METHODOLOGICAL INSTRUCTIONS
FOR THE INDEPENDENT WORK OF STUDENTS
FOR PREPARATION TO PRACTICAL CLASSES
AND DURING PRACTICAL CLASSES

<table>
<thead>
<tr>
<th>Educational discipline</th>
<th>Surgeon, including oncology and neurosurgery</th>
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</thead>
<tbody>
<tr>
<td>The module № 4</td>
<td>Neurosurgery</td>
</tr>
<tr>
<td>Employment theme</td>
<td>Traumatic lesions of the brain and spinal cord</td>
</tr>
<tr>
<td>Course</td>
<td>IV</td>
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<tr>
<td>Faculty</td>
<td>Foreign Students Training (stomatological)</td>
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Poltava 20___
1. **Actuality of the theme:** Neurotraumatism is one of the important sections of modern medicine. Trauma of CNS constitutes 30-40% in the general structure of traumatism, and death rate in the young and middle age considerably exceeds vascular and oncological diseases in peace, time, and also is a principal cause of death in wartime. This problem has not only medical, but also a big social significance as the level of traumatism tends to grow staidly.

Pathology of the spine and spinal cord of traumatic character is significant general medical and social problem. The doctor should be able to put the preliminary clinical diagnosis in primary inspection of the sick spine with pathology and the spinal cord and to render adequate medical aid.

2. **Specific objectives.** To determine the etiology, pathogenesis, classification, clinical picture of craniocerebral trauma; To define methods of clinical and instrumental diagnostics depending on the type of craniocerebral trauma; To evaluate the results of laboratory and instrumental examination of patients with craniocerebral trauma; Carry out a preliminary assessment of the severity of the condition of patients with craniocerebral trauma; Differentiate different types of craniocerebral trauma; Determine the algorithms of emergency care for patients with craniocerebral trauma; Determine the algorithms of conservative and surgical treatment, depending on the type of craniocerebral trauma; To determine the indications for urgent surgical interventions in case of severe craniocerebral trauma and to explain the basic principles of these interventions; Determine the prognosis after a craniocerebral injury, explain the development of major complications and possible consequences of craniocerebral trauma; To explain the principles of postoperative treatment and rehabilitation of patients with craniocerebral trauma; Demonstrate the ability to provide emergency medical care in emergency conditions in patients with the pathology of the vessels of the brain and spinal cord. Be able to provide first aid to a patient with CCT. Be able to conduct medical sorting of the wounded with CCT. Determine etiology, pathogenesis, biomechanics, classification, clinical picture of spinal cord injuries; Determine the methods of clinical and instrumental diagnosis, depending on the type of traumatic lesions of the spine, spinal cord; To be able to evaluate the results of laboratory and instrumental methods of examining victims with spinal cord trauma, spinal cord; Carry out a preliminary assessment of the severity of the condition of the victims with spine and spinal cord injury using the ASIA scale; Conduct differential diagnosis of various types of spinal cord injury, spinal cord; Determine the algorithm for emergency care for those injured with spinal cord trauma, spinal cord; To determine the algorithm of conservative and surgical treatment of injuries of the spine, spinal cord; To determine the indications for urgent surgical interventions for spinal trauma and to determine the basic principles of these interventions; Determine the prognosis, explain the development of major complications and consequences of spine trauma, spinal cord; To explain the principles of post-operative treatment and rehabilitation of victims with MSPT; Demonstrate possession of methods of restorative treatment of patients with spinal cord injuries, spinal cord, demonstrate the ability to provide
emergency medical care and carry out transport immobilization in case of spine and spinal cord injuries.

### 3. Basic level of preparation.

<table>
<thead>
<tr>
<th>The name of the previous disciplines</th>
<th>The skills</th>
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<tbody>
<tr>
<td>Anatomy</td>
<td>To know an anatomical constitution main and a spinal cord, a brain tunic, liqor conductive pathes, anatomy of bones of a skull, a column. Features of the structure of the spine and spinal cord, membranes, liquor-conducting pathways.</td>
</tr>
<tr>
<td>Histology</td>
<td>To know a histological composition of a brain, a constitution of peripheral nerves, concepts of a neuroglia. Microscopic structure of the spinal cord.</td>
</tr>
<tr>
<td>Neurology</td>
<td>To know procedure of neurologic survey of the patient, localisation of functions in a cortex and a brain fulcrum, principles of an establishment of the topical diagnosis. Knowledge of topical diagnosis, features of clinical symptoms in case of damage to the spinal cord, depending on the type and level of damage.</td>
</tr>
<tr>
<td>Physiology</td>
<td>To know physiology of generation and transmission of nervous impulse, concept of excitability of the nervous cell. To know procedure of recording EEG, interpreting of the gained effects.</td>
</tr>
<tr>
<td>Radiology</td>
<td>X-ray changes on spondylograms with traumatic injuries of the spine and spinal cord.</td>
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<tr>
<td>Topographical anatomy</td>
<td>To know an interrelation of structures of a brain among themselves, brain pots, and osteal structures of a skull and a column, plan of Kronlein.</td>
</tr>
<tr>
<td>Operative surgery</td>
<td>To know trepanation views (bone-plastic, excision), to the technician of carrying out of trepanation and laminectomy. A constitution and a principle of operation of the stereotaxic...</td>
</tr>
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</table>
Pharmacology and Clinical Pharmacology

Know the groups of drugs that are used in the treatment.

4. Task for self-study during the preparation for the lesson.

4.1. List of main terms, parameters, characteristics that students must learn in preparing for lesson: brain stem, focal symptomatology, dislocation, coma, sopor, intracranial pressure, perfusion pressure, mean arterial pressure, penetration furrow, Willisium circle, trepanation, subdural hematoma, epidural hematoma, intraventricular hemorrhage, subarachnoid hemorrhage, liquororrhea, Diffuse axonal injury, mass-effekt, edema-swelling of the brain, osmolarity, bruise of the brain.

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Brain detritus</td>
<td>Crushed brain tissue in the form of porridge, in which there are particles of gray and white matter of the brain, bone fragments, as well as small foreign bodies (scraps of headgear tissue, hair, etc.)</td>
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<tr>
<td>Dislocation syndrome</td>
<td>Focal symptom complexes resulting from impaired function of a part of the brain that is located at a certain distance, sometimes significant, from the primary pathological focus, which is associated with the dislocation (displacement) of the brain due to pressure differences in different cavities of the craniovertebral space and its alignment.</td>
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<tr>
<td>Cranioplasty</td>
<td>Surgical intervention, the purpose of which is to restore the integrity of the cranial box after traumatic and surgical defects of the skull bones.</td>
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<tr>
<td>Cerebral edema</td>
<td>An increase in the water content in brain tissues arising as a result of increased vascular permeability of the blood-brain barrier and accompanied by the release of plasma and its components into the intercellular space from the lesion, where the tissue pressure is higher, into the underlying white matter.</td>
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<tr>
<td>Brain swelling</td>
<td>An increase in brain volume due to an increase in its blood supply in the acute period of traumatic brain injury.</td>
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<tr>
<td>Milling hole</td>
<td>Method for surgical diagnosis of traumatic intracranial hematomas, the initial stage of craniotomy. It is used in cases of impossibility of preoperative verification of the causes of the provided brain compression using modern diagnostic methods (CT, MRI, carotid angiography, etc.)</td>
</tr>
<tr>
<td>Term</td>
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<tr>
<td>Unstable damage</td>
<td>The instability of the lesion is caused by a violation of the anatomical integrity of the vertebrae, discs, joints and ligamentous apparatus, in which it is possible to re-shift the vertebrae with additional compression of the spinal cord and roots. Unstable injuries of the spine include many-lobed (explosive) fractures, rotational injuries, vertebral dislocations, fractures and dislocations of articular processes, ruptures of intervertebral discs and their combination with damage to vertebral bodies. All patients with instability of the spine require therapeutic stabilization (using corsets, tires, screeds, using surgical methods).</td>
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<tr>
<td>Hematomyelia</td>
<td>Hematomyelia is a hemorrhage into the gray matter of the spinal cord, manifested by dissociated sensitivity disorders.</td>
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<tr>
<td>Open spinal cord injury</td>
<td>Open are the injuries of the spine and spinal cord, in which the wound site of soft tissues coincides with the site of spinal injury and there are prerequisites for infection of the spinal cord and its membranes.</td>
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<tr>
<td>Spondylodez</td>
<td>Operative intervention, whose goal is to fix and stabilize the spine with the help of bone, plastic material or metal structures.</td>
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<tr>
<td>Laminectomy</td>
<td>Operative intervention on the spine, in which the resection of the spinous processes and arches of the vertebrae is performed.</td>
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<tr>
<td>Electroneuromyography</td>
<td>The method of investigation, allowing to register the potential of the action of the nerve and separate groups of muscle fibers, determine the speed of the pulse in different groups of fibers in different parts of the nerve. This method most fully characterizes the degree of impairment of nerve conduction and denervation changes in muscles, allows to determine the level of damage and trace the dynamics of the regeneration process.</td>
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4.2. Theoretical questions to the lesson:

2. Primary factors of biomechanics of destructive action on brain tissue.
6. Diffuse axonal damage.
7. Classification of traumatic brain injury by nature, criteria for determination.
10. Types of trepanation, technique, indications.
12. Levels of consciousness, Glasgow com scale.
15. Classification of military injuries of the skull and brain.
17. Classification of traumatic injuries of the spine and spinal cord.
18. What are the clinical signs of spinal cord injury?
19. What clinical symptomatology of vertebral-spinal trauma, depending on the level of lesion? First aid at the prehospital stage.
20. What should be the tactic of a neurosurgeon in the case of fracture-spinal dislocations, depending on the level of lesion?
21. What could be the reason for the compression of the spinal cord in vertebral-spinal trauma?
23. Modern principles of surgical treatment of injuries of the spine and spinal cord. What are the early and late complications of traumatic spine and spinal cord injuries?
25. What types of treatment are used in the recovery period?
26. How is rehabilitation of patients with traumatic injury of the spine, spinal cord carried out?

4.3. Practical work (task) that run in class:
1. Investigation of the general-somatic and neurological status.
2. Registration of medical documentation.
3. Lumbar puncture.
4. Interpretation of CT, MRI.
5. Ability to carry out resection and bone-plastic trepanation (theoretically).
6. Ability to conduct PCW of head wounds.
7. Definition of the projection of the main trunk of the middle shell artery, the main grooves of the brain according to the Cronlein scheme.
8. Conduct a clinical examination of vital indicators (determination of the pulse, blood pressure, auscultation of the heart and the main arteries of the neck, respiration).
10. Determination of focal symptoms of lesions of the spinal cord, roots, nerves.
11. Definition of meningeal symptoms.
12. Determination of the presence and type of disorder of the pelvic organs.
13. Evaluation of auxiliary methods of investigation in trauma (general blood test, coagulogram, CSF analysis, spinal angiography, myelography, spine X-ray, CT, MRI of the spinal cord).
14. To be able to conduct differential diagnosis of concussion, contusion and complete break of the spinal cord, compression of the brain.
15. Provide emergency assistance in case of spinal cord injury.

**Content of the topic:**

**Craniocerebral trauma**

1. Anatomy-physiological features of the nervous system
   Division of the nervous system into the central nervous system and peripheric nervous system. The central nervous system
   - Brain
     - Constitution of an end brain
     - Localization of functions in a cortex of a brain
     - Constitution of a fulcrum of a brain
     - Leading pathes
   - Peripheric NS
     - Segmentary constitution of a spinal cord
     - Metameric constitution of the person
     - Leading pathes
   - Spinal cord
     - Roots of a spinal cord
     - Spinal ganglions
     - Spinal nerves
     - Plexus
     - Peripheral nerves

2. Liquor conductive pathes
   - Brain ventricles
   - Arachnoidal tanks
   - Liquor circulation
   - Function and liquor composition
   - Brain tunic

3. Topographical anatomy
   - Skull constitution
   - Constitution of the backboned canal

4. Blood supply main and a spinal cord:
   - Brain
     - Basin of an intrinsic carotid artery
     - Vertebrobasilar circle
     - Viliziev circle
- Venous system of a brain
  - brain Sines
- Spinal cord
  - Spinal arterial tracts
  - Additional arteries (arteries of Adamkevich, Deprozh-Gotterona)

5. Pathophysiological aspects of the nervous activity
- Intracranial pressure components
- Methods of measuring ICP
- Brain perfusion
- Dislocation - development mechanisms, views

6. Clinical implications
- Cerebral symptoms
- Focal symptoms (primary focal and secondary focal):
  - irritation symptoms
  - Symptoms of abaissement (function)
- Consciousness levels
- Measure of definition of a state of gravity of the patient

7. The basic methods of surgical interventions
- Craniotrypesis
- Bone-plastic
- Excision
- Laminectomy

8. Clinical implications
- Hyperkinesias
- Children's cerebral paralysis
- Painful syndromes

9. Neurosurgical methods of the functional and regenerative neurosurgery
- Stereotaxic operations
- Electrostimulating methods
- Transplantation of founder cells

Craniocerebral trauma is a type of head trauma in which soft tissues of the head, skull and intracranial contents (brain, brain membranes, vessels, cranial nerves) are damaged by mechanical energy. Craniocerebral trauma plays a dominant role in the disease and mortality of the population of economically developed countries, it is a global problem not only of neurosurgery, it is a multidisciplinary problem of medicine in general and the whole social sphere. Annually statistics fix craniocerebral trauma in the range of 200 cases per 10000 population. It is the main cause of death and disability of patients under the age of 45 years. Of the 4 lethal cases due to injuries of all localizations - one is the result of a craniocerebral injury.
Complications of CCT.

**Abscess** - a cavity filled with pus and delimited by a capsule from the brain substance.

**Layers of the abscess:**
- zone of disintegration;
- granulation a layer (vessels are located radially in it);
- fibrous (several circularly located vessels);
- perifocal zone.

The capsule is formed from vessels, hence, the deeper the abscess is located in the white substance, the thinner is it capsule. The capsule is the thickest in the cortex.

**Microbes:** more often it is staphylococci (the thickest capsule), diplococci, coli facillus.

**Pathogenesis:** an embolus - ischemia - microbes with walls of the vessel - vasculitis - perivasculitis - distribution of the abscess.

**Kinds of abscess perforation:**
- microperforation - abscesses as ”cluster of grapes”;
- macroperforation - abscesses can perforate in the brain substance, in ventricles of the brain, in to the subarachnoidal space.

By terms of occurrence there are early (till 3 months) and late (after 3 months) abscesses. The capsule of the abscess is formed not earlier than 3-6 weeks. Till this time it is possible to wash it out with solutions of antiseptics and antibiotics (Canamycin, Levomycetin, Gentamycin) through a fistula and drainage. As a result of it the cavity is formed which is then removed, or the cavity is closed gradually by itself and pushes out a drainage. This tactics is acceptable in deeply located abscesses.

**Kinds of a surgery:**
- drainage;
- puncture;
- total removal.

The clinical picture of the early abscess develops from general brain and focal symptoms.

Late abscesses have a course as:

**apoplexies:** sudden development of general brain symptoms. More often death occurs the first 24 hours. Blood and the eye fundus can not react in any way.

By the course there are acute the period, latent, marked clinical manifestation (or - early manifestation, latent manifestation of the abscess, terminal. The latent period is a silent course of the abscess).

**pseudotumorous variant of the course:** Blood does not react. On the eye fundus stagnation is determined. The general brain and focal manifestations gradually grow.

More than 50 % of all abscesses are accompanied by epileptic attacks. In 30 % of cases they are first signs of the abscess. They have s course as the general and focal generalized attacks. There is marked polymorphism of attacks (multifoci in a trauma and growth of the abscess).
Diagnostics:
- examination of the head;
- anamnesis;
- R-graphy of the skull;
- Echoencephalography;
- investigation of the eye fundus;
- EEG - in development of seizures (only for lateralization of the process);
- angiography, CT-SCAN, MRI, scintigraphy.

**LATE COMPLICATIONS OF CCT - THE POSTTRAUMATIC EPILEPSY**

**Epilepsy** is a pathological excitation of the brain accompanying by convulsive or convulsive-free attack.

**Factors:**
- convulsive readiness of the brain;
- presence of the epileptogenic center - cicatrices (cerebral, meningeal - cerebral, it is cutaneous - meningeal-cerebral);
- consequences of the inflammatory process;
- development of subarachnoidal cysts (inflammation, subarachnoidal haemorrhages);
- presence of foreign bodies (a bone, a bullet, splinters, soft tissues);
- hydrocephalus of the brain.

**Structure of epileptic attacks:**
- great
- the general the patient - suddenly loses consciousness, falls, the face reddens, the tonic spasms passing in to clonicoues, cyanosis of integuments, involuntary urination.
- the focal - always there is an aura (motor, sensor, psychosensor, vegetative)
- Jackson attacks without loss of consciousness and without generalization of spasms (motor and sensor) - convulsive discharge in some group of muscles, paresthesias in some extremity. It upper parietal lobule is affected the attack proceeds by hemitype.
- psychosensor equivalents - petit mall - short-term, loss of consciousness for 20-30 seconds without any convulsive component and without falling down.

**Phases of formation of the cicatrix of the brain:** glial, argirophil, collagenic.

**Examination of the patient with seizures:**
- R-graphy of the skull in two projections;
- EEG - acute wave are determined in the focus. In irritation (by light, sound, hyperventilation, bemegrid 1 ml, thyopental sodium - slower waves are determined in the focus)

**Indications to the operation:**
- morphological
- cicatrices;
- abscesses;
- foreign bodies;
- the pressed-in fractures;
- adhesive or cystic arachnoiditis
- clinical
- absence of effect of medicamentous treatment at often attacks;
- progredient course of the disease;
- increasing degradation of the personality.

_Contra-indications to the operative intervention:_
- massive adhesive processes;
- multiple wounds (fraction);
- processes in the vital sections of the brain.

**COMBINED CCT**

_The combined trauma_ - simultaneous injury by one kind of mechanical energy of two or more anatomical-topographical systems (craniovertebral, cranio-transabdominal).

_Multiple trauma_ - simultaneous injury by one kind of damaging energy of one body, or several bodies of one system (multiple contusions of the brain, multiple fractures of the lower extremity).

_The combined affection_ - injury of the organism by various damaging factors working simultaneously (mechanical, thermal, radial energy).

**Classification:**
- damages of the facial skeleton
- damages of the thorax and respiratory organs
- damages of the abdominal cavity
- damages of the spine and spinal cord
- cranial damages

**Classification of combined CCT by a degree of severity:**
- severe CCT and severe extracranial (shock in 70 %)
- severe CCT and not severe extracranial (shock in 14-15 %)
- not severe CCT and severe extracranial (shock in 40-50 %)
- not severe CCT and not severe extracranial (shock in 4-5 %)

The leading part in development of shock in CCT is played by an extracranial pathology. The shock in isolated CCT develops in:
- multiple injures of the bones of the arch and the basis of the skull (of type)
- multiple injures of soft tissues of the head (of hemorrhagic type)
- in children (any haematomas can cause a hypovolemic shock)

Difference of shock from damages of the brain stem. If there is a decrease of hemodynamics, disturbances of breath and stem (floating eyeballs, anisocoria, Chaine-Stocks respiration) it should be attributed to CCT. Isolated CCT has shock in 1-1.5 % of cases.

In mild CCT there is an amplification of function of the hypophysis (secretion of CTH grows), promoting the prompt formation of an osseous callous. And in severe CCT - function of the hypophysis is suppressed.
Facial damages:
- single fracture of the jaw
- traumatic extraction of a tooth
- injuries of soft tissues, without a severe bleeding
- Лежфор 2, 3
- multiple damages of the facial skeleton

Thoracic damages
- fracture of the clavicle
- fracture up to 3 ribs without damage of the organs of the chest, nerves and vessels
- fracture of ribs with damage of the vessels
- damage of the organs of the chest
- hemo-pneumothorax
- damage of the organs of the mediastinum

Transabdominal damages
- subserous rupture of the gut
- any damages of hollow and parenchymatous organs

Vertebral damages
- fracture of bodies, arches, but without damage of the spinal cord and roots
- fracture of bodies, arches with damage of the spinal cord or roots

Damages of the locomotor system:
- the closed single fracture of the forearm, shin
- fracture of the pelvis, hip, open fractures, multiple fractures of bones, tearing off of the feet

FATTY EMBOLISM
Fatty embolism is characterized by sudden, quick onset (hemiparesis or a plegia, disturbances of consciousness, narrow pupils). In LP - liquor is pure or hemorrhagic. On the 2nd-3rd day there is fat in urine.

Typically haematoma has a gradual onset.

Fatty microthrombembolism occurs more often in the diencephalic areas.

**Differential diagnostics of fatty embolism and intracranial haematoma**

<table>
<thead>
<tr>
<th>Intracranial haematoma</th>
<th>Fatty embolism</th>
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<tbody>
<tr>
<td><strong>Severity of a craniocerebral trauma</strong></td>
<td></td>
</tr>
<tr>
<td>characteristic severe CCT</td>
<td>the combined damages and CCT are usually a little milder</td>
</tr>
<tr>
<td><strong>Severity of the combined damages</strong></td>
<td></td>
</tr>
<tr>
<td>various</td>
<td>usually severe</td>
</tr>
</tbody>
</table>
**Disturbance of consciousness**

| gradual aggravation of a degree of disturbance of consciousness | sudden sharp disturbance of consciousness |

**Pyramidal symptoms**

| gradual increase | are sharply expressed at once. If there is no pyramidal manifestation, diencephalic and mesencephalic signs develop (paresis of the look, narrowing of pupils, floating eyeballs) |

**Eye fundus**

| vessels are dilated | spasm of the arteries, haemorrhages, veins are fragmented |

**Echoencephalography**

| displacement of the M - echo | There is no displacement of the median structures |

**Lumbar puncture**

| the increased pressure, blood in liquor | transparent or xanthochromatic |

**Diagnostic mill apertures**

| Haematoma | Nothing is defined |

**R-graphy of the lungs**

| There is no thing | The lung with "snow" |

| petechial rash on a lateral surface of the stomach, fat in urine on the 2nd-3rd day |

For the prevention of development of fatty embolism in patients with the combined trauma it is necessary to administer the preparations having immediate effect:

- rheologic properties of blood
- Lipin, Lipostabil, Essentiale
- antagonists of calcium (Verapamil, Nifedipine)
- to increase ОЦК (10 % NaCl 100 ml + 100 ml Rheopolyglucin)
- ГОМК, Nootropics, Difenin
- transfusion of liquids under the control of intracranial pressure (the control of blood osmolarity: if osmolarity is normal - intracranial pressure is normal too, if osmolarity of plasmas is reduced - intracranial pressure is always increased). It implies, that in the first 2-3 days it is better to administer osmodiuretics, and then saluretics.
- Carrying out of functionally stable osteosynthesis (plates, a spoke, but not a nail):
• the first 4-6 hours – in mild or moderate CCT without disturbances (shock)
• on removing from a shock - in mild or moderate CCT and a shock accompanying it
• after stabilization of vital functions – in severe CCT with vital disorders
  If fatty embolism has developed, it is necessary to influence development of collateral vessel. Transfusion of liquids under the control of intracranial pressure (the control of blood osmolarity: if osmolarity is normal - intracranial pressure is normal too, if osmolarity of plasma is reduced - intracranial pressure is always increased).

• Antagonists of calcium
• increase of resistency of the brain tissue to hypoxia
• The Signay-cocktail - is introduced once a day intravenously

<table>
<thead>
<tr>
<th>500 ml of 20% Mannitol</th>
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<tr>
<td>50 mg Dexasone (Metipred)</td>
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<tr>
<td>500 mg of vitamin E</td>
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<tr>
<td>500 mg Difenine</td>
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</tbody>
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• solutions
• Lipostabil, Inhibitors of proteases, Essentiale

**TRAUMA OF THE SPINE AND SPINAL CORD**

Mechanisms of Spine and Spinal Cord Injury. Although trauma may involve the spinal cord alone, the vertebral column is almost invariably injured at the same time. A useful classification of *spinal injuries* is one that divides them into fracture-dislocations, pure fractures, and pure dislocations. The relative frequency of these types is about 3:1:1. Except for bullet, shrapnel, and stab wounds, a direct blow to the spine is a relatively uncommon cause of serious spinal cord injury. One such effect is embolism of disc material, in which nucleus pulposus is propelled under pressure into the adjacent vasculature of the cord. In civilian life, most spinal injuries are the result of *force applied at a distance*. All three types of spinal injury mentioned above are produced by a similar mechanism, usually a vertical compression of the spinal column to which anteroflexion is almost immediately added (anterohyperflexion injury); or, the mechanism may be one of vertical compression and retrohyperflexion (commonly referred to as hyperextension). The most important variables in the mechanics of vertebral injury
are the nature of the bones at the level of the injury and the intensity, direction, and point of impact of the force.

When the cervical spine is sharply retroflexed, the spinous and articular processes of the midcervical vertebrae (C4 to C6) are forced together, and these, now acting as a fulcrum, cause a separation between the vertebral body and the adjacent lower intervertebral disc. This results in dislocation, and the cord is caught between the laminae of the lower vertebra and the body of the higher one. Depending upon the intensity of the driving force, the separation may increase, with rupture of the anterior ligament. Hyperextension injury to the spinal cord may occur without apparent damage to the vertebrae, being caused by a sudden inward bulge of the ligamentum flavum. Damage to the central cord is usually due to an extension injury.

In the case of severe forward flexion injury, the head is bent sharply forward when the force is applied. The adjacent vertebrae are forced together at the level of maximum stress. The anterior-inferior edge of the upper vertebral body is driven into the one below, sometimes splitting it in two. The posterior part of the fractured body is displaced backward and compresses the cord. Concomitantly, there is tearing of the interspinous and posterior longitudinal ligaments. Less severe degrees of anteroflexion injury produce only dislocation. Vulnerability to the effects of anteroflexion and retroflexion injuries is increased by the presence of cervical spondylosis or ankylosing spondylitis or a congenital stenosis of the spinal canal.

The spinal cord may be damaged without radiologic evidence of fracture or dislocation, particularly in children, but sometimes one cannot determine the full extent of spinal injury even at autopsy, because of the difficulty in examining the vertebrae. Computed tomography, MRI, and lateral spine films are all satisfactory means of demonstrating the vertebral injury, but the tearing of ligaments from vertebral dislocation can only be inferred from the spinal displacement. Radiologic studies during cautious flexion or extension of the neck are the only way one can demonstrate instability from ligamentous injury alone.
Another mechanism of cord and root injury, involving extremes of extension and flexion of the neck, is so-called whiplash or recoil injury. This type of injury is most often the result of an automobile accident. When a vehicle is struck sharply from behind, the head of the occupant is flung back uncontrollably; or, if a fast-moving vehicle stops abruptly, there is sudden forward flexion of the neck, followed by retroflexion. Occipitonuchal and sternocleidomastoid muscles and other supporting structures of the neck and head are affected much more often than the spinal cord or roots. Nevertheless, in rare instances, quadriplegia, temporary or permanent, results from a violent whiplash injury. The exact mechanism of neural injury in these circumstances is not clear; perhaps there is a transient posterior dislocation or momentary retropulsion of the intervertebral disc into the spinal canal. Again, the presence of a congenitally narrow cervical spinal canal or of spinal diseases such as cervical spondylosis, rheumatoid arthritis, or ankylosing spondylitis adds to the hazard of damage to the cord or roots. Spondylitic symptoms may be aggravated. Also, there are examples of spinal cord compression that result from the persistent hyperextension of the cervical spine during a protracted period of coma. Arterial hypotension may be an added factor in particular instances. This combination accounts for some of the cases of quadriplegia in opiate or other drug addicts following a period of sustained unresponsiveness.

A special type of spinal cord injury, occurring most often in wartime, is one in which a high-velocity missile penetrates the vertebral canal and damages the spinal cord directly. In some cases the missile strikes the vertebral column without entering the spinal canal but virtually shatters the contents of the dural tube or produces lesser degrees of impairment of spinal cord function. Rarely, the transmitted shock wave will cause a paralysis of spinal cord function that is completely reversible in a day or two (spinal cord concussion). This latter condition may also be produced by forceful falls flat on the back, as occurs not infrequently in athletes engaged in contact sports or in falls from a ladder. Little is known of the underlying pathologic changes. Acute traumatic paralysis may also
be the consequence of a vascular mechanism. As mentioned earlier, fibrocartilaginous emboli from an intervertebral disc that has ruptured into radicular arteries and/or veins of the spinal cord may cause infarction. Or, a traumatic dissecting aneurysm of the aorta may occlude the segmental arteries of the spinal cord.

The usual circumstances of spinal cord injury have been motor vehicle accidents, falls (mainly during a state of alcoholic intoxication), gunshot or stab wounds, diving accidents, motorcycle accidents, crushing industrial injuries, and birth injury, in that order of frequency. The majority of the fatal cases were associated with fracture dislocations or dislocations of the cervical spine. Respiration is paralyzed by lesions of C1, C2, and C3 segments. Among nonfatal cases, fracture-dislocation of the lower cervical spine is the most frequent established mechanism of spinal cord injury in civilian life. In the United States, the annual incidence of spinal cord injury is from 5 to 5.5 cases per 100,000 population. Males predominate (4:1). Each year about 3500 persons die in close relation to their injury, and another 5000 are left with complete or nearly complete loss of spinal cord function.

Pathology of Spinal Cord Injury. As a result of squeezing or shearing of the spinal cord, there is destruction of gray and white matter and a variable amount of hemorrhage, chiefly in the more vascular central parts. These changes are maximal at the level of injury and one or two segments above and below it. Rarely is the cord cut in two, and seldom is the pia-arachnoid lacerated. The condition is best designated as traumatic necrosis of the spinal cord tissue. Separation of pathologic entities such as hematomyelia, concussion, contusion, and hematorrhachis (bleeding into the spinal canal) that are concomitant is of little value either clinically or pathologically. As a lesion heals, it results in a gliotic focus or cavitation with variable amounts of hemosiderin and iron pigment. Progressive meningeal fibrosis and a tension syringomyelia will sometimes develop and lead to a delayed central cord syndrome.
In most traumatic lesions, the central part of the spinal cord with its vascular gray matter suffers greater injury than the peripheral parts. And in some instances, the lesion is virtually restricted to the anterior and posterior gray matter, giving rise to segmental weakness and sensory loss in the arms with few long tract signs. This has been called the central cervical cord syndrome (or Schneider syndrome, see further on). Fragments of the syndrome are not uncommon as transient phenomena that reverse over several days.

As with most lesions, the total clinical effect is compounded of an irreversible structural component and a reversible disorder of function, each of which may vary in degree. The extent and permanence of the clinical manifestations are determined by the relative proportions of these two elements.

**Clinical Effects of Spinal Cord Injury.** When the spinal cord is suddenly and completely or almost completely severed, three disorders of function are at once evident: (1) all voluntary movement in parts of the body below the lesion is immediately and permanently lost; (2) all sensation from the lower (aboral) parts is abolished; and (3) reflex functions in all segments of the isolated spinal cord are suspended. The last effect, called spinal shock, involves tendon as well as autonomic reflexes; it lasts for weeks to months and is so dramatic that Riddoch used it as a basis for dividing the clinical effects of spinal cord transection into two stages: (1) spinal shock or areflexia and (2) heightened reflex activity. The separation of these two stages is not as sharp as this statement might imply but is nevertheless fundamental. Less complete lesions of the spinal cord may result in little or no spinal shock, and the same is true of any type of lesion that develops slowly.

**Stage of Spinal Shock or Areflexia.** The loss of motor function at the time of injury quadriplegia (better termed tetraplegia) with lesions of the fourth to fifth cervical segments, paraplegia with lesions of the thoracic cord is accompanied by atonic paralysis of bladder and bowel, gastric atony, loss of sensation below the level corresponding to the spinal cord lesion, muscular flaccidity, and complete or almost complete suppression of all spinal segmental reflex activity below the
lesion. The neural elements below the lesion fail to perform their normal function because of their sudden separation from those of higher levels. Impaired also in the segments below the lesion is the control of autonomic function. Vasomotor tone, sweating, and piloerection in the lower parts of the body are temporarily lost. Systemic hypotension may be severe and contribute to the spinal cord damage. The lower extremities lose heat if left uncovered, and they swell if dependent. The skin is dry and pale, and ulcerations may develop over bony prominences. The sphincters of the bladder and the rectum remain contracted to some degree due to the loss of inhibitory influence of higher central nervous system centers but the detrusor and smooth muscle of the rectum are atonic. Urine accumulates until the intravesicular pressure is sufficient to overcome the sphincters; then dribblets escape (overflow incontinence). There is also passive distention of the bowel, retention of feces, and absence of peristalsis (paralytic ileus). Genital reflexes (penile erection, bulbocavernosus reflex, contraction of dartos muscle) are abolished or profoundly depressed.

The duration of the stage of complete areflexia varies greatly. In a small number it is permanent, or only fragmentary reflex activity is regained many months or years after the injury. In such patients the spinal segments below the level of transection may have themselves been injured perhaps by a vascular mechanism, although this explanation is unproven. More likely there is a loss of the brainstem-spinal facilitatory mechanisms and an increase in inhibitory activity in the isolated segments as indicated below. In some patients, minimal genital and flexor reflex activity can be detected within a few days of the injury. In the majority of patients, this minimal reflex activity appears within a period of 1 to 6 weeks. Usually the bulbocavernosus reflex is the first to return.

The explanation of spinal shock, which is brief in submammalian forms and more lasting in higher mammals, especially in primates, is believed to be the sudden interruption of suprasegmental descending fiber systems that normally keep the spinal motor neurons in a continuous state of subliminal depolarization (ready to respond). In the cat and monkey, Fulton found the facilitatory tracts in question
to be the reticulospinal and vestibulospinal. Subsequent studies showed that in monkeys, some degree of spinal shock can result from interruption of the corticospinal tracts alone. This cannot be the significant factor, however, at least in humans, because spinal shock may be very mild or inapparent as a result of acute cerebral and brainstem lesions that interrupt the corticospinal tracts. F waves of the isolated cord are suppressed. Interest in recent years has focused on a possible role for neurotransmitters (catecholamines, endorphins, substance P, and 5-hydroxytryptamine). The claim that naloxone and the endogenous opiate antagonist thyrotropin releasing factor might reduce the extent of an acute spinal cord lesion has not been corroborated. Clonidine, a noradrenergic receptor activator is reported to reduce flexor spasms and spasticity and to restore the balance between excitatory and inhibitory activity, allowing the spinal reflex generator for locomotion to function.

**Stage of Heightened Reflex Activity.** Usually, after a few weeks, the reflex responses to stimulation, which are initially minimal and unsustained, become stronger and more easily elicitable and come to include additional and more proximal muscles. Gradually the typical pattern of heightened flexion reflexes emerges: dorsiflexion of the big toe (Babinski sign); fanning of the other toes; and later, flexion or slow withdrawal movements of the foot, leg, and thigh with contraction of the tensor fascialata (triple flexion). Tactile stimulation of the foot may suffice as a stimulus, but a painful stimulus is more effective. The Achilles reflexes and then the patellar reflexes return. Retention of urine becomes less complete, and at irregular intervals urine is expelled by active contractions of the detrusor muscle. Reflex defecation also begins. After several months the withdrawal reflexes become greatly exaggerated, to the point of flexor spasms, and may be accompanied by profuse sweating, piloerection, and automatic emptying of the bladder (occasionally of the rectum). This is the “mass reflex,” which is evoked by stimulation of the skin of the legs or by some interoceptive stimulus, such as a full bladder. Varying degrees of heightened flexor reflex activity may last for years. Heat-induced sweating is defective, but reflex-evoked ("spinal") sweating
may be profuse. Presumably, in such cases the lateral horn cells in much of the thoracic cord are still viable and disinhibited. Above the level of the lesion, thermoregulatory sweating may be exaggerated and is accompanied by cutaneous flushing, pounding headache, hypertension, and reflex bradycardia. This latter syndrome ("autonomic dysreflexia") is episodic and occurs in response to a particular stimulus, such as a distended bladder or rectum. It has been ascribed to the reflex release of adrenalin from the adrenal medulla and of noradrenalin from the disinhibited sympathetic terminals caudal to the lesion.

Extensor reflexes eventually develop in most cases, but they do not lead to the abolition of the flexor reflexes. The overactivity of extensor muscles may appear as early as 6 months after the injury, but this only happens, as a rule, after the flexor responses are fully developed. Extensor responses are at first manifest in certain muscles of the hip and thigh and later of the leg. In a few patients extensor reflexes are organized into support reactions sufficient to permit spinal standing.

From these observations one would suspect that the ultimate posture of the legs flexion or extension does not depend solely on the completeness or incompleteness of the spinal cord lesion. The development of paraplegia in flexion relates also to the level of the lesion, being seen most often with cervical lesions and progressively less often with more caudal ones. Repeated flexor spasms, which are more frequent with higher lesions, and the ensuing contractures ultimately determine a fixed flexor posture. Conversely, reduction of flexor spasms by elimination of nociceptive stimuli (infected bladder, decubiti, etc.) favors an extensor posture of the legs (paraplegia in extension). The positioning of the limbs during the early stages of paraplegia greatly influences their ultimate posture. Thus, prolonged fixation of the paralyzed limbs in adduction and semiflexion favors subsequent paraplegia in flexion. Placing the patient prone or placing the limbs in abduction and extension facilitates the development of predominantly extensor postures. Nevertheless, strong and persistent extensor postures are observed only with partial lesions of the spinal cord.
Of some interest is the fact that many patients report sensory symptoms in segments of the body below the level of their transection. Thus, a tactile stimulus above the level of the lesion may be felt below the transection (synesthesia). Patients describe a variety of paresthesias, the most common being a dull, burning pain in the lower back and abdomen, buttocks, and perineum. We have encountered patients in whom aching testicular or rectal pain were the main problem. The pain may be intense and last for a year or longer, after which it gradually subsides. It may persist after rhizotomy but can be abolished by anesthetizing the stump of the proximal (upper) segment of the spinal cord, according to Pollock and his collaborators. Transmission of sensation over splanchnic afferents to levels of the spinal cord above the lesion, the conventional explanation, is therefore not the most plausible one.

The overactivity of neurons in the isolated segments of the spinal cord has several explanations. One assumes that suprasegmental inhibitory influences have been removed by the transection, so that afferent sensory impulses evoke exaggerated nocifensive and phasic and tonic myotatic reflexes. But isolated neurons also become hypersensitive to neurotransmitters.

Various combinations of residual deficits (lower and upper motor neuron and sensory) are to be expected. Some of the resulting clinical pictures are complete or incomplete voluntary motor paralysis; a flaccid atrophic paralysis of upper limb muscles (if appropriate segments of gray matter are destroyed) with spastic weakness of the legs (amyotrophy with spastic paraplegia in flexion or extension); a partial or complete Brown-Sequard syndrome; and each of these with variable sensory impairment in the legs and arms. High cervical lesions may result in extreme and prolonged tonic spasms of the legs due to release of tonic myotatic reflexes. Under these circumstances, attempted voluntary movement may excite intense contraction of all flexor and extensor muscles lasting for several minutes. Segmental damage in the low cervical or lumbar gray matter, destroying inhibitory Renshaw neurons, may release activity of remaining anterior horn cells, leading to spinal segmental spasticity. Any residual symptoms persisting after 6 months are
likely to be permanent, although in a small proportion of patients some return of function (particularly sensation) is possible after this time. Loss of motor and sensory function above the lesion, coming on years after the trauma, occurs occasionally and is due to an enlarging cavity in the proximal segment of the cord.

**Central Cord Syndrome of Schneider and "Cruciate Paralysis".** In the acute central cord lesion, the loss of motor function is characteristically more severe in the upper limbs than in the lower ones and particularly severe in the hands. Bladder dysfunction with urinary retention occurs in some of the cases and sensory loss is often slight (hypermastia over the shoulders and arms may be the only sensory abnormality). The destruction of gray matter (motor and sensory neurons) may leave an atrophic, areflexive paralysis and a segmental loss of pain and thermal sensation. Retroflexion injuries of the head and neck are the ones most often associated with this central cord syndrome, but hematomyelia, necrotizing myelitis, fibrocartilagenous embolism, and possibly infarction due to compression of the vertebral artery in the medullary-cervical region are other causes.

4% of patients who survive high cervical cord injuries demonstrate what these authors refer to as "cruciate paralysis." The latter state is similar to the central cord syndrome except that the weakness is even more selective, being practically limited to the arms, a feature that is attributable to the segregation of corticospinal fibers to the arms (rostral) and to the legs (caudal) within the decussation. The patients described in the literature have had injuries, basically contusions, of the C1-C2 region. The arm weakness may be asymmetrical or even unilateral; sensory loss is inconsistent.

**Examination of the Spine-Injured Patient.** The level of the spinal cord and vertebral lesions can be determined from the clinical findings. A complete paralysis of the arms and legs usually indicates a fracture or dislocation at the fourth to fifth cervical vertebrae. If the legs are paralyzed and the arms can still be abducted and flexed, the lesion is likely to be at the fifth to sixth cervical vertebrae. Paralysis of the legs and only the hands indicates a lesion at the sixth to seventh cervical level. Below the cervical region, the spinal cord segments and roots are
not opposite their similarly numbered vertebrae. The spinal cord ends opposite the first lumbar interspace. Vertebral lesions below this point give rise predominantly to cauda equina syndromes; these carry a better prognosis than injuries to the lower thoracic vertebrae, which involve both cord and multiple roots. The level of sensory loss on the trunk, determined by perception of pinprick, is also an accurate guide to the level of the lesion, with a few qualifications. With lesions of the lower cervical cord, even if complete, sensation may be preserved down to the nipple line, because of the contribution of the C3 and C4 cutaneous branches of the cervical plexus, which innervate skin below the clavicle. Rarely, a lesion will involve only the outermost fibers of the spinothalamic pathways, sparing the innermost ones, in which case the sensory level (to pain and temperature) will be below the level of the lesion. In all cases of spinal cord and cauda equina injury, the prognosis for recovery is more favorable if any movement or sensation is elicitable during the first 48 to 72 h.

If the spine can be examined safely, it should be inspected for angulations or irregularities and gently percussed to elicit signs of bony injury. Collateral injury of the thorax, abdomen, and long bones must always be sought, to direct early radiologic studies and detect serious complications such as pneumothorax, splenic rupture, or fat embolism.

**Management of Spinal Injury.** In all cases of suspected spinal injury, the immediate concern is that there be no movement (especially flexion) of the cervical spine from the moment of the accident. The patient should be placed supine on a firm, flat surface (with one person assigned to keeping the head immobile) and should be transported by a vehicle that can accept the litter. Preferably, the patient should be transported by an ambulance equipped with spine boards, to which the head is rigidly fixed by straps. The latter provide a more effective means of immobilization than sandbags or similar objects placed on each side of the head and neck. On arrival at the hospital, it is useful to have the patient remain on the backboard until a lateral film and MRI of the cervical spine have been obtained.
A careful neurologic examination with detailed recording of motor, sensory, and sphincter function is necessary to follow the clinical progress of spinal cord injury. A common practice is to define the injury according to the standards of the American Spinal Injury Association and to assign the injury to a point on the Frankel Scale.

**Complete: motor and sensory loss below the lesion**

Incomplete: some sensory preservation below the zone of injury

Incomplete: motor and sensory sparing, but the patient is nonfunctional

Incomplete: motor and sensory sparing and the patient is functional (stands and walks)

Complete functional recovery: reflexes may be abnormal

Obviously, groups B, C, and D have a more favorable prognosis for recovery of ambulation than does group A.

Once the degrees of injury to spine and cord have been assessed, corticosteroids are given in high dosage. In patients receiving methylprednisolone (bolus of 30 mg/kg followed by 5.4 mg/kg every hour), beginning within 8 h of the injury and continuing for 23 h, Bracken and colleagues reported a slight but significant improvement in both motor and sensory function compared to controls. Naloxone was found to be of no value. Also, in a small series of patients, the administration of GM1 ganglioside (100 mg intravenously each day from the time of the accident) was found to enhance ultimate recovery to a modest degree, but this finding needs to be corroborated.

Next, radiologic examinations are undertaken to determine the alignment of vertebral bodies and pedicles, compression of the spinal cord or cauda equina due to malalignment or bone debris in the spinal canal, and the presence of tissue damage within the cord. The MRI is ideally suited to display these processes but if it is not available, or if there is uncertainty regarding cord compression, it is useful to perform myelography with CT scanning to obtain more detailed views of the spinal subarachnoid space. Instability of the spinal elements can often be inferred from dislocations or from certain fractures of the pedicles, pars articularis, or
transverse processes, but gentle flexion and extension of the injured areas must sometimes be undertaken and films obtained in each position.

If the spinal cord injury is associated with vertebral dislocation, traction on the neck is necessary to secure proper alignment and maintain immobilization. This is best accomplished by use of a halo brace, which, of all the appliances used for this purpose, provides the most rigid external fixation of the cervical spine. This type of fixation is usually continued for 4 to 6 weeks, after which a rigid collar may be substituted.

In general, concerning the early surgical management of spinal cord injury, there have been two schools of thought. One, represented by Guttmann and others, advocates reduction and alignment of the dislocated vertebrae by traction and immobilization until skeletal fixation is obtained, and then rehabilitation. The other school, represented by Munro and later by Collins and Chehrazi, proposes early surgical decompression, correction of bony displacements, and removal of herniated disc tissue and intra- and extramedullary hemorrhage; often the spine is fixed at the same time by a bone graft or wiring. Most American neurosurgeons take the less aggressive stance, delaying operation or operating only on patients with compound wounds or in those with progression or worsening of the neurologic deficit despite adequate reduction and stabilization. With complete spinal cord lesions, most surgeons do not favor surgery.

The results of the conservative and aggressive surgical plans of management have been difficult to compare and have not been evaluated with modern neurologic techniques. Collins, a participant in the National Institutes of Health (NIH) study of acute management of spinal cord injury, concluded that the survival rate was increased as a result of early surgical stabilization of fractures and fixation of the spine (in addition to the usual measures for the prevention of respiratory, urinary, and cutaneous complications and the early institution of rehabilitation measures). Other neurosurgeons, however, have not been able to document a reduction in neurologic disability as a result of early operation, and increasingly have inclined toward nonoperative management of both complete and partial spinal
cord lesions (Clark; Murphy et al). In any given case, the approach must be guided by the particular features of the patient's injuries.

The greatest risk to the patient with spinal cord injury is in the first week or 10 days when gastric dilatation, ileus, shock, and infection are the main threats to life. The mortality rate falls rapidly after the first 3 months; beyond this time, 86% of paraplegics and 80% of quadriplegics will survive for 10 years or longer. In children, the survival rate is even higher. The cumulative 7-year survival rate in spinal cord-injured patients (who had survived at least 24 h after injury) are 87%. Advanced age at the time of injury and being rendered completely quadriplegic were the worst prognostic factors.

The aftercare of patients with paraplegia is concerned with management of bladder and bowel disturbances, care of the skin, prevention of pulmonary embolism, and maintenance of nutrition. Decubitus ulcers can be prevented by frequent turning to avoid pressure necrosis, use of special mattresses, and meticulous skin care. Deep lesions require debridement and full-thickness grafting. At first continual catheterization is necessary; then, after several weeks, the bladder can be managed by intermittent catheterization once or twice daily, using a scrupulous aseptic technique. Close watch is kept for bladder infection, which is treated promptly should it occur. Bacteruria is common and does not require treatment with antibiotics unless there is associated pyuria. Morning suppositories and periodically spaced enemas are the most effective means of controlling fecal incontinence. Chronic pain (present in 30 to 50% of cases) requires the use of nonsteroidal anti-inflammatory medication, injections of local anesthetics, and transcutaneous nerve stimulation. A combination of carbamazepine and either clonazepam or tricyclic antidepressants may be helpful in cases of burning leg and trunk pain. Recalcitrant pain may require more aggressive therapy, such as epidural injections of analgesics or corticosteroids, but often even these measures are ineffective. Spasticity and flexor spasms may be
troublesome; oral baclofen, diazepam, or tizanidine may provide some relief. In permanent spastic paraplegia with severe stiffness and adductor and flexor spasms of the legs, intrathecal baclofen, delivered by a self-administered pump in doses of 12 to 400 mg/day, has reportedly been helpful. The drug is believed to act at the synapses of spinal reflexes. One must always be alert to the threat of pulmonary embolism from deep-vein thrombi, although the incidence is surprisingly low after the first several months. Physiotherapy, muscle reeducation, and the proper use of braces are all important in the rehabilitation of the patient. All this is best carried out in special centers for rehabilitation of spinal cord injuries.

**Materials for self-monitoring:**

**A. Tests.**

1. The compression of the brain belongs:
   A. subdural hematoma  
   B. linear fracture of the skull  
   C. Starry cranial fracture  
   D. contusion of the brain  
   E. concussion of the brain

2. Concussion of the brain refers to biomechanism:
   A. shock-shockproof type  
   B. type acceleration-deceleration  
   C. The hydrodynamic type  
   D. Cavitation type  
   E. there is not a single correct answer

3. A bruised brain contusion of light degree requires treatment:
   A. Conservative  
   B. Surgical  
   C. Radial  
   D. chemotherapeutic  
   E. does not require treatment

4. Impaired consciousness with concussion of the brain observed:
   A. to 20 minutes.  
   B. 30-60 minutes.  
   C. 1-2 hours.  
   D. Day 1-2  
   E. 10-12 hours.

5. In the clinic of a mild severity bruised brain is main:
A. Focal symptomatology
B. Impairment of vital functions
C. General cerebral symptoms
D. Radicular symptoms
D. All answers are correct

6. In the clinic, a moderate level of brain contusion is prevalent:
A. Focal symptomatology
B. Disruption of vital functions
C. Cerebral symptomatology
D. Radicular symptoms
E. All the answers are correct

7. In the clinical picture, a severe brain contusion is prevalent:
A. Focal symptomatology
B. Impairment of vital functions
C. General cerebral symptoms
D. Radicular symptoms
E. All the answers are correct

8. For a severe brain contusion, a characteristic loss of consciousness:
A. To 20 minutes.
B. 30-60 minutes.
C. 1-2 hours.
D. A few hours and more
E. 10-12 hours.

9. Diffuse axonal injury by biomechanism:
A. Shock-shockproof type
B. Type acceleration-deceleration
C. The hydrodynamic type
D. Cavitation type
E. There is not a single correct answer

10. Depressed fracture by the type of ball for ping-pong characteristic for:
A. Children
B. People of advanced age
C. Older people
D. Teenagers
E. There is not a single correct answer

11. Causes of cerebral compression:
A. Epidural hematoma
B. Subdural hematoma
C. Crushed fracture of the skull bones
D. Pneumocephaly
12. Methods of neuroimaging:
A. REG
B. EEG
C. ECG
D. MRI
E. Angiography

13. For a concussion of the brain is characteristic:
A. no change in CT
B. presence of a fracture of the bones of the cranial vault
C. the presence of blood in a general analysis of cerebrospinal fluid
D. The presence of vital disorders
E. all answers are correct

14. For a mild brain injury on CT, it is characteristic:
A. no change
B. hypodense focus
C. subdural hematoma
D. Hyperintensive focus
E. intracerebral hematoma

15. Cerebral edema can have
A. local character
B. hemispheric character
C. Generalized nature
D. answer a, b, c, faithful
E. there is not one correct answer

**VST**
1. Where is the 2nd motor neuron located?
A) anterior central gyrus
B) anterior horn of the spinal cord
C) spinal cord node
D) posterior horn of spinal cord
E) the nuclei of Gauill and Burdach

2. The patient has a peripheral paresis of the feet, imperative urges to urinate, impaired sensitivity in the crotch and medial thighs. Where is the focus of defeat?
A) transverse lesion of the spinal cord at the level of the cervical region
B) half of the spinal cord injury to the right
C) half of the spinal cord lesion on the left
D) ponytail
E) cone of the spinal cord

3. 10 days of traumatic disease SC refer to:
   A) Acute period
   B) Early period
   C) Interim period
   D) Late period
   E) There is not a single correct answer

4. The development of automatism in the spinal cord, located below the level of lesion, occurs during
   A) Acute period
   B) Early period
   C) Interim period
   D) Late period
   E) There is not a single correct answer

5. The amplitude of the neck in the projection of flexion-extension is:
   A) 170°
   B) 150°
   C) 120°
   D) 100°
   E) 90°

6. Absence of reflexes in the acute period of PSMT, instability of hemodynamics, with a bruise at the level of the cervical spinal cord is explained
   A) Only high levels of damage
   B) Instability of damage
   C) Ligamentous lesion
   D) Spinal shock
   E) There is not a single correct answer

7. On the Frankel scale, patients with incomplete sensitivity disorders are below the level of trauma, there are movements, muscle strength is sufficient for walking with outside help, belong to the group:
   A) A
   B) B
   C) C
   D) D
   E) E

8. When the spinal cord is damaged at the level of the cervical region, typical impairment of urination by type
   A) Delays
   B) Incontinence
C) There are no violations
D) Paradoxical ishuria
E) There is not a single correct answer

9. In clinical forms is divided into
A) Concussion SC
B) Bruise
C) Compression of a SC
D) Damage to the cauda equina
E) all the answers are correct

10. The evaluation of the functional state of spinal cord is performed on a scale
A) Glasgow
B) Hanta Hessa
C) Frankel
D) Milesi
E) all the answers are correct

11. Indications for early decompression of SC is:
A) Severity of the general condition of the patient
B) There is a disturbance of consciousness
C) There is a dislocation of the brain
D) Increase in spinal cord dysfunction
E) Damage to the anterior longitudinal ligament

12. For the defeat of spinal cord at C4 is characterized by:
A) Diaphragmatic breathing
B) Tetraparesis
C) Impaired urination by type of delay
D) Durability of the conductor type
E) all the answers are correct

13. An indication for early decompression of spinal cord is
A) The increase in secondary respiratory failure as a result of the ascending edema of the cervical spinal cord
B) Damage to the rear support complex
C) Damage to anterior longitudinal ligament
D) Compression fracture of vertebral body I st.
E) all the answers are correct

14. The indication for early decompression of SC is
A) Damage to the rear support complex
B) Motor aphasia
C) Damage to anterior longitudinal ligament
D) Brachiofascial homolateral paresis
E) partial or complete blockade of the cerebrospinal fluid of the spinal cord

15. The length of the SC is on the average
A) 43-45cm
B) 35-40cm
C) 40-43cm
D) 45-50cm
E) There is not a single correct answer

B. Situational tasks
1. Patient A, 67 years, a long time suffers an idiopathic hypertensia, to the doctor was not converted, has entered in hospital in a grave condition with acute disturbance of a cerebral circulation. In the neurologic status the quick aphasia, a right-hand penetrating hemiparesis, consciousness level - the penetrating deafenation is observed. To specify vascular basin in which there is a circulation disturbance, tactics of the subsequent conducting the patient.

2. Patient B, 34 years, is delivered by brigade in a reception from a road accident place. The skin and visible mucosas acyanotic, acrocyanosis, bubbling rales are spotted distantly, breath superficial, at a thorax palpation the crepitation is spotted, at a palpation of pelvic bones - pathological motility. BP 80/40 mm hg, Pulse 130 in minute the Abdomen slightly exerted. To painful stimuliuses of the patient does not react, the tonus of muscles is reduced, reflexes without an accurate odds of the legs, meningeal symptoms do not cause, the photoreaction of pupils is reduced, corneal jerk is maintained, speech contact misses. Locally - scalped wound of the right frontally-parietotemporal field, with presence of a moderate bleeding, plural grazes of a trunk, the extremities, the expressed paraorbital hematomas of both eyes, with presence chemosis, traces of a stomatorrhagia, a nose, the right ear. Define gravity of a state of the patient, level of disturbance of consciousness, set the previous diagnosis and algorithm of granting of the help.

3. Patient B, 74 years, is delivered by brigade СМП in a reception, is found in the street. A skin and visible mucosas pale, acrocyanosis, breath superficial, ЧДР 40. The BP 170/100 mm. hg, Pulse 62 in minute. On painful stimuliuses of the patient does not react, the tonus of muscles is reduced, reflexes D> S, meningeal symptoms do not cause, pupils D <S the photoreaction is reduced, corneal jerk is maintained, speech contact misses, response to a pain misses. Local - a chin graze. A nucha muscle tension (2p/p), c-m of Kernig (+++) from 2 sides. Define consciousness level, gravity of a state of the patient, the list of necessary surveys to (prove), tactics of conducting the patient, the topical diagnosis.

4. The young man of 18 years is delivered in a reception after road accident. In the consciousness, completely oriented, but witnesses reporting about the short season of a syncope after a trauma. On a craniography fracture of the left temporal bone
becomes perceptible. After roentgenography the patient subitaneously loses consciousness and the doctor notes dilatation of the left pupil. What most reliable diagnosis? What, in your opinion, has caused an aggravation of symptoms of the patient?

5. The 25-year-old patient complains of a headache, vomiting by "fountain". Was ill within a week. Has ceased to walk in connection with sharp unsteadiness, sight was broke. Objectively: an atony, ataxy, a nystagmus. In posture of Romberg drops to the left. The Finger-nose test carries out with intentional tremor, miss from both legs. The most probable diagnosis?

6. During a lumbar puncture the 4-year-old child with suspicion on a brain tumour had an intensive headache in a nucha, rotation nystagmus, vomiting, a dysarthria, pathological reflexes from two legs, breath and pulse disorder. What educes at the child?

7. The patient of 65 years, going in the winter in the morning on slippery road, has fallen, has strongly hit a nucha, there was a transient loss of consciousness. Home has returned independently with a moderate headache. Towards evening the headache has considerably strengthened, there was a vomiting, the patient became sleepy, there was a delicacy and a numbness in the right extremities, was observed Jackson's epilepsy attack. He was hospitalised in neurosurgical unit where the acute subdural hematoma is diagnosed. What treatment in this case should be made quickly to salvage life to the patient?

8. The patient of 36 years as a result of road and transport incident has gained a serious craniocerebral trauma, is in a coma. At M-ehoentsefalografi the right on 12 mm. On a computer tomography shift of median structures of a brain is found in a parietofrontal field at the left the subdural hematoma in the dimension 6x7x7 is found. What most correct tactics of treatment see?

9. Patient M, 60 years, in the evening, after operation has felt a headache, has fallen, has lost consciousness. From the anamnesis it is known: 15 years are ill with an idiopathic hypertensia. Objectively: the BP of 200/100 mm hg, pulse 80 in minute Consciousness misses. Right nasolabial fold is smoothed. Tendinous and periosteal disorders are not observed. A muscular tonus low. During survey in the left extremities the minimum locomotions are noted.

10. During carrying out of trepanation of a brain in a frontotemporal field on the right the patient age of 48 years, at an access stage has an intensive bleeding against rising of arterial pressure. You know what methods of a stopping of a bleeding, and at what stages of carrying out of bone-plastic trepanation they are used.
11. The patient of 36 years as a result of road and transport incident has gained a craniocerebral trauma. At survey consciousness level on Glasgo scale - 11, hemodynamically stable, breath independent, vesicular, is auscultated from both legs, in the neurologic status: a link sided hemiparesis, roentgenologic damages of bones of a calvaria it is not revealed, the ECHO-ES without shift. Define gravity of a state of the patient, subsequent tactics of conducting.

12. The 7-year-old child is delivered in clinic after accident with fracture of the upper jaw. Losing consciousness, the child tore. In 2 hours has regained consciousness, answered a question. In 4 hours the sopor has educed. Pulse 180 / minute Convulsive cuttings of the left extremities. An anisocoria. What diagnosis can be admitted in this case?

13. The patient of 60 years, is delivered from natural apartment by brigade, at survey the patient in consciousness, the treatment answers truly, is oriented in a place and space, complains of the expressed headache, is promptly exhausted, was ill acutely after a psychoemotional strain. Somatic - without an acute pathology. In the neurologic status - an easy paresis in the right arm, speech is maintained. Define gravity of a state of the patient, subsequent tactics of conducting.

14. The patient of 24 years has fallen to feet from a balcony 4 floors. Has felt a sharp dorsodynia through which self-contained could not rise. At survey the lordosis smoothness in lumbar department of a column becomes perceptible. Locomotions in lumbar department of a column are sharply circumscribed. Thrust force on a column the positive. A palpation of acanthas painful, especially 12-thoracal. Sensitivity and locomotions of the inferior extremities are not broken. What most reliable diagnosis?

15. The patient of 24 years during diving in the river has felt delicacy, a numbness in extremities which have occured after impact by a head in river bedrock. Has got out on coast by means of associates. At hospitalisation at the patient a skin light pink, breath is relaxed in n/departments, a BP of 100/70 mm hg, pulse of 84/minute, a gaste soft, takes over restricted participation in the breath certificate, the emiction does not check. In the neurologic status - the upper paraparesis, the inferior paraplegia, a hypesthesia from level 5 thoracal segments, the penetrating sensitivity miss. Specify the diagnosis, define gravity of a state of the patient, subsequent tactics of conducting.

VST
1. A 16-year-old patient was taken by an ambulance. Disturbing headache, nausea, pain in the lower parts of the thoracic spine, weakness in the legs. From an anamnesis it is found out, that the patient has fallen from a tree, from height of 4 meters. The condition is satisfactory. Reflexes from the lower extremities are
absent, anesthesia on the conductor type from the level of D12, the retention of urination. Establish a diagnosis, schedule an examination.

2. The patient is 17 years old, was taken by ambulance from the river. According to the accompanying, the victim dived from the tree into the river, as a result of an unsuccessful jump hit his head on the bottom of the river. Neurological status: reflexes tendon and periosteal from the extremities are not caused, impaired sensitivity (surface) on the conductor type from the level of D1, delay urination. Consciousness is preserved, the questions are answered correctly, the forced position of the head and neck. Establish a diagnosis, assign a checkup.

3. A 27-year-old patient was taken to a hospital after a car accident with complaints of pain in the lumbar spine with irradiation in the legs, impossibility of free movements in the spine. Neurological status: Achilles reflexes are lowered, sensitivity in a site of a perineum is broken. Hypotension in the muscles of the lower limbs, urination disorder - urinary incontinence. Establish a diagnosis, prescribe treatment and paraclinical methods of research.

4. Patient 30 years old, taken from the street after a car crash with complaints of pain in the cervical spine, lack of movements in the hands and feet. Neurological status: tetraplegia, reflexes from the hands and feet are not caused. Hypesthesia in the conductor type from the level of C8-D1, urinary retention. Consciousness is preserved, the patient is in contact. After an hour the patient's condition improved significantly: the sensation of numbness disappeared, and urination resumed. Until the end of the first day, movements in the limbs resumed. In the neurological status there were reflexes from tendons and periosteal, sensitivity was renewed. Establish a diagnosis, prescribe treatment, survey methods.

5. The patient is 20 years old, athlete, was taken to the hospital with complaints of sharp pain in the neck with irradiation in the right hand, weakness in the right hand, numbness in her, slight weakness in the right leg. Complaints appeared after a failed exercise (falling on the back). In a neurological status: a patient in consciousness, contact. Movement in the cervical spine is severely limited, forced position of the head. Paresis of the right hand of the middle degree, reduction of strength in the right leg to 4 points. Reflexes on the right are reduced. Segmental hyphesis of the right upper limb. Establish a diagnosis, assign additional methods of examination and treatment.

6. A 24-year-old patient fell to his feet from the balcony of the 4th floor. He felt a sharp pain in his back, through which he himself could not get up. When examined, the lordosis is flattened in the lumbar spine. Movement in the lumbar spine is severely limited. Axial load on the spine is positive. Palpation of the osteitis processes is painful, especially the 12-thoracic. Sensitivity and movements of the lower limbs are not violated. What is the most reliable diagnosis?
7. A 24-year-old patient during diving into the river felt a weakness, numbness in the limbs that appeared after striking his head in the river bed. I got ashore with the help of others. When hospitalized, the patient's skin is pale pink, breathing is weakened in parts, blood pressure 100/70 mm Hg, pulse 84 / min, abdomen is mild, takes limited part in the act of breathing, does not control urination. In the neurological status - upper paraparesis, lower paraplegia, hypoesthesia from the level 5 thoracic segment, deep sensitivity is absent. Painful palpation of the spinous process of the C6 vertebra. Specify the diagnosis, determine the severity of the patient's condition, the subsequent tactics of reference.

8. The patient is 34 years old, entered the admissions department, the trauma was received as a result of diving into the river. I got out of the water myself. Complaints of pain in the cervical spine, weakness in the hands. There are no injuries to the spine on the X-ray. Your preliminary diagnosis, examination, treatment.

9. The patient is 40 years old, taken from the street after a car crash with complaints of pain in the cervical spine, lack of movements in the hands and feet. Neurological status: tetraplegia, reflexes from the hands and feet are not caused. Hypoesthesia in the conductor type from the level of C8-D1, urinary retention. Consciousness is preserved. On the X-ray of the spine there is a compression fracture of C6, The configuration of the spinal canal is not broken. Establish a diagnosis, prescribe treatment, survey methods.

10. A 30-year-old patient, a sportsman, was taken to the hospital with complaints of severe pain in the neck with irradiation in his right hand, weakness in his right hand, numbness in her, slight weakness in his right leg. Complaints appeared after a failed exercise (falling on the back). In a neurological status: a patient in consciousness, contact. Movement in the cervical spine is severely limited, forced position of the head. Paresis of the right hand of the middle degree, reduction of strength in the right leg to 4 points. Reflexes on the right are reduced. Segmental hypoesthesia of the right upper limb. Radiographically there is a subluxation of the C5 vertebra. Establish a diagnosis, assign additional methods of examination, tactics of patient management.

11. The patient B, 43 years old, applied for medical help with complaints of pain in the cervical spine. According to the patient, the trauma was caused by a blow to the neck about a week ago. Consciousness did not lose. Objectively: focal pathology is absent, soreness in palpation of the cervical spine, meningeal (with Kernig not caused), the patient takes a forced position with the head thrown back, radiologically pathology of the cervical spine and skull not found. What kind of pathology can you think of in this case? What methods of examination should be used?
12. The patient, 56 years old, was taken to the reception room after an accident, his consciousness was lost for a short time. On examination: in consciousness, available to productive contact, complains of pain in the neck, movements in the legs are absent, in the hands only in the proximal sections, anesthesia from the D4 level. Your diagnosis, which examinations should be carried out?

13. The patient is 40 years old, taken from the street after a car accident with complaints of pain in the cervical spine, lack of movements in the hands and feet. Neurological status: tetraplegia, reflexes from the hands and feet are not caused. Anesthesia by the conductor type from the level of C8-D1, delayed urination. Consciousness is preserved. On the X-ray of the spine there is a compression fracture of C6 I st., The configuration of the spinal canal is not broken. Assess the functional state of the spinal cord.

14. A 17-year-old patient was taken by ambulance from the river. According to the accompanying, the victim dived from the tree into the river, as a result of an unsuccessful jump hit his head on the bottom of the river. Neurological status: reflexes of tendons and periostal from the extremities are not caused, impaired sensitivity (surface) on the conductor type from the level of D1, delay of urination, deep sensitivity is preserved. Consciousness is preserved, the questions are answered correctly, the forced position of the head and neck. Assess the functional state of the spinal cord.

15. A 24-year-old patient fell to his feet from the balcony of the 4th floor. He felt a sharp pain in his back, through which he himself could not get up. When examined, the lordosis is flattened in the lumbar spine. Movement in the lumbar spine is severely restricted. Axial load on the spine is positive. Palpation of spinous processes is painful, especially 12-thoracic. Sensitivity and movements of the lower limbs are not violated. Assess the functional state of the spinal cord.

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Methodical development is revised and ratified on meeting of department nervous illnesses

with additions (by changes)

Head of the Department of Nervous Diseases with Neurosurgery and Medical Genetics
D.Med.Sci., Professor M.Yu.Delva