

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

Antiplatelet agents:

Acetylsalicylic acid 100-300 mg (per day)

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

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

Anticoagulants

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

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

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

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

Phlebodia 600 mg 1 pill per day continued

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

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

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

Nootropic drugs

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

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

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

Glycine 1-2 g per day under the tongue

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

Antihypoxants:

Tocopherol 5%-2.0 ml or 10%-1.0 ml intravenously 1-2 times a day or in capsules - 2 capsules. x 3 years/ per day.

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

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

Mexidol 100 mg per 1 kg of body weight IV.

Differential ateralia of hemorrhage in the brain involves a complex:

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

B) stabilization of brain perfusion

C) appointment of differentiated drug therapy

D) use according to indicators of surgical methods of treatment

Differentiated drug therapy includes:

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

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

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

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

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

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

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

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

Constant laboratory control of blood coagulation indicators is carried out.

To prevent the development of angiospasm in case of subarachnoid hemorrhage, Nimotop (an active blocker of L -type calcium channels and transspinous brain-membrane influx

of calcium) is recommended for 2 pills (one pill contains 30 mg of Nimotop) every 6 hours for 21 days; or nemotan (nimodipine) 2 pills every 4 hours for 14-21 days.

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

Violation of spinal blood circulation

Etiological factors :

A) ***Pathology of the aorta.***

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

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

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

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

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

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

Occurs stroke-like over the course of one hour to one day,

sometimes at night while sleeping.

The clinical course depends on the level of damage.

Movement disorders are combined with sensitive (segmental in the ear area

coding, below - conductive type). Pathogenetic connections with compression and irritation of the anterior spinal or radiculomedullary artery (most often Adamkevich's artery), degeneratively - a changed disc or osteophyte.

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

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

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

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

Main syndromes **:**

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

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

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

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

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

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

Treatment

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

In the acute period of ischemic spinal strokes, drugs are prescribed that eliminate swelling of the spinal cord (mannitol, furosemide, ethacrynic acid, glycerin), normalize heart activity and blood pressure, improve microcirculation and metabolism of brain tissue

(reopoliglucin, cavinton, pentoxifylline, dipyridamole, nicotine preparations acids, nootropil, cerebrolysin), preventing thromboembolism (heparin).

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

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

Epidural hematomas require surgical treatment.

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

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

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

Initial manifestations of insufficient blood supply to the brain

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

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

Patients have no subjective complaints.

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

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

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

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

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

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

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

The diagnosis is made on the basis of complaints, the clinical picture of the disease, as well as based on the data of electrophysiological and laboratory research methods. It is important

to identify symptoms of general vascular disease: atherosclerosis, atrial hypertension, vegetative-vascular dystonia.

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

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

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

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

Syndrome of damage to various vascular basins .

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

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

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

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

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

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

A system of step-by-step treatment has been developed: polyclinic - hospital - resort - polyclinic, which includes dispensary supervision.

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

In the stage of clinical manifestations of the disease, outpatient or inpatient treatment is carried out taking into account the vascular pathology against which PPNKM developed. In the presence of neurosis-like complaints in the case of autonomic dystonia syndrome, sedatives (bromine, valerian), intermittent courses of treatment with tranquilizers in small doses, as well as belataminal, diphenhydramine, and calcium preparations are recommended. In case of arterial hypertension, Halidor, Cinnarizine, Anaprilin, Obzidan, etc. are prescribed. If blood pressure is low, caffeine, tinctures of ginseng, Chinese lemongrass, pantocrin, levzei are recommended. Various methods of reflexology and electrosleep are used.

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

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

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

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

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

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

Prevention.

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

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

Materials for student activation during lectures

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

General material and methodological support of the lecture:

1. Educational premises.
   - lecture room

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

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

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

Literature.

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

Additional literature

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

Electronic information resources

Clinical guidelines in neurology. (Order of the Ministry of Health of Ukraine No. 487 dated August 17, 2007)
https://zakon.rada.gov.ua/rada/show/v0487282-07#Text