THE MINISTRY OF HEALTH OF UKRAINE
ZAPORIZHZHIA STATE MEDICAL UNIVERSITY
Department of nervous diseases

NEUROLOGY IN TABLE
(*General neurology*)

for practical employments for the students of the
4th course of II international faculty
speciality "General medicine" English medium of instruction

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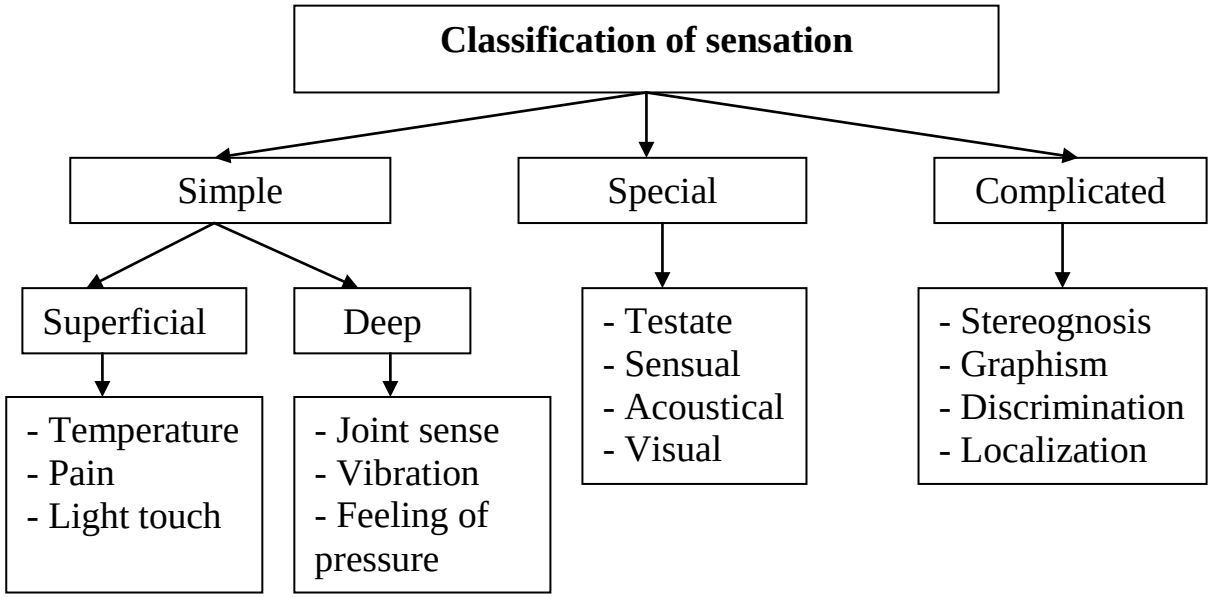
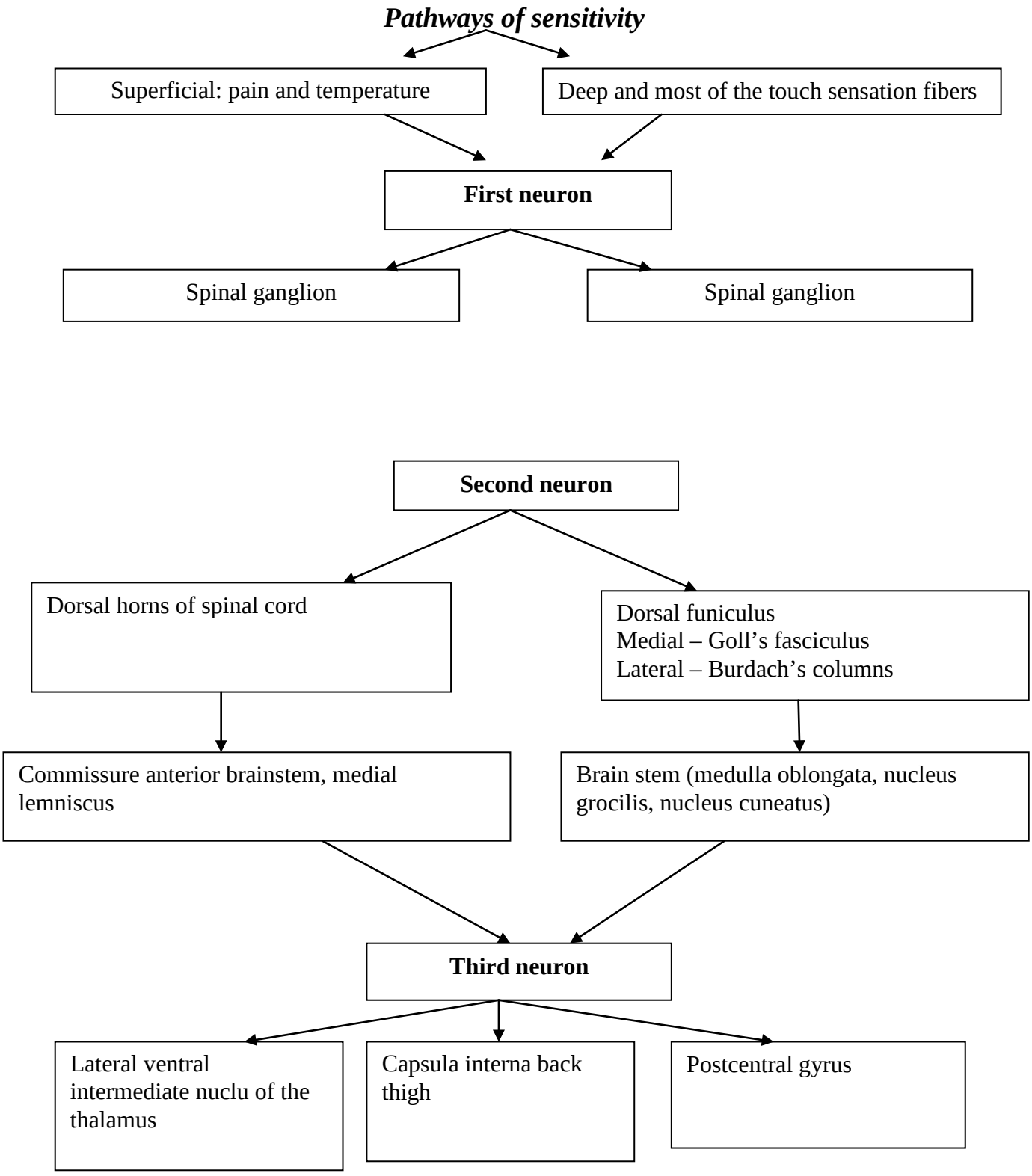
CONTENTS

1. SENSITIVE FUNCTION

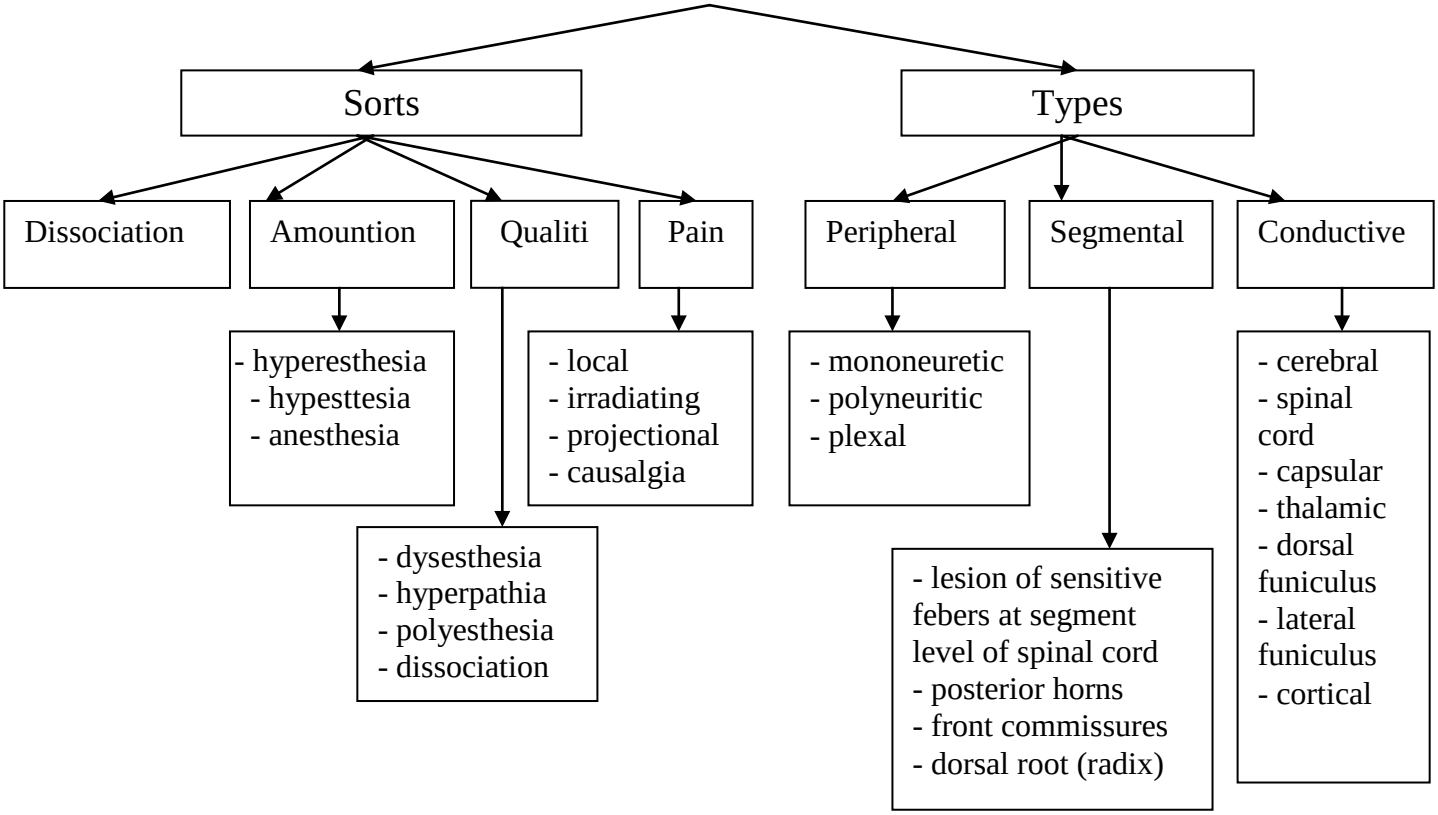
The human capacity to feel the impact of various exogenous and endogenous factors on his/her receptor apparatus is called the **sensation.**

Classification of sensation

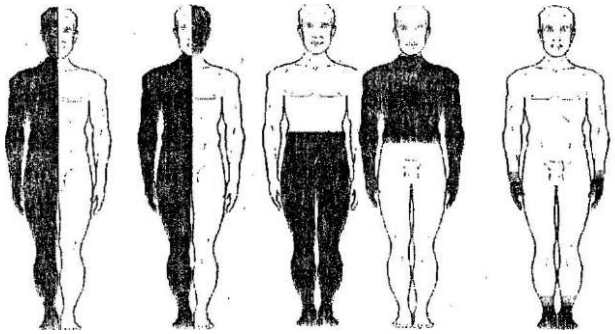




Sorts and types of sensory disorders

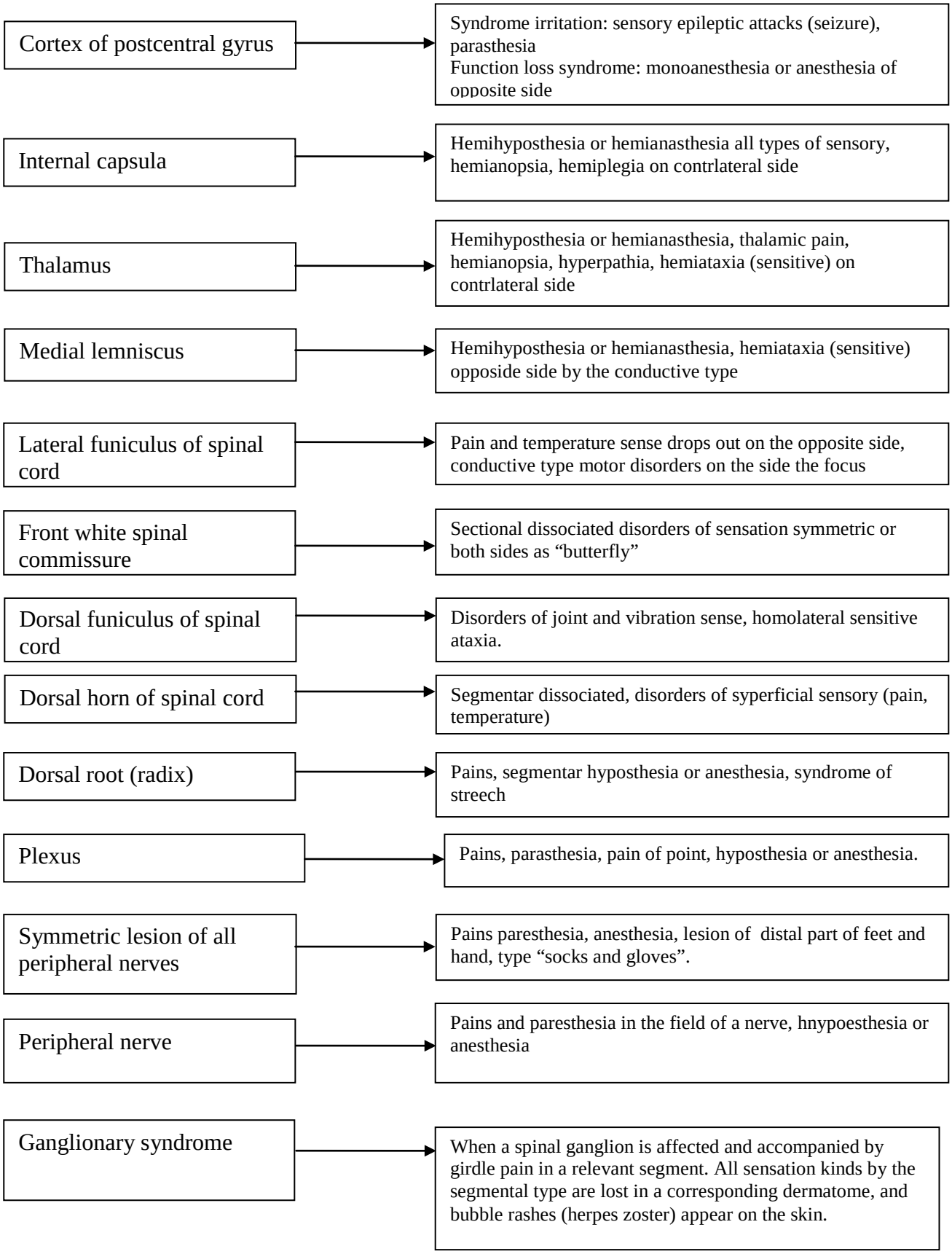


Types of sensitive disorders



1 – conductive – hemianesthesia
2 – conductive – alternative hemianesthesia
3 – conductive
4 – segmental
5 – peripheral

Syndrome's defeat of sensory


Cortex of postcentral gyrus
Syndrome irritation: sensory epileptic attacks (seizure), parasthesia
Function loss syndrome: monoanesthesia or anesthesia of opposite side
Internal capsula
Hemihyposthesia or hemianasthesia all types of sensory, hemianopsia, hemiplegia on contrlateral side
Thalamus
Hemihyposthesia or hemianasthesia, thalamic pain, hemianopsia, hyperpathia, hemiataxia (sensitive) on contrlateral side
Medial lemniscus
Hemihyposthesia or hemianasthesia, hemiataxia (sensitive) opposide side by the conductive type
Lateral funiculus of spinal cord
Pain and temperature sense drops out on the opposite side, conductive type motor disorders on the side the focus
Front white spinal commissure
Sectional dissociated disorders of sensation symmetric or both sides as “butterfly”
Dorsal funiculus of spinal cord
Disorders of joint and vibration sense, homolateral sensitive ataxia.
Dorsal horn of spinal cord
Segmentar dissociated, disorders of syperficial sensory (pain, temperature)
Dorsal root (radix)
Pains, segmentar hyposthesia or anesthesia, syndrome of streech
Plexus
Pains, parasthesia, pain of point, hyposthesia or anesthesia.
Symmetric lesion of all peripheral nerves
Pains paresthesia, anesthesia, lesion of distal part of feet and hand, type “socks and gloves”.
Peripheral nerve
Pains and paresthesia in the field of a nerve, hnypoesthesia or anesthesia
Ganglionary syndrome
When a spinal ganglion is affected and accompanied by girdle pain in a relevant segment. All sensation kinds by the segmental type are lost in a corresponding dermatome, and bubble rashes (herpes zoster) appear on the skin.

Methods of sensitive function study

Exploring a sensation, a doctor gets subjective information from a patient about his/her feelings that arise during irritation of the receptor apparatus. Therefore, it is necessary to adhere to certain conditions during the study. The study should be carried out in a quiet atmosphere, in a warm room, with a patient's eyes closed. Irritations should be inscribed on symmetric sections, have the same force and duration, they should be conducted with different intervals, compared the sensation of irritants with "sick" and "healthy" areas.

During delimitation of sensitive disorders, one should be aware of some guidelines: the clavicle's level is approximately equal to the C3 segment; the level of the nipples is equal to the T5; the level of the rib arch — to the T7; the level of the navel — to the T10; the inguinal fold level — to the T12 segment.

Study of superficial sensation. Needle is used in order to test *pain sensation. A temperature sense* is studied with using test tubes filled with hot and cold water. *A Touch sense* is investigated with using a piece of cotton or paper, with which the skin is abruptly touched.

Study of deep sensitivity. The proprioception and vibration are usually studied in clinical practice. *The proprioception* is tested by performing the passive movements of small amplitude on small joints of the hands, and then on the patient's legs, who is lying with the closed eyes. If any disorders of sensation are found out in small joints (in the distal phalanxes of fingers), it is required to move to larger. A vibration sense should be checked with a tuning fork, which foots put on the bony prominences of the limb and define the time during which the patient feels vibration. Normal duration of a vibration sensation is 14-16 sec.

Test and typical task

1. Where is the III neuron (deep sensation) localized?

A. Brainstem.

B. Dorsal horns.

C. Thalamus.

D. Holl’s and Burdach’s nuclear of oblong brain.

2. This analgesia:

A. Loss of deep joint sense.

B. Loss of pain sense.

C. Lowering of sensation.

D. Loss of a temperature.

3. What reflex behaves to superficial?

A. Scapula-humeral.

B. Palatal.

C. Triceps.

D. Achilles (ankle jerk).

4. What types of sensation belong to deep?

A. Pain sense.

B. Touch sense

C. Joint sense.

D. Localization.

5. The thalamic pattern (at a lesion of thalamus) is observed:

A. Hemihypoesthesia.

B. Sensory convulsive attacks.

C. Superfacial reflex absent.

D. Lession sensation «jacket» type.

6. Patient has stroke, localization of internal capsule. What clinical sign?

A. Hemianesthesia, hemianopsia, hemiplegia.

B. Hemianesthesia sensitive hemiataxia, hemianopsia.

C. Monoanesthesia on the opposite side.

D. Polyneuropathy.

7. At engaging in the pathological process of the second branch of trigeminal nerve can be been in pain in the area of innervation of the third branch (lower jaw). What type of pain is characteristic?

8. The patient has defeat sensitivity: paresthesia in half face on the left side. Where is the focus of irritation localized?

9. At a patient, suffering during 10 years diabetes, produces complaints about numbness, sense of «crawl of small ants» in the distal parts of extremities. Objectively: violation of sensitivity on the type of «socks» and «gloves». What type of violation of sensitivity?

10. At a patient a pain and temperature sensitivity is absent, touch sensitivity not broken. Where is the pathologic focus localized?

List of answers: 1.C. 2.D. 3.D. 4.C. 5.A. 6.A. 7.Irradiating pains. 8. The postcentral gurus, middle part. 9. Polyneuritis type. 10. Dorsal horns of spinsl cord.

2. REFLEX-MOTOR FUNCTION OF THE NERVOUS SYSTEM. SYNDROMES OF MOVEMENT DISORDERS

Reflex actions are the simplest form of movement. **A reflex action is a stereotyped response to a specific sensory stimulus.** The reflex elicited depends on the site of the stimulus and the strength of the stimulus determines the amplitude of the response. Reflex responses are used by higher motor centers to generate more complex movements and behaviors. The neural circuitry responsible for reflex actions is present at different levels of the motor system and disturbances in these reflexes are important for localizing lesions in the motor system.

All reflexes are divided into unconditioned and conditioned ones. Unconditioned (instinctive, inborn) reflexes are an inborn motor reaction, phylogenetic old, under cortex regulative influence and are the basis of conditioned reflexes. Inborn reflexes are closed in the spinal cord, brainstem and basal ganglia. Conditioned reflexes are closed in the brain cortex and lay the foundation to higher nervous functions.

Methods of the inborn reflexes study

To master the methods of the inborn reflexes study fulfill the reflexes study in the following sequence. The hammer strikes are performed with equal force. Pay attention if the below mentioned normal reaction is achieved:

► *corneal reflex*—a carefully touch of the cornea above the iris (not above the pupil) with a soft paper stripe leads to lids closing

► *pharyngeal reflex*— a touch of the posterior pharyngeal wall with a spatula leads to swallowing or coughing movements occur,

► *palatal reflex*— touch the soft palate with a spatula leads to the soft palate elevation,

► *mandibular (jaw) reflex*— strike your index finger put on the patient's mandible with a hammer (mouth is half-open) leads to the mandible elevation,

► *flexor ulnar (Biceps) reflex* — half-bended arms are placed on the patient's abdomen. Press the arm biceps muscle tendon with your left pollex. Strike your pollex nail with a hammer — as a result forearm flexion will appear,

► *extensor ulnar (Triceps) reflex* — a patient's arm is bent under an obtuse angle. Strike the arm triceps muscle tendon (2 cm above the ulnar process) with a hammer — as a result forearm extension will appear,

► *brachioradial (carporadial) reflex*— patient's arms are bent in the ulnar joint under an obtuse angle, they are half-proned and placed on the abdomen. Strike the radius styloid process with a hammer and arm flexion in the ulnar joint, fingers pronation and flexion will occur,

► *abdominal reflexes* — make a quick hatched irritation of the abdominal skin with a pointed object from the peripheral to the middle lower the costal arches (superior), on the umbilical level (middle), above the fallopian ligament and abdominal wall muscles contraction will occur,

► *knee (Quadriceps) reflex* — a patient's legs are half-bent in his knee joints. Place your left arm under the patient's joints. Strike the thigh quadrate muscle tendon

under the kneecap with a hammer — as a result legs extension in the knee joint will appear,

► *Achilles reflex*— a patient's leg is bent in the hip and knee joints. Strike the Achilles tendon with a hammer and foot plantar extension will occur,

► *plantar reflex* — perform a hatched skin irritation of the sole external edge with a blunt object and toes plantar flexion will occur.

An interruption of the reflex arches at any point weakens or abolishes the reflex. Reflexes changes:

► areflexia — absence of reflex,

► hyporeflexia — decrease of reflex,

► hyperreflexia — increase of reflex,

► anisoreflexia -— different expression of symmetric reflexes.

Pathological reflexes

Some reflexes, especially spinal and brainstem reflexes are normally observed or elicited only in the developing nervous system. As the nervous system and higher motor centers get mature, these reflexes are suppressed, only to reemerge if damage of the higher motor centers modulates the reflex. Reflexes that can he elicited only in the diseased state are called *pathological reflexes.* It indicates dysfunction of the pyramidal (corticospinal) tract.

Pathological reflexes on feet

extensor	**flexor**
- Oppenheim	- Zhukovski’s
- Gordon	- Rossolimo
- Babinski’s	- Bechterew
- Shaffer	
- Chaddock	

Oral automatism reflexes:

- nasal-lip (nasolabial),
- lip,
- palmar-chin (palmomental),
- distant-oral.

Pathologic synkinesises:

- global,
- imitative,
- coordinative.

Defense (protective) reflexes (withdrawal leg).

Methods of pathological reflexes study

Exploring the extensor group foot pathological reflexes:

- *Babinski reflex*— make a hatched skin irritation of the plantar external edge, slow hallux (great toe) extension with a flaccid separation of other toes will occur,
- *Oppenheim reflex*— same response to a downward stroke of the examiner's thumb on the patient's shin,
- *Gordon reflex*— the same response to squeezing of the calf muscles
- *Sheffer's reflex*— press the Achilles tendon, slow hallux extension with a flaccid separation of other toes will occur.

Exploring the flexor group foot pathological reflexes:

- *Rossolimo reflex*— strike easily with your fingers on the plantar surface of the terminal phalanges of the patient's II-IV toes, quick plantar flexion of the toes will occur.
- *Becliterew reflex*— strike with a hammer on the dorsal foot above III-IV metatarsal bones, quick plantar flexion of toes will occur,
- *Zhukovski reflex* — strike with a hammer on the sole under the toes, quick plantar flexion of the toes will occur.

Exploring the oral automatism:

► *nasal-lip reflex*— strike easily with a hammer on the nose root, the lips are stretched ahead,

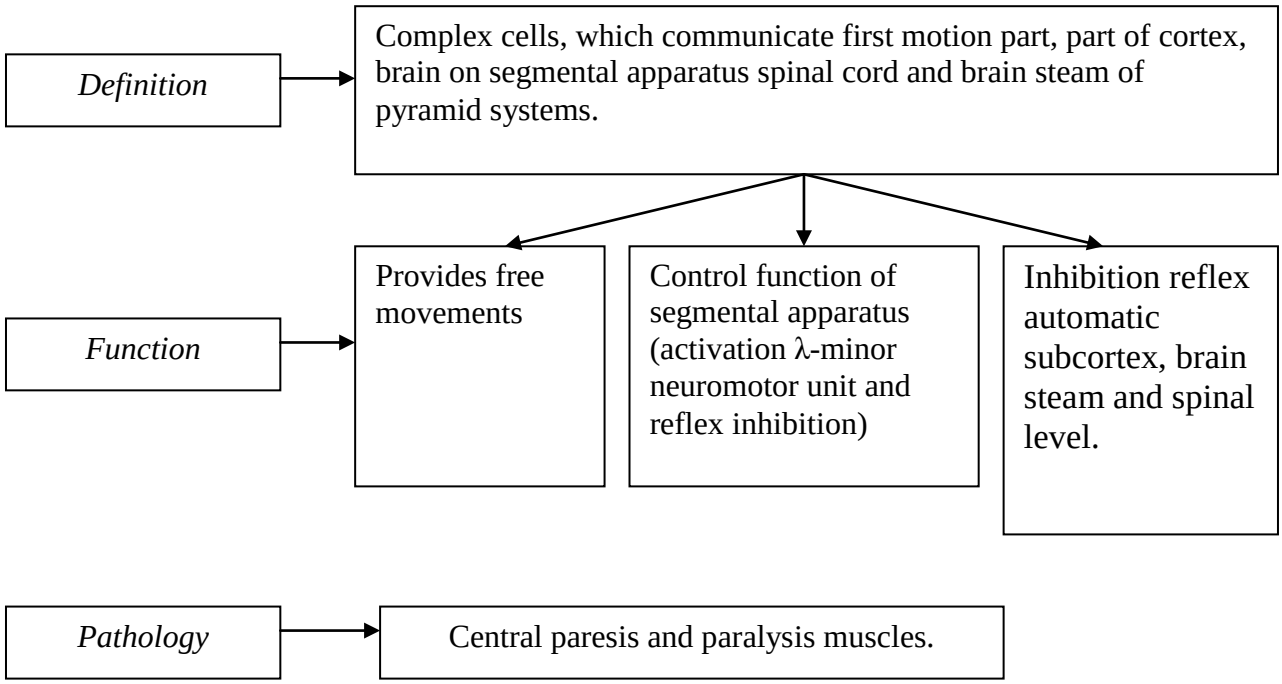
► *lip reflex*— strike easily with a hammer on the lips, the lips are stretched ahead,

► *palmar-chin reflex*— make a hatched irritation of the palmar skin over the thenar, chin muscles at the same side are contracted.

The levels of the spinal reflex arches locking

Sorts of reflex	Reflex	Reflexes arch	Level locking
From mucosa	Corneal	n.n.optic facialis	Pons of brain
	Conjunctival	n.n. optic facialis	Pons of brain
	Pharyngeal	n.n.glossopharyngeal vagus	Medulla oblongata
	Palatal	n.n.glossopharyngeal vagus	Medulla oblongata
Cutaneous	Abdominal	Intracostal nerves	Thoracica part of spinal cord
	Upper	Th7-Th8	
	Middle	Th9-Th10	
	Lower	Th11-Th12	
	Cremasteric	n.n.genitofemorales	Segments of spinal cord L5-S1
	Plantar	n.n.ishiadicus	Segments of spinal cord L5-S1
	Anal	n.n.anococcygei	Segments of spinal cord S4-S5
Deep tendon reflex	Flexor ulnar (biceps)	n.musculocutaneus	Segment C5-C6
	Extensor ulnar (triceps)	n.radialis	Segment C7-C8
	Knee	n.femoralis	Segment L3-L4
	Achilles (ankle)	n.femoralis	Segment S4-S5
Deep periostal reflex	Sypraorbital	n.n.optic facialis	Pons of brain
	Brachioradial	n.n.medianus radialis, musculocutaneus	Segments C5-C8
	Scapulohumoral	n.sybscapularis	Segment C5-C6
	Mandibular lower	n.trigemenal n.mandibular lower	Pons of brain

PYRAMIDAL SYSTEM



Pathway of pyramidal system

(tractus corticospinalis lateralis, anterior)

First neuron localization: upper motor neuron (central neuron)

Layer 5 in primary motor cortex contains distinctive giant pyramidal neurons Betz cells

↓

Capsula interns back thigh

↓

Brain steam (mesencephalon, pons, medulla oblongata)

↓

Lower part of medulla oblongata decussating pyramidal

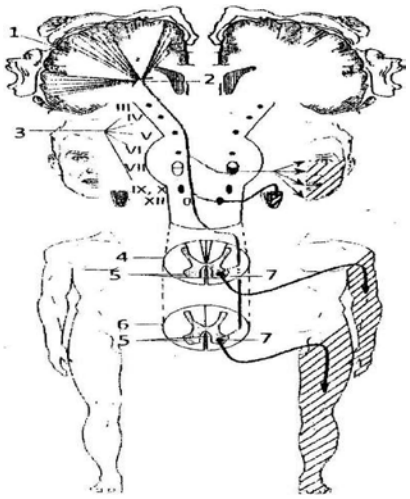
90% tractus corticospinalis lateralis (decussating)	10% tractus corticospinalis anterior (not decussating)
↓	↓
Lateral funiculus	Anterior funiculus

Second neuron lower motor neuron (peripheral neuron): anterior horns

Corticonuclear pathway

Corticonuclear fibers destined for the motor nuclei of the cranial nerves leave the corticospinal tract in the brainstem. Muscles of the head, except for the lower facial muscles (VII cranial nerve) and tongue (XII cranial nerve), receive both crossed and uncrossed corticonuclear fibers. Therefore, as a rule, in a patient with a lesion of the corticonuclear tract on one side, one seldom sees significant weakness of the jaw, pharynx, or larynx. The motor nuclei of cranial nerves VII (lower portion) and XII receive contralateral cortical innervation only.

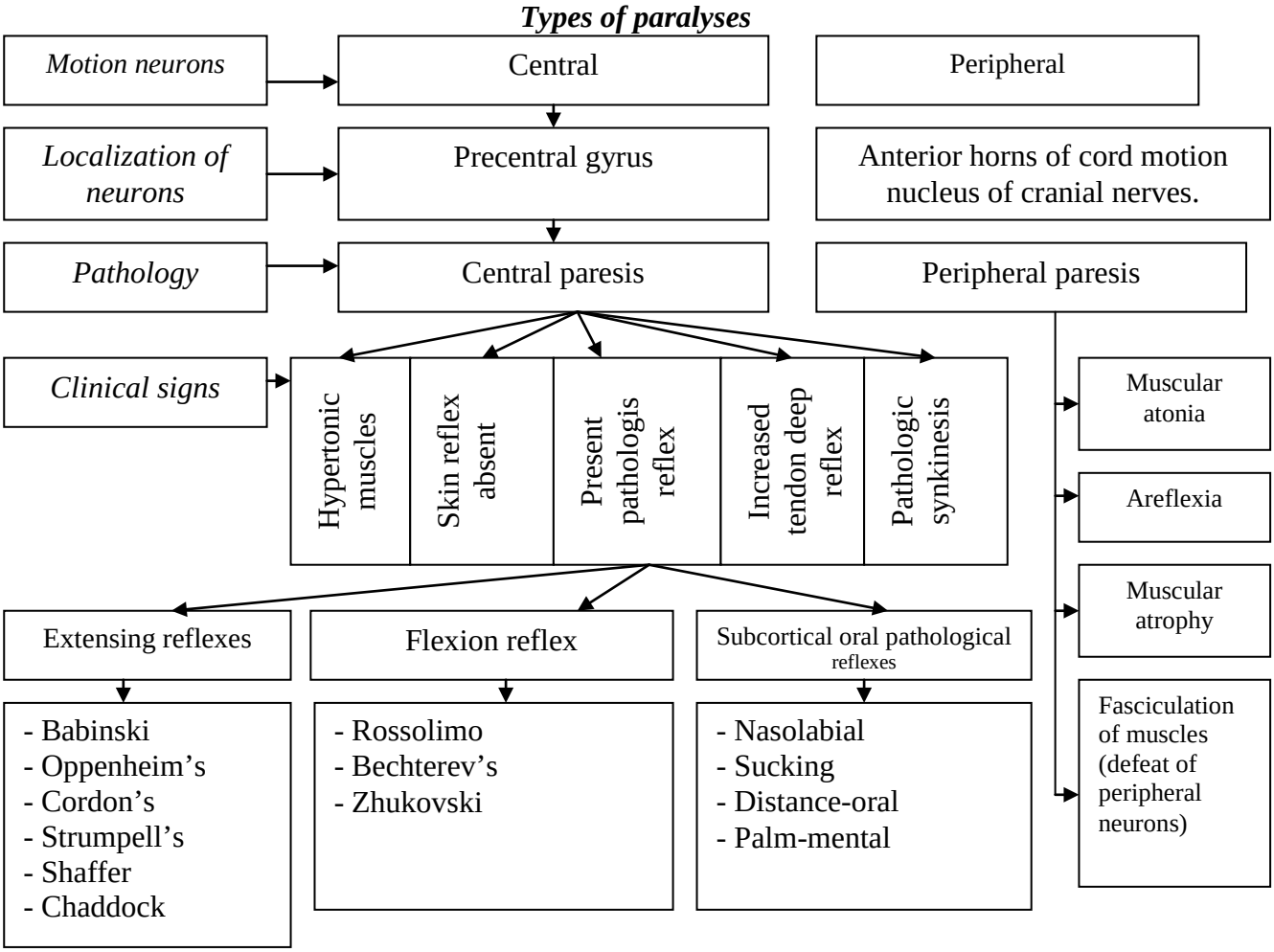
A comatotopic organization of the motor cortex



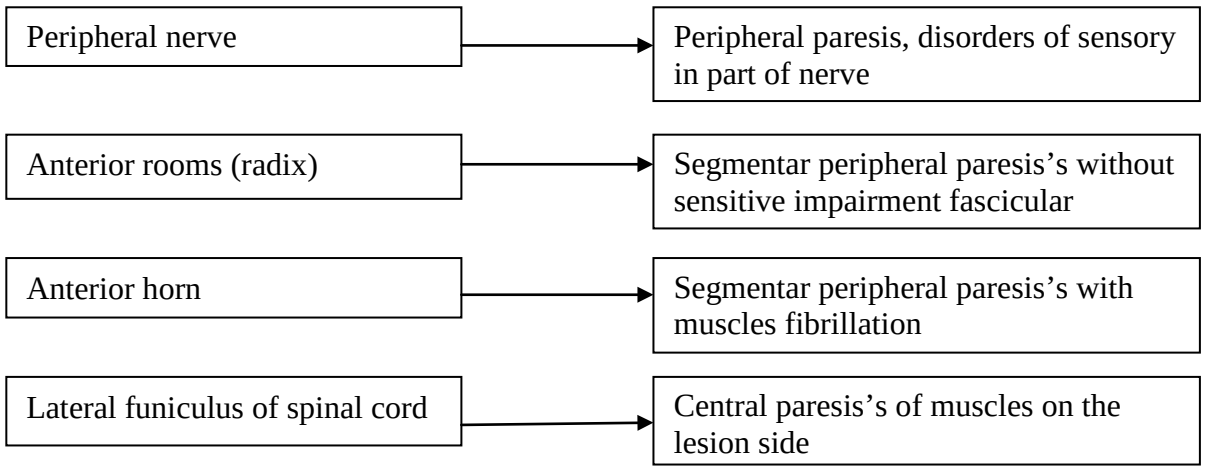
Pyramidal system

1 – precentral gyrus; 2- internal capsule; 3- cranial nerves nucleuses; 4 – cervical intumescence; 5 – anterior horns; 6 – lumbar intumescence; 7 – motoneurons of anterior horns.

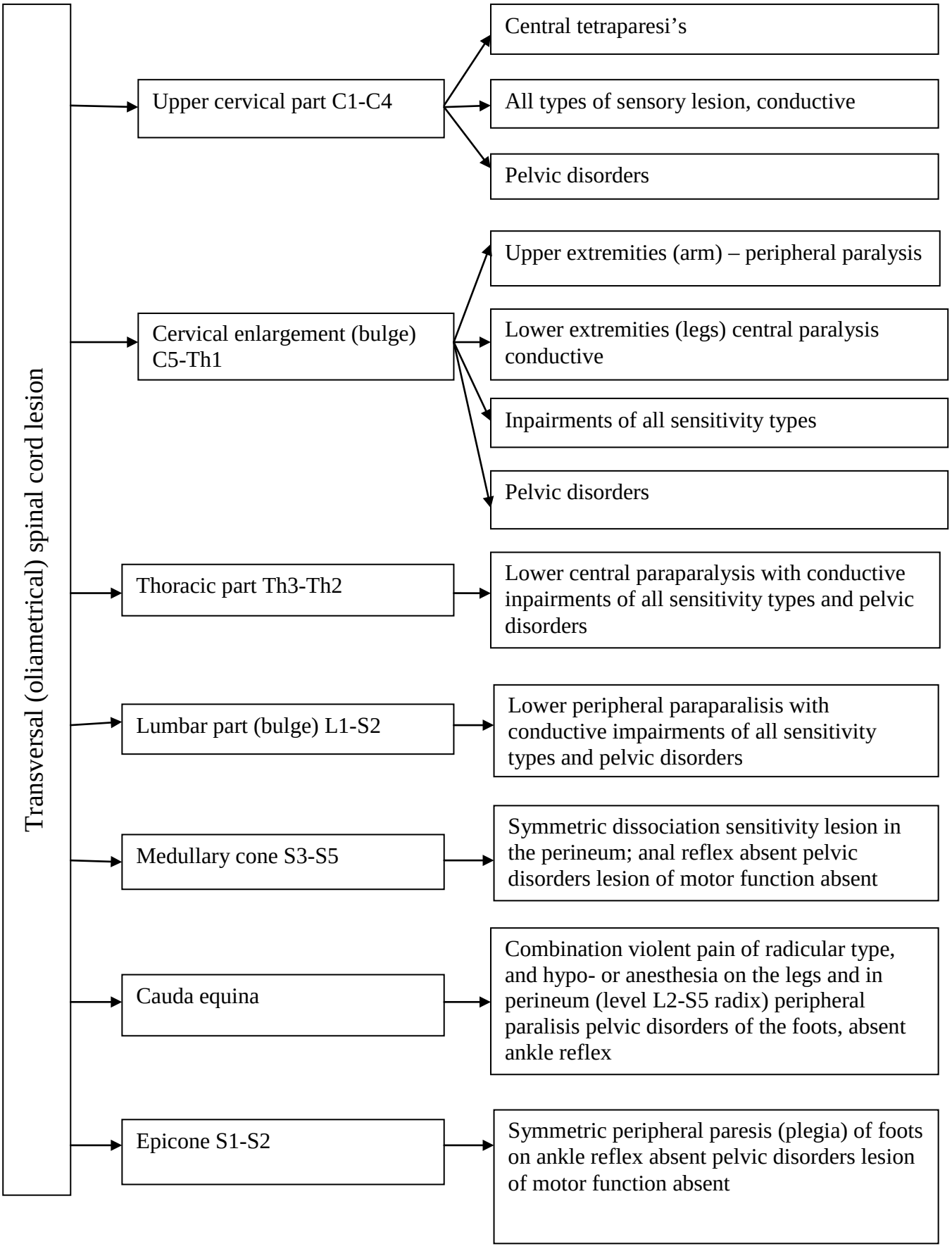
Central and peripheral paralyses

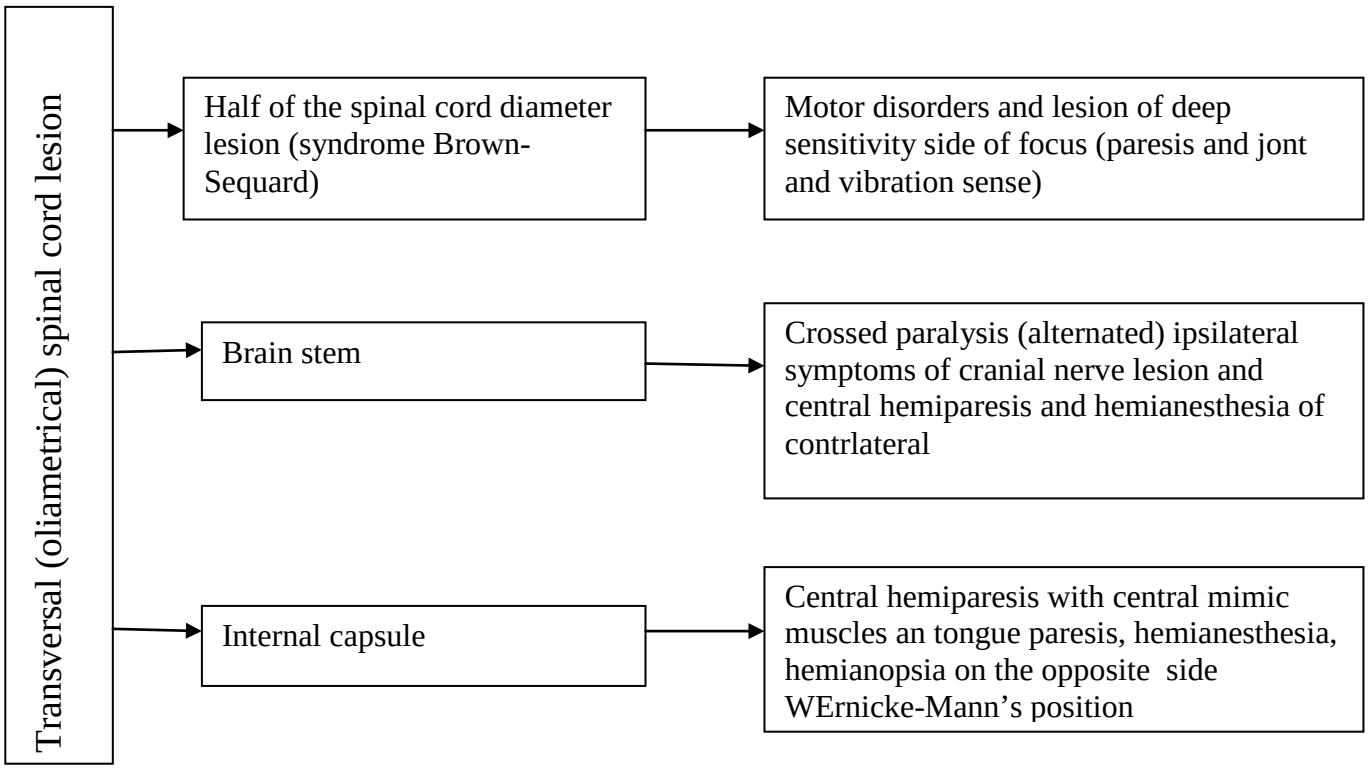


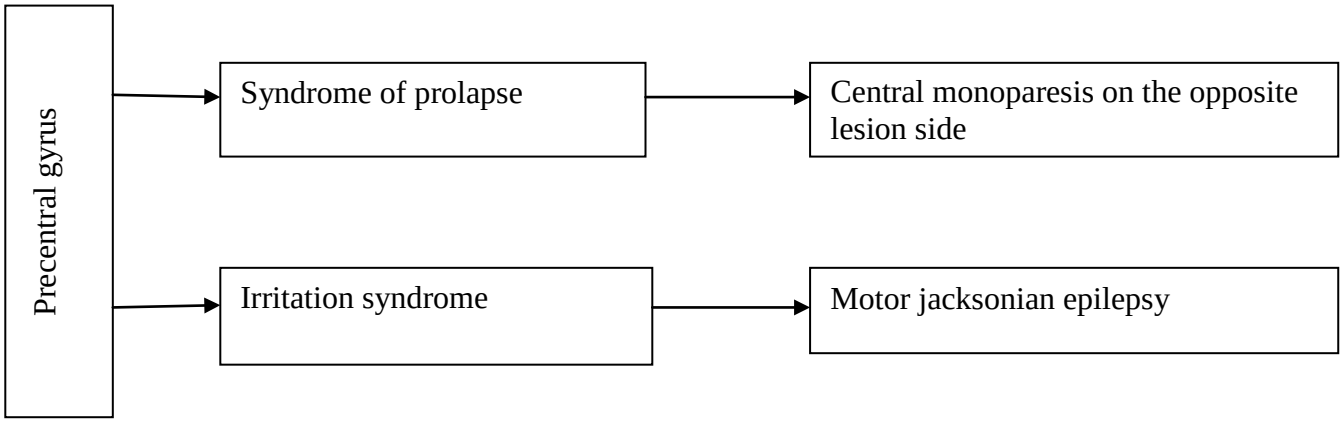
Syndrome's defeat of pyramidal system (motor tract in different levels)



Spinal cord disorders







Test and typical task

1. Where is the primary cortical field of motor analyzer located?

A. Precentral gurus

B. Postcentral gurus

C. Brainstem

D. Thalamus

E. Dorsal horns

2. The lesion of anterior horn of spinal cord. What disorders?

A. Peripheral paresis, fascicular twitches

B. Central paresis

C. Hemiataxia

D. Asteriognosis

E. Thalamic pain

3. The patient has in neurology examination: left side hemiplegia, hemianesthesia, hemianopsia, gait of Vernic-Mann. Where is the focus of defeat localization?

A. Consyla internal of the right side

B. Consyla internal of the left side

C. Brainstem

D. Thalamus right side

E. Radiate crown of the right

4. The patient has hyperreflexia of ankle reflexes is exposed. What the level of shorting?

A. $L_3 – L_4$

B. L_5 - S_1

C. S_1 - S_2

D. S_4 - S_5

E. Th_7 - Th_8

5. What from the indicated reflexes does not behave to superficial?

A. Pharyngeal

B. Corneal.

C. Plantar.

D. Knee jerk or patellar reflex.

E. Cremasteric.

6. A patient has peripheral paresis of lower extremities. What is not characteristic?

A. Spastic hypertonia.

B. Fasciculation twitches.

C. Deep reflex absent

D. Hypotonia.

E. Pathological reflexes absent.

7. A child is diagnosis: Poliomyelitis. At examination: areflexes, hypotonia, hypotrophy, fasciculation of muscles. What is the dysfunction?

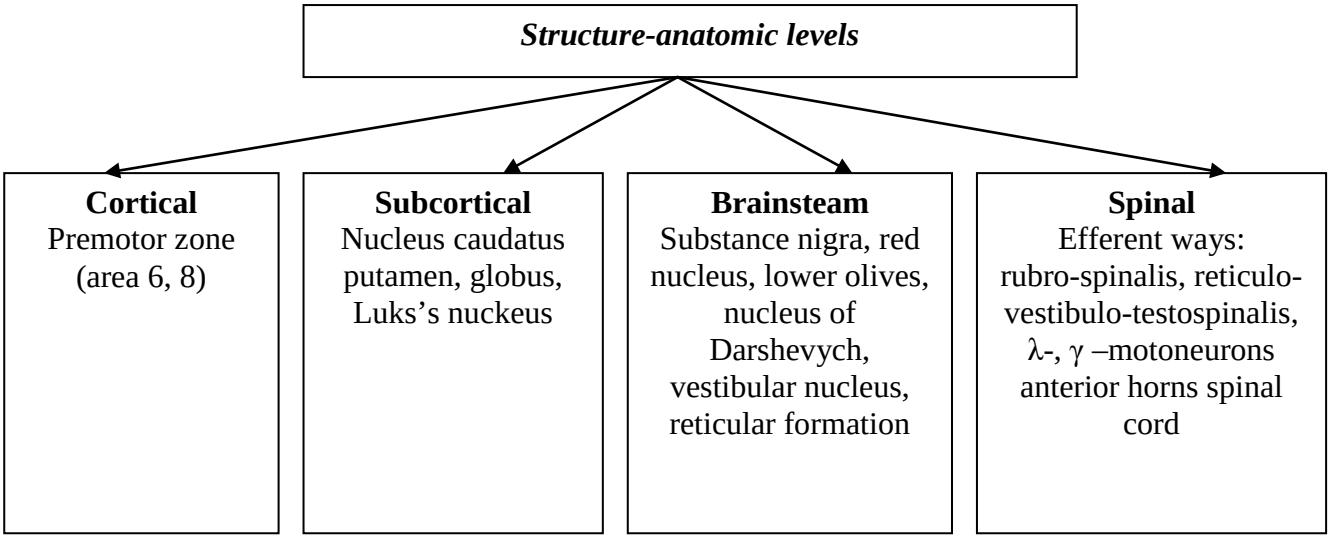
8. A patient has a weakness in a right leg. Objectively: atrophy of muscle of thigh, is absent knee-jerk, fasciculation twitch, violation of sensitiveness. Where the pathology focuses of is localized?

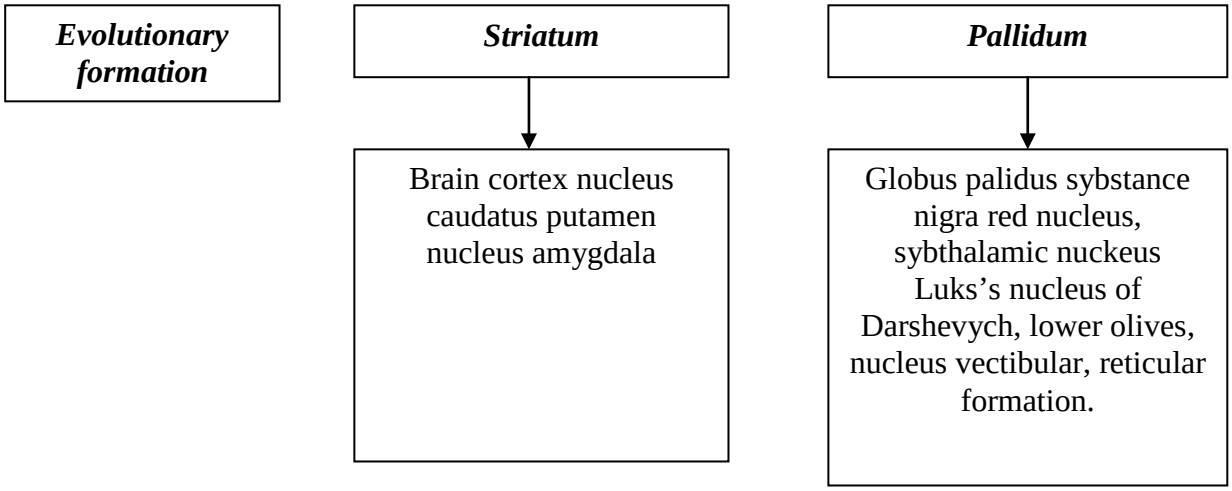
9. The patient has a flassid lower paraplegia (more of feet) with atrophy of muscles, anesthesia on a segmentar type (on the internal surface of thighs), great pains in feet, violation function of pelvic organs. Where is the focus of defeat localized?

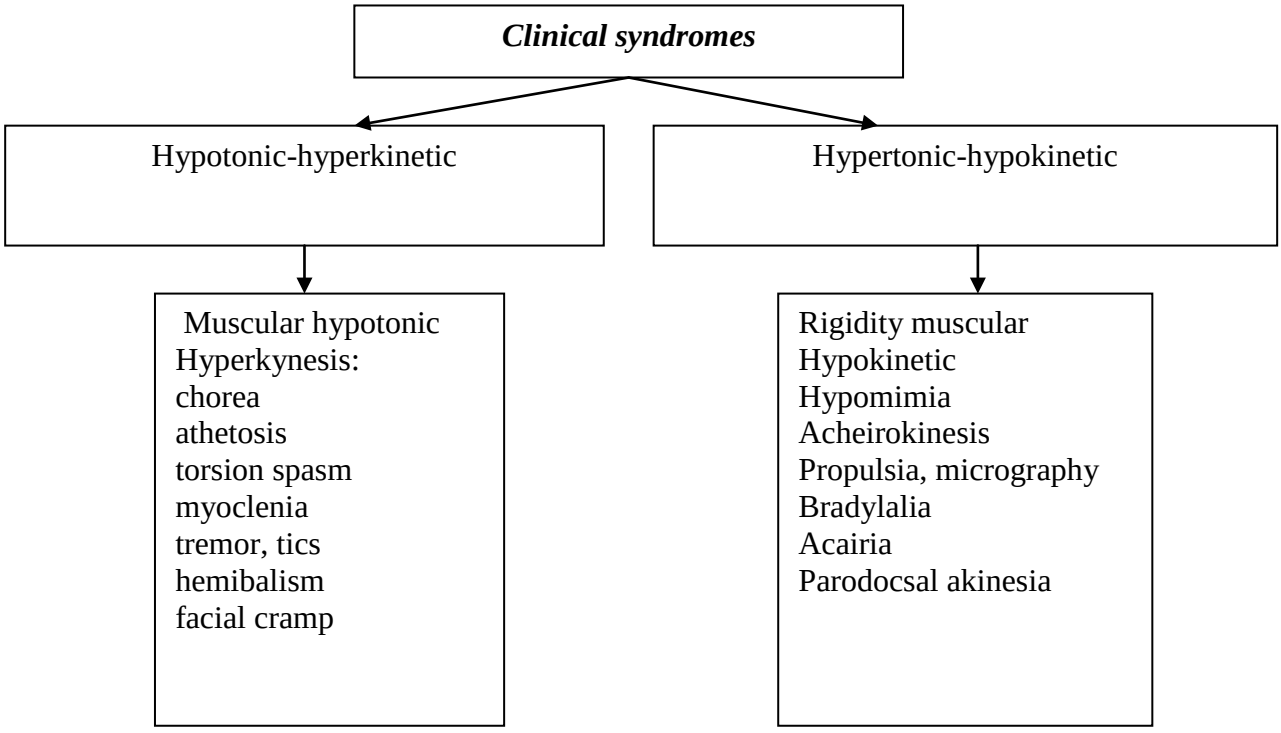
10. At a patient in neurology status: left-side hemiplegia, hemianesthesia, hemianopsia, pose Vernic-Mann send. Where is the focus of defeat localized?

List of answers: 1.A. 2.A. 3.A. 4.C. 5.D. 6.A. 7. Frontal horns. 8. Lumbar part on the right. 9. Cauda ecvina. 10. Internal capsule on the right.

3. THE EXTRAPYRAMIDAL SYSTEM AND SYNDROMES OF ITS LESION







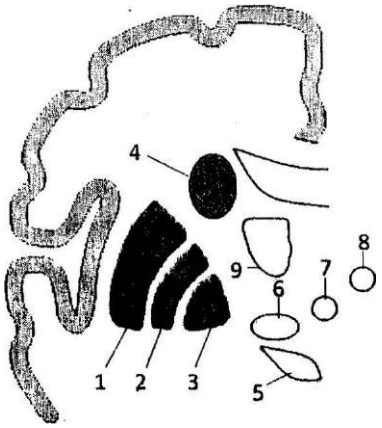
Correction defeat of metabolism dopamine with presence Parkinsonism

Increase synthesis DA
Preparation have L-DOPA (nacom or sinimet, madopar)

Stimulation ejection DA of granule maintenance in presynaptic part DA-synapse:
1. Blockers of glutamatis receptors (amantadine, norakine).
2. Antogonicts DA (bramckiptin's, pergolid)

Inhibition process backward capture DA:
1. Anticholinergics preparations (ciklodols, parcopans)
2. Antidepressants (amitriptyline)

Inhibition process catabolism DA:
1. Inhibitor MAO (selegiline)
2. Inhibitor catecholaamintransferas (COMT) (tolpocans)



The structures of the extrapyramidal system:
1 — putamen;
2 — globus pallidus dorsalis;
3 — globus pallidus ventraiis;
4 — nucleus caudatus;
5 — substatia nigra;
6 — Lues' body;
7 — upper hill;
8 — vestibular nucleus;
9 — thalamus

In 1817 the English doctor James Parkinson was the first who described the major manifestation of this syndrome and this disease was called Parkinson's disease. In 1920 Tretiakov noticed that the greater is a cell loss in the substantia nigra, the lower concentration of dopamine is in the striatum.

Now there are two forms of parkinsonism: primary and secondary. Primary parkinsonism (94-96 %) is named Parkinson's disease (idiopatic parkinsonism).

Secondary parkinsonism: postencephalitic, vascular, toxic, post-traumatic, drug-induced, oncologic are seldom.

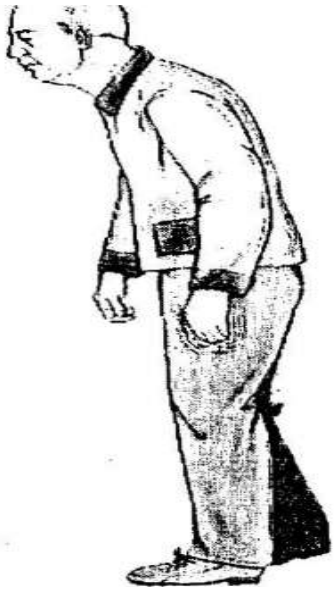


Fig. 3.2. The appearance of a Parkinson's patient

Chorea is characterized by fast polymorphic movements, non-stereotypic chaotic involuntary movements in different muscular groups against the background of the low muscular tonus. It is usually more pronounced in distal segments of the extremities. When severe, however, they may be of a very high amplitude, randomly directed and extremely disturbing. Grimacing and lip-smacking may be prominent.

Athetosis consists of slow, irregular, exaggerated, uncomfortable- and cramped-appearing involuntary movements that are more pronounced in distal portions of the extremities. It is snakelike movement of any combination of flexion, extension, adduction and abduction in varying degrees.

Hemibalism — lateral swinging movements of proximal parts of the extremities. These disorders are characterized by lighthing-like, high-amplitude, flinging ("ballistic") movements simultaneously involving multiple segments of a limb. It is similar to "wingbeat".

Tics are stereotypic hyperkinesias of the face and upper shoulder girdle muscles, which remind voluntary movements (winking, neck, shoulder, head twitching), but never prevent voluntary movements.

Torsion dystonia (lat. torsjo — twisting) — corkscrew-like movements of the body, neck and pelvic girdle muscles.

Spastic curvature of the neck — a local form of torsion dystonia.

Hemispasm of the face — rhythmic twitching of half face muscles.

Paraspasm of the face — bilateral twitching of face muscles.

Writing spasm — reminds "an obstetrician's hand".

Test and typical task

1. A patient the rigidity muscles, bradykinesia, stiff, shuffing gait, “pill-rolling” tremor. Specify the most credible syndrome.

A. Hypotonicohyperkinetic syndrome.

B. Diencephalic syndrome.

C. Hyperkinetic syndrome

D. Omiostatic syndrome.

E. Hypertonic-hipocinetic syndrome.

2. At patient sweeping, throw, rotator motions appeared with, mainly in the departments of extremities. Specify the syndrome of defeat.

A. Chorea.

B. Athetosis.

C. Parkinsonian syndrome.

D. Hemibalismus

E. Dejerine’s-Rusi syndrome.

3. The a child has of Fridreykh disease slow, unsynchronous vermiform fanciful motions appeared in the distal departments of extremities. What type of hyperkinesia at a patient?

A. Chorea.

B. Mioklonia.

C. Hemiballism.

D. Athetosis.

E. Torsion spasm.

4. What syndrome is not characteristic for the syndrome of parkinsonis?

A. Syndrome Noik's.

B. Syndrome «air pillow».

C. Syndrome Stuart-Cholm's.

D. Tremor of "pill-rolling"

E. Postural instability (propylsia).

5. What type of syndrome is not characteristic for the hypotonic-hypercinetic?

A. Hemibalism.

B. Tics.

C. Parkinson's

D. Atehetosis.

E. Chorea.

6. Describe physiological functions of the extrapyramidal system.

A. Realization of automatic movements, muscle tone support

B. Realization of conditioned reflexes, coordination

C. Realization of voluntary movements, constriction of smooth muscles

D. Constriction of striated muscles, realization of voluntary movements

E. constriction of striated muscles, function of equilibrium

7. The patient has complaint about the constraint of motions, change of gait, tremor. Objectively: a pose is «asking», amimia, tremor, the Noik syndrome is positive from two sides, the type of tremor «pill-rolling». What syndrome developed at a patient?

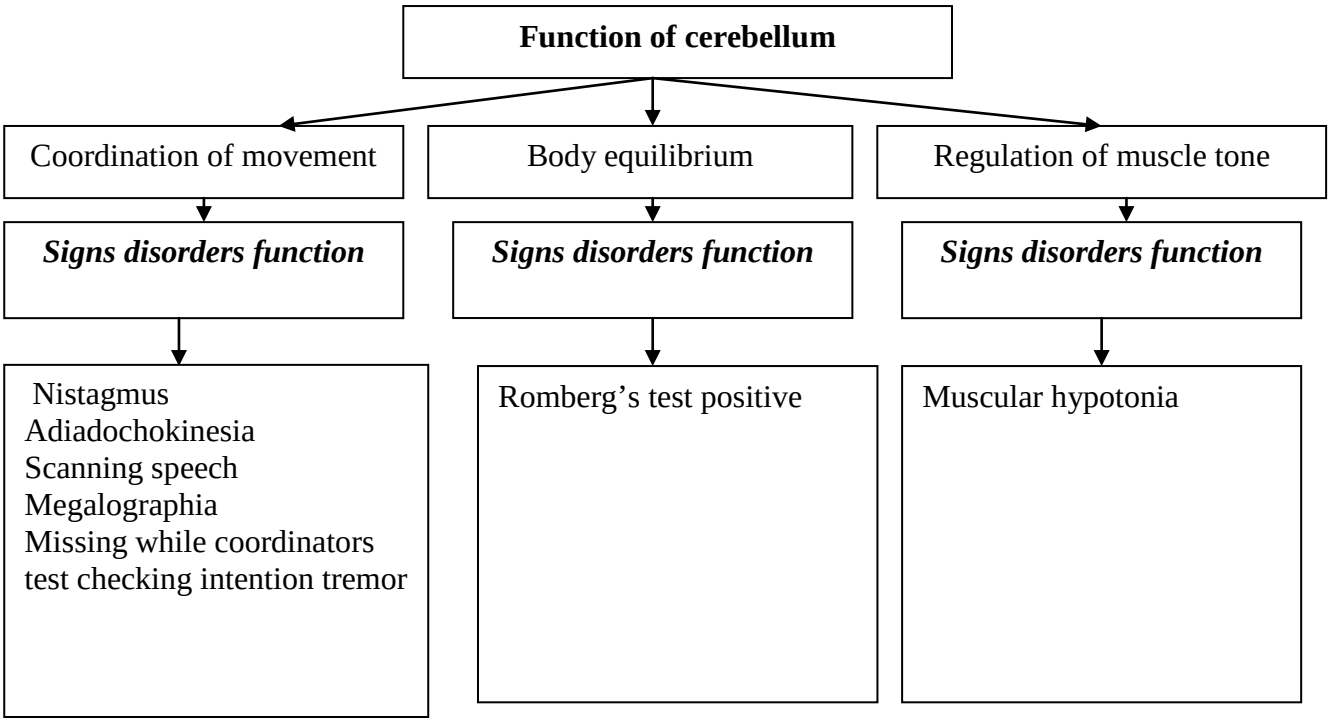
8. The child has by rheumatism, rapid, brief, disorderly, unrhythmical motions appeared with involving in process simultaneously different muscles of face, extremities and trunk. What syndrome developed at a patient?

9. The patient has used a narcotic drug for a long time. In neurological status the doctor finds muscule rigidity, tremor, postural imbalance, hypomimia. Name the pathological syndrome. Which structures are impaired?

10. The patient has worm-like slow movements in distal parts of the extremities, the muscle hypotonus, disorder of hepar function. How is this syndrome called? Which structures are impaired?

List of answers: 1. E. 2. D. 3.D. 4. C. 5. C. 6.A. 7. Parkinson's syndrome. 8. Chorea. 9. The pallidal system is impaired (substantiia nigra). 10. Hypotonic-hyperkinetic syndrome (athetosis), the striatic system is impaired (caudate nucleus).

4. THE CEREBELLUM AND IT'S PATHOLOGY



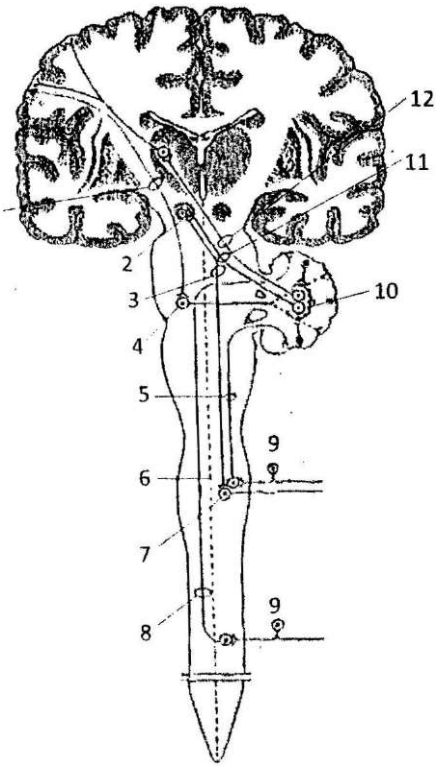
The cerebellum is connected with all other parts of the central nervous system by means of its peduncles. Cerebellar proprioreception gets to the cerebellum by two

spino-cerebellar tracts — anterior and posterior. As a part of the first neuron irritation from proprioreceptors of muscles, joints, tendons, perosteum gets to the basis of the posterior horn of the spinal cord by peripheral nerves through the posterior roots. Here Clark cells (the second neuron) are located, axons of which, not making the decussation, go up to the posterior surface of the lateral funiculus of the spinal cord, creating the posterior spino-cerebellar tract or Flexig's tract (the tractus spino-cerebelaris dorsalis). Reaching the medulla oblongata, this tract as a part of the lower peduncle of the cerebellum enters the cerebellum, finishing mainly in its worm.

The spino-cerebellar anterior tract or Hoover's tract (the tractus spino- cerebelaris ventralis) takes its origin from the cells of the posterior horns of the spinal cord, axons of which pass to the anterior part of the lateral funiculus of the opposite side, and going up to the spinal cord and brain post, in the level of the upper of cerebellum make the second decussation and through the upper cerebellar peduncles reach the worm. The cortex of the cerebellum contains third neurons, the axons of which switch on the cortex cells of the cerebellar hemisphere. The shoots of these cells go the dentate nucleus (tractus cerebellodentatus), and from it as a part of the dento-rubral tract through the upper peduncle of the cerebellum go to the opposite red nucleus, making the decussation of the upper cerebellar peduncle. Axons of the cells of red nucleus create the Forel's decussation just after leaving and as a part of rubrospinal tracts reach alpha- and gamma mononeurons of spinal cord.

The cerebellum gets afferent proprioceptive impulses from vestibular tracts (the tractus vestibule-cerebelaris), olives (tractus olivo-cerebelaris) and nuclei of the posterior funiculi — the thin and the wedge-like ones.

As far as Flexig's pathway does not make decussation, and anterior spinocerebellar Hoover's tract makes it twice, all irritations from the left part of the body get to the left part of the cerebellum, and from the right part — to the light one. Thus brain is connected with the body homolaterally.



Cerebellum and it's communications:

1— tr. cortico-pdntinus;

2— nucleus ruber;

3— tr. rubro-spinalis,

4— nucleus of pons;

5— Flexig's tract (tr. spino-cerebellaris dorsalis);

6 — posterior horn of spinal cord;

7 — anterior horn of spinal cord;

8— Hoover's tract (tr.spinocerebellars ventralis);

9 — spinal ganglion;

10 — nucleus dentatus;

11 — tr. dento-rubralis;

12 – tr.cerebello-thalamicus

The *lower peduncles* provide connection with the brainstem and spinal cord: tr. spinocerebellaris dorsalis (Flexig's). tr. vestibulocohlearis, tr. olivoce- bellaris, fibre arcuate externe. The cerebellum is included in a system of voluntary movement’s coordination due to its links with the brain cortex. The afferent cortico-cerebellopontine

tracts go to the cerebellum, carrying impulses about the planned action by the brain cortex. These are two-neural tracts. The first neuron is a corticopontine tract. It takes beginning from frontal, occipital, temporal lobes. First one passes through the semioval centre, anterior limb of the internal capsule and ends in the nuclei of the pons on its side. Occipitotemporopontine tract starts from the occipital lobe and posterior parts of the temporal gyri, goes through the posterior limb of the internal capsule and ends in the nuclei of the same side of the pons. The *middle peduncles* provide connection with pons. They are presented by fibers of tr. pontocerebellaris. They connect the nuclei of the pons with the opposite hemisphere of the cerebellum. The *upper peduncles* of the cerebellum connect cerebellum with the middle brain. They include two systems: the afferent one — from the spinal cord to the cerebellum — tr. spinocerebellars ventralis (Hover's); efferent one — from the cerebellum to the structures of the extrapyramidal nervous system — tr. cerebellotegmentalis and tr. dentorubralis.

Equilibrium and regulation of the muscle tone are the functions of the flocculo-nodular lobe (vermix).The main function of the cerebellum hemisphere is coordination of movement and synergy. Impairment of the cerebellum produces cerebellar ataxia. There are two types of ataxia: the static one (it develops at lesion of the vermix) and the dynamic one (it develops at lesion of hemispheres).

Static ataxia means standing and walking disorders. It is checked in Romberg test.

Dynamic ataxia can be observed while moving. The main signs are the following: nystagmus, scanning speech, intention tremor, missing while coordinator tests checking, dysmetria, muscular hypotonia, adiadochokinesia, macrographia, asynergia.

Nystagmus. Discoordination in the work of muscles that ensure the eyeballs movements lead to involuntary rhythmic quickly repeated jerking of eyeballs when looking aside or looking up.

Scanning speech. Chopped, explosive speech with separate, effortful pronunciation of each syllable.

Intention tremor. Oscillating deviation from the optimal path of movement that increases in the amplitude as the target is approached, generally due to lesions of the

dentate nucleus or its efferent tract. It is easily detected with finger-nose and heel-knee tests.

Dysmetria. Incorrect amplitude or velocity of a planned movement.

Muscle hypotonia. A diminished muscle tone on passive movement.

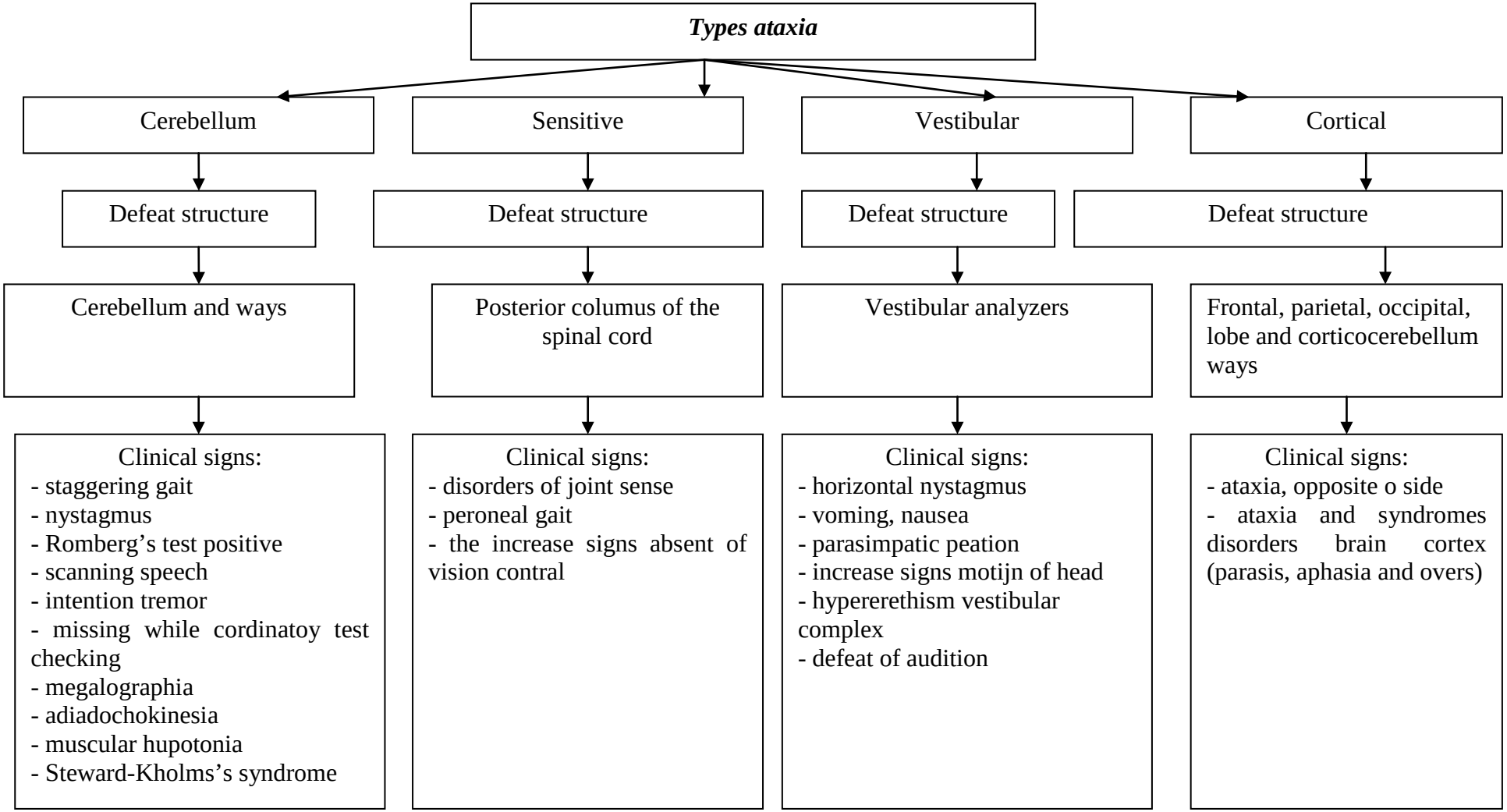
Adiadochokinesia. Impaired performance of rapid alternating movements, due to inadequately rapid and fluid alternation of agonist and antagonist contraction. One may test this with rapid alternating pronation and supination of the forearm.

Macrographia. Writing with big letters.

Asynergia. Backward bending of the trunk without concomitant knee flexion, resulting in a loss of balance.

The main kinds of ataxias:

- ✓ Cerebral
- ✓ Sensitive
- ✓ Vestibular
- ✓ Cortical


Types ataxia
Cerebellum
Sensitive
Vestibular
Cortical
Defeat structure
Defeat structure
Defeat structure
Defeat structure
Cerebellum and ways
Posterior columus of the spinal cord
Vestibular analyzers
Frontal, parietal, occipital, lobe and corticocerebellum ways
Clinical signs:
- staggering gait
- nystagmus
- Romberg's test positive
- scanning speech
- intention tremor
- missing while cordinatoy test checking
- megalographia
- adiadochokinesia
- muscular hupotonia
- Steward-Kholms's syndrome
Clinical signs:
- disorders of joint sense
- peroneal gait
- the increase signs absent of vision contral
Clinical signs:
- horizontal nystagmus
- voming, nausea
- parasimpatic peation
- increase signs motijn of head
- hypererethism vestibular complex
- defeat of audition
Clinical signs:
- ataxia, opposite o side
- ataxia and syndromes disorders brain cortex (parasis, aphasia and overs)

Test and typical task

1. The patient disturbed dizziness, nausea, vomiting, and decline of ear on a right ear. Objectively: horizontal nystagmus to the right, AP 150/90 mm Hg hyperhidrosis, pallor of skin covers. What type of ataxia at a patient?

A. Cerebellum.

B. Sensitive.

C. Vestibular.

D. Frontal.

E. Temporal.

2. The patient of complaint of change of handwriting, tremor, speech is irregular. Objectively: positive syndrome Styuart-Cholms, intencion tremor, adiadochokines, megalographia, nystagmus. What syndrome developed at a patient?

A. Syndrome defeats of hemispheres of cerebellum.

B. A syndrome is the defeat of vermis of cerebellum.

C. Syndrome of defeat of thalamus.

D. Syndrome of defeat of frontal lobe.

E. Syndrome of defeat of labyrinth or vestibular nucleus of brainstem.

3. For the syndrome of defeat of cerebellum not characteristic:

A. Hemianesthesia.

B. Intention tremor

C. Hearing impairment

D. Ataxia

E. Megalographia.

4. A patient is disturbed by unsteadiness at walking, change of handwriting, speech. Objectively: goes around with the widely placed feet, horizontal nystagmus, intension

tremor, megalographia, adiadochokinesis, and decline of muscular tone. Specify the syndrome of defeat.

A. Defeats of internal capsule syndrome.

B. Parkinsonian syndrome.

C. Defeats of cerebellum syndrome.

D. Defeat of frontal lobe syndrome of.

E. Dejerine’s-Rusi syndrome

5. What kind of speech disorder appears in the case of cerebellum impairment?

A. Silent speech

B. Dysarthria

C. Aphasia

D. Scanning speech

E. Anarthria

6. Indicate the coordination tests

A. Reflexes exam finger-nasal test, diadochokinesia test

B. Rhinne's and Weber's tests

C. Upper and lower Barre's tests

D. Examination of the muscular tone

E. Examination of the muscular tone

7. At a patient as a result of the stress loading: in the pose Romberg is a tendency to falling back at the distraction of attention, kept balance, intencion shaking, nystagmus, scanned speech is absent. Sensitiveness is stored; a patient is angry, feel like stormy emotional reactions. How is this syndrome named?

8. A patient has problems with speech. It is chopped, explosive speech with a separate, effortful pronunciation of each syllable. The neurological examination shows missing the mark with finger-nasal and heel-to knee, tests. Which syndrome has this patient?

9. In a patient were observed acute ischemic stroke in the cerebellum. Now he has scanning speech, impairments in coordinational tests. There is imbalance in Romberg's test. Which syndrome has this patient? How will muscle tonus be changed?

10. A tumor has destroyed the right hemisphere of the cerebellum. A patient has nystagmus, missing the mark with finger-nasal and heel-to knee tests in the right side, adiadochokinesia in the right side. Which syndrome has this patient?

List of answers: 1.C. 2. A. 3.A. 4. C. 5.D. 6.B. 7. Hysterical neurosis. 8. Dynamic ataxia. 9. Cerebellar ataxia, muscular hypotonia. 10. Cerebellar ataxia in right side.

5. PATHOLOGY OF VEGETATIVE NERVOUS SYSTEM

The autonomic (vegetative) nervous system is responsible for the process of nutrition of an organism, metabolism, growth, reproduction, circulation of liquids, so it controls the activity of the internal organs.

According to the international anatomic nomenclature, the term "autonomic nervous system" is commonly used. However, the previous name "vegetative nervous system" is traditionally used in Ukrainian literature.

The main functions of the autonomic nervous system

a) trophotropic—regulation of the activity of the internal organs, maintenance of the stability of the internal environment of the organism — homeostasis;

b) ergotropic — provision of adaptive processes — all forms of the psychic and physical activity of an organism.

Mentioned physiological functions are regulated independently (autonomicly), without a conscious control of them.

The autonomic nervous system is divided into the following parts: supra- segmental and segmental levels. The last one according to the structure and functional peculiarities is divided into the sympathetic and parasympathetic nervous system. The sympathetic nervous system innervates all the organs and tissues of an organism, unlike the parasympathetic one. The central nervous system, most vessels, uterus,

modular layer of adrenal glands, sudoriferous glands don't have parasympathetic innervations.

The suprasegmenlal level is presented by the hypothalamo-limbico-retic- uiaris complex.

Hypothalamus

The hypothalamic zone plays an important role among subcortical structures. The hypothalamus is connected with many structures of the central nervous system, it provides integration of the somatic and vegetative activity of an organism.

The main functions of the hypothalamus are regulation of heart-vascular activity, regulation of endocrine glands' function, regulation of lipid, water, mineral metabolism, thermoregulation, the emotional behavior, the homeostasis of the internal environment of an organism.

Pathogenesis of hypothalamic syndrome is conditioned by the peculiarities of its vascularization: the intensity of capillar blood supply and high penetrability of its vessels lead to an increase of the penetrability of vessels of this part for great-molecular compounds (toxins, viruses, hormones and other humoral substances). It leads to high susceptibility of the hypothalamic zone in the case of arising of different pathological processes.

The Limbic System

The complex'of limbic system structures is an organizer of the unity of many functions of an organism. It consists of: the bulbus, tractus and trigonum ol- phactori, substantia perforate anterior, septum pellucidum, gyrus cinguli, gyrus hypocampalis, corpus amygdaloidem, mediobasal surface of the frontal lobe.

Here primary synthesis of all sensivity, the analysis of the state of the internal environment are carried out and elementary needs, motivations, emotions are formed. The limbic system provides interaction of vegetative, visceral, sensor- motor and emotional systems. Its state influences on the level of consciousness, attention,

memory, ability to orientate in space, motor and psychic activity, ability to perform automatic movements, disorders of sleep and awakeness.

Formatio reticularis

Thet reticular formation of the brainstem plays a significant role in the supra-segmental part also. It has its independent role, but is also one of the integrative apparatuses of the brain. The nuclei of the reticular formation (there are about 100 ones) form suprasegmental centres of vital functions: respjrative, vascularmotor, cardial activity, swallowing, vomiting and so on. The reticular formation also controls the state of sleep and awakeness, the physical and tonic state of muscles, decodes informative signals from the environment.

Interaction of the reticular formation with the limbic system provides organization of conscious activity according to changeable conditions of the environment.

Pathology of the suprasegmental level

The lesions of *the hypothalamus* cause hypothalamic syndromes. There are main forms of hypothalamic syndromes.

1. *Neuroendocrine form* manifests as Itsenko — Cushing syndrome, adiposogenital dystrophy, central/hypothalamic obesity, pituitary cachexia (Simmonds' disease), diabetes insipidus, idiopathic edemas, persisting lactorrhea-amenorrhea.
2. *Vegetative vascular dystonia form.* It is associated with the crisis of the paroxysmal character. There may be 3 variants of crisis: sympathy-adrenal, vagoinsular and mixed attacks. The signs of a sympathy-adrenal attack are: a pale and dry skin, shortness of breath, dizziness or faintness, high blood pressure, tachycardia, feeling of internal tremor, fear of dying. The opposite signs of a vagoinsular attack: hyperemia of the face, sweating, bradycardia, decreasing blood pressure. Mixed attacks begin as the sympathy-adrenal and finish as the vagoinsular crisis or vice versa.
3. *Thermoregulative disturbances.* The permanent body temperature rise is up to 37.1-37.5. There is asymmetry under the arms and in the rectum.

4. *Neurotrophic form* is associated with trophic disturbances (skin dryness, neurodermitis, ulcers, bed sores, acute perforates of stomach and esophagus).
5. *The neuromuscular form* is characterized by myastheno-like, myotono-like syndroms. Sometimes may be cases of paroxysmal myoplegia.
6. *Disorders of sleep and awakeness* are associated with insomnia, lethargy and sleeping inversion.

The symptoms of Limbic System *lesion are* emotional disturbances, changes of eating behavior (anorexia or bulimia), sleeping disorders, sexual disturbances, memory disorders.

The segmental part of the autonomic nervous system

The parasympathetic part is divided into the craniobulbar and sacral parts.

The craniobulbar part

There are the fibers from parasympathetic Yakubovich's and Perlea's nuclei included in the oculomotor nerve that provide innervation of smooth eye muscles: the muscle sphincter pupillae and muscle ciliaris, the latter ensures accomodation of crystalline lens.

There are the fibers from secretory lacrimal nucleus included in the facial nerve that provide innervation of the tear-exciting gland.

There is the same anatomic structure of the superior and inferior salivatori- us nuclei included in the facial (VII pair of cranial nerve) and glossopharyngeal nerves (IX pair) that innervates parotid, sublingual, submandibular glands.

Dorsal (visceral) nucleus of the nervus vagus (X pair of the cranial nerve) innervates heart, gastrointestinal tract, gastric glands and other internal organs (except for the small pelvis).

The sacral pan of the vegetative nervous system

There are the fibers from segments S2-S4 included in the pelvic nerves (nn. pelvici) that provide innervation of the urinary bladder, rectum, genitals.

The sympathetic nervous system consists of cells of the lateral horns of the spinal cord (C8-L2). The axons of these cells form preganglionary fibers (white connecting branches). Some of them end in the sympathetic column, which consists of 20-23 nodi: cervical-3, thoracic — 10-12, lumbar — 3-4, pelvic — 4. Then postganglionary fibres (grey connecting branches) are formed for all organs and tissues of anorganism.

Some fibres are not disconnected in the sympathetic column, but go directly to prevertebtal ganglions, making plexuses, for example, the plexus celiacus.

Sympathetic innervation of the eye — the ciliospinal centre (cells of the lateral horn C8-T2). The axons of these cells are interrupted in the upper cervical ganglion. Then postganglionary fibres form sympathetic plexus around a.carotus internal and rise upwards into the skull for the innervation of such muscles as m.dilatator puppilae, m.orbitalis, m.tarsalis superficialis.

Sympathetic fibers from the ganglion stellate go around vertebral arteries, innervate vessels in the vertebra-basilar basin and give branches to the heart and larynx. The thoracic pad of the sympathetic column gives branches to the heart, lungs, pleura and organs of abdominal cavity. Sympathetic fibers from the sacral part go to organs and vessels of the small pelvis.

Clinical symptoms ot segmental parts lesion

They include the symptoms of III, VII, IX and X cranial nerves lesion if craniobulbar part is damaged.

Lesion of the ciliospinal centre or cervical sympathetic nerve leads to Bernard — Horner syndrome: partial ptosis, miosis and enophtalmia. Irritation of sympathetic fibers results in syndrome Pourfour du Petit: widening of palpebral fissure, mydriasis, exophthalmia.

Afterwards, there may be the following symptoms:

1. Sympathalgias (heart-like pain in the innervation area, senestopathies, paresthesias).

2. Vasovegetative syndrome — a change in skin color, tissue swelling, numbness, cold hands and feet.
3. Trophic vegetative syndrome — dryness, skin desquamation, a loss of hair, formation of ulcers, hyperkeratosis, nail fragility, arthropathy.

Vegetative innervation of the urinary bladder

Regulation of urinary function is performed by reflex, involuntary, and conscious mechanisms.

The urinary bladder has double vegetative (sympathetic and parasympathetic) innervation of smooth muscles: the detrusor and internal sphincter. The spinal parasympathetic centre is located in the lateral horns S2-S4 of the spinal cord. Parasympathetic fibers from this centre are included in pelvic nerves and innervate detrusor urine. Sympathetic innervation begins from the spinal sympathetic centre — the lateral horns T1-L2 of the spinal cord, and innervates the internal sphincter,

Regulation is performed with the help of cortical control and the voluntary reflex of regulation of urination. The afferent part of this reflex begins from receptors of the internal sphincter, includes spinal ganglions, posterior radixes, posterior funiculars, medulla, pons and finish in sensory zones of the cortex (girus fornicatus). The efferent part from the cortical motor centre of urination (the paracentral lobule) passes through the bilateral cortico-spinal tracts (the lateral and anterior funicles of the spinal cord) to the spinal centers of urination, which fibers are included in anterior radixes, genital plexus, n.pudendus and innervate the external sphincter of the urinary bladder.

The neurogenic bladder is a syndrome that unites disorders of urination, which appear in the case of lesion to nervous tracts or centres which innervate the urinary bladder and provide conscious function of urination. There are central and peripheral types of disorders of urinary bladder function.

Central types of disorders of urination appear in the case of bilateral lesion of the cortex and its connections with spinal (sacral) centres of urination.

There are:

a) urinary retention (retentia urinae);

b) periodic urinary incontinence (incontinentia intermittens);

c) an imperative urinary urge.

Peripheral types of urination disorders appear in the case of the localization of the pathologic process in sacral segments of the spinal cord, radixes of the cauda equina and peripheral nerves, so parasympathetic innervation of the urinary bladder is impaired. There are:

a) paradoxal urinary retention (the ishuria paradoxa);

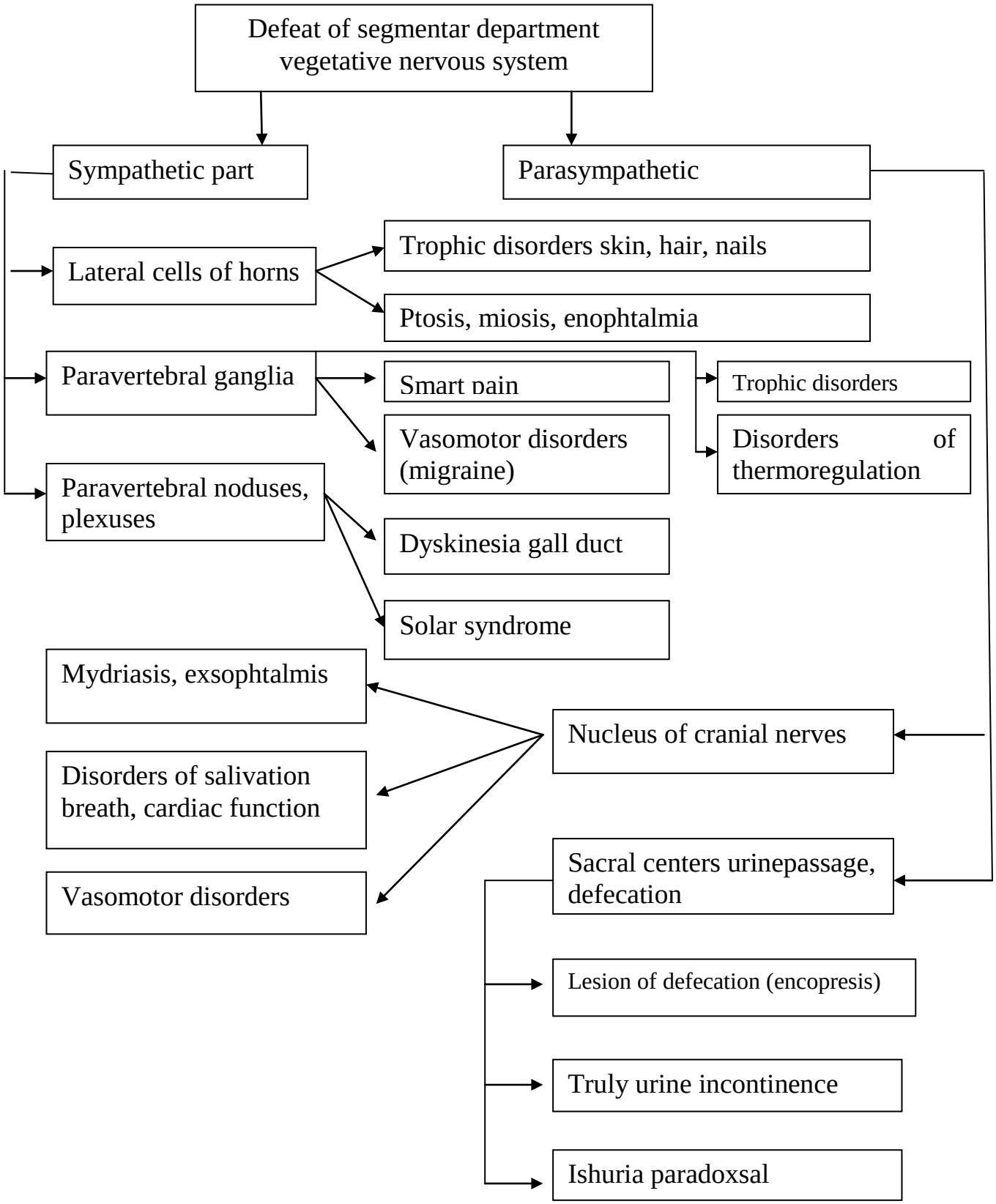
b) real urinary-incontinence (the incontinentia vera).

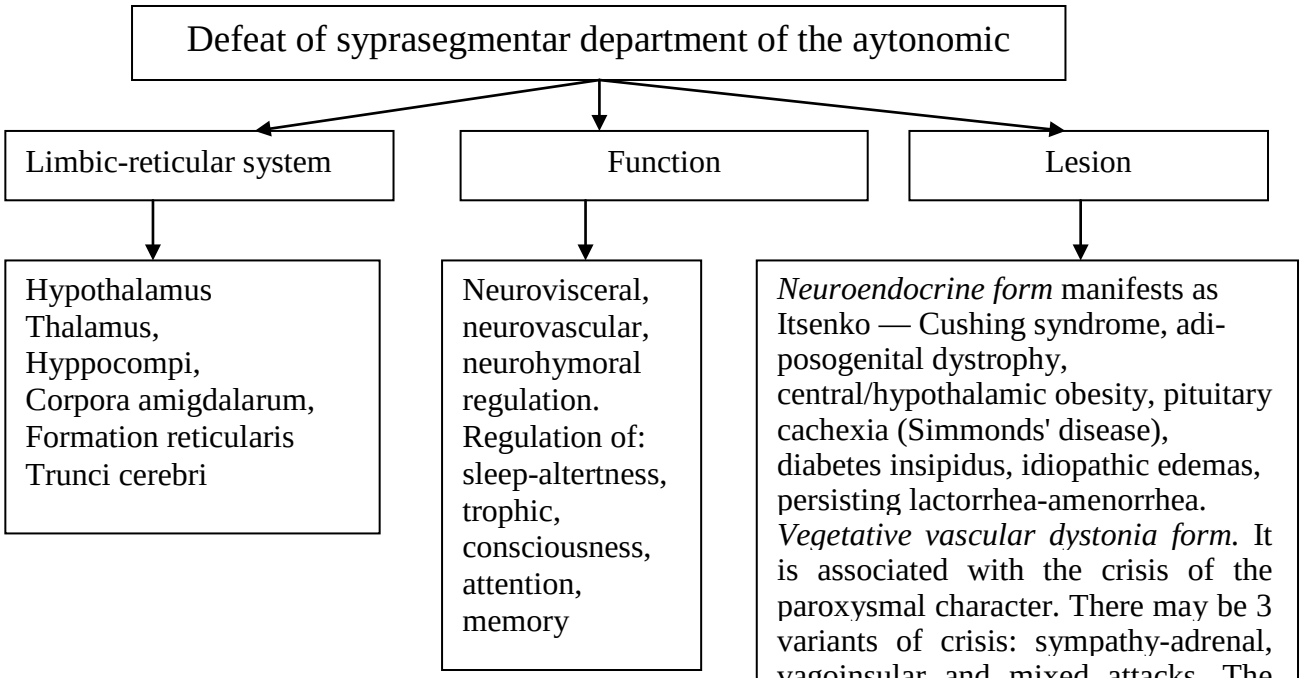
Classification of vegetative disorders

1. Syprasegmental (cerebral) lesion.

2. Segmentar (peripheral) lesion.

3. Combined syprasegmentar and segmentar vegetative (autonomic) lesion.


Defeat of segmentar department vegetative nervous system
Sympathetic part
Parasympathetic
Lateral cells of horns
Trophic disorders skin, hair, nails
Ptosis, miosis, enophtalmia
Paravertebral ganglia
Smart pain
Vasomotor disorders (migraine)
Trophic disorders
Disorders of thermoregulation
Paravertebral noduses, plexuses
Dyskinesia gall duct
Solar syndrome
Mydriasis, exsophtalmis
Disorders of salivation breath, cardiac function
Vasomotor disorders
Nucleus of cranial nerves
Sacral centers urinepassage, defecation
Lesion of defecation (encopresis)
Truly urine incontinence
Ishuria paradoxsal



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The neuromuscular form is characterized by myastheno-like, myotono-like syndroms. Sometimes may be cases of paroxysmal myoplegia.

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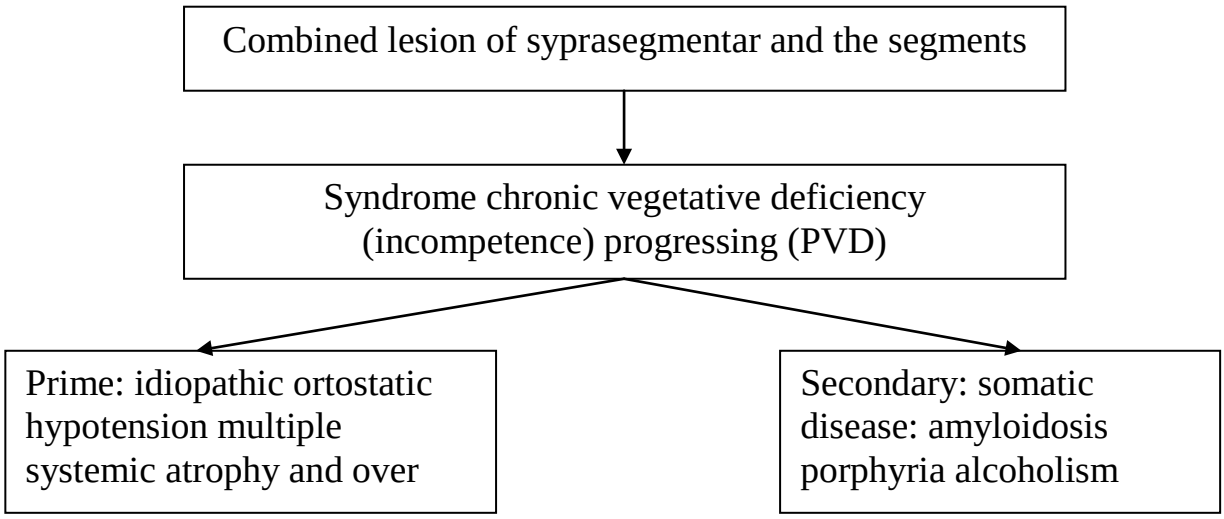
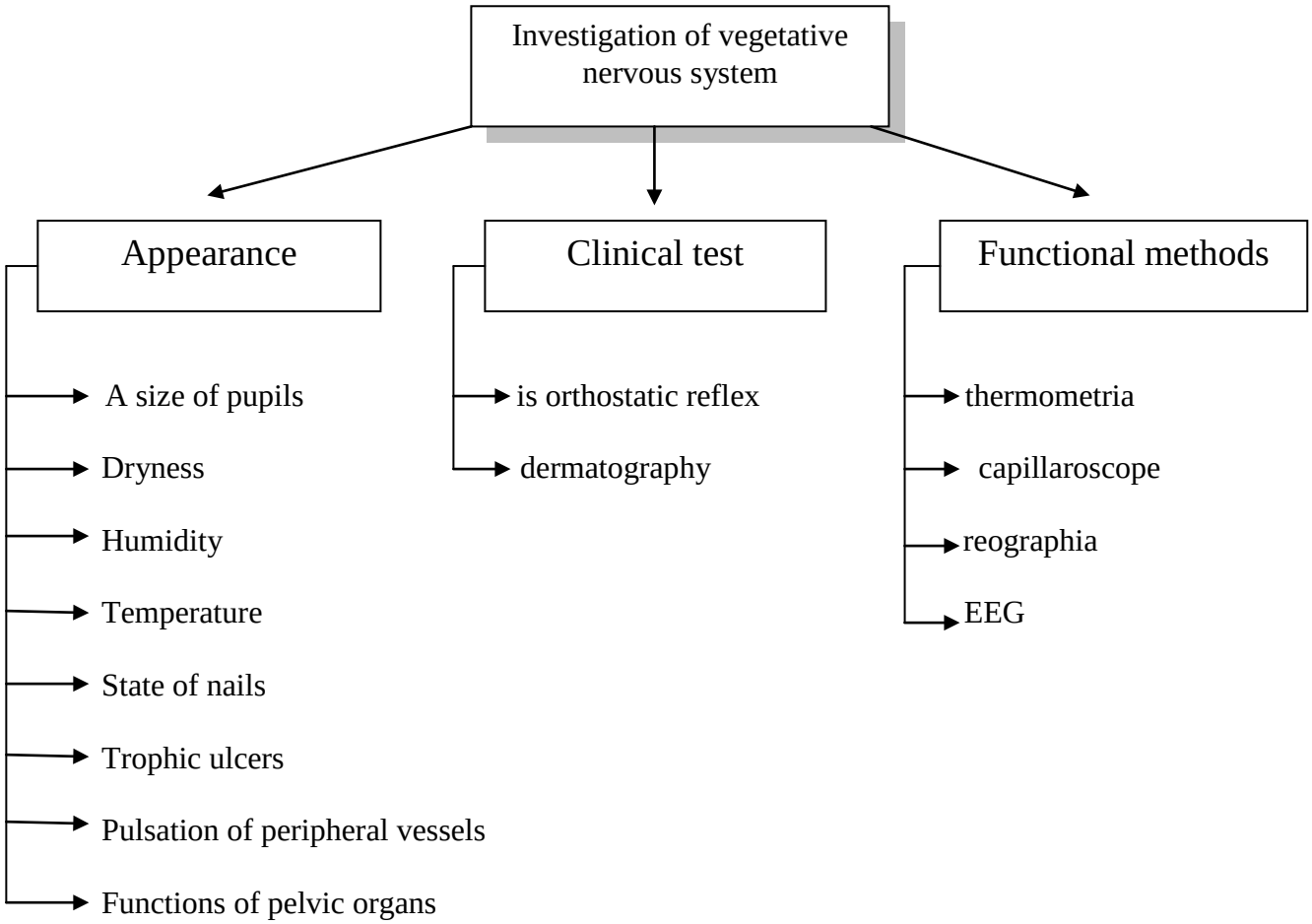


Clinical syndromes of syprasegmentar part

- syndrome of vegetative distonia permanent or paroxysmal type
- psychovegetative (syndrome Berbery-ortostatic hypotension)
- neurovegetative visceral syndrome (sendrome Reyle-Dey) and over
- neurovegetative throphic syndrome

Clinical syndromes of segmentar part

- syndrome vegetative distonia of local
- vegetative-vascular-throhic (local syndrome)
- chronic progressing vegetative deficiency



Influence of likable and parasympathetic nerves is on the function of organs

Organ	Nervous system	
	Sympathetic	parasympathetic
Pupil	Papillary mydriasis	Narrows
Glands (except for sweat)	Weakens a secretion	Increase a secretion
Sweat-glands	Increase a secretion	Absent (not innervation)
Heart	Tachycardia	Bradicardia
Smooth musculature of internalss	Relaxed	Contraction
Vessels (except for coronal)	Contraction	Not innervation
Coronal vessels	Dilatation	Contraction
Sfinkteri	Elevate tone	Relaxed

Tests and typical tasks

1. What structures of the brain belong to the suprasegmental level of the autonomic nervous system?

A. Hypothalamus, limbic-reticular complex

B. Substantia nigra, sympathetic trunk

C. Spinal cord lateral horns, medulla

D. Sympathetic trunk, postganglionary sympathetic fibers

E. Parasympathetic nuclei of cranial nerves, medulla

2. What is the formation of the segmentary level of the sympathetic autonomic nervous system?

A. Hypothalamus

B. Lateral horns cells of C8-L2 segments

C. Limbic system

D. Substantia nigra

E. Medulla

3. What is the formation of the segmentary level of the parasympathetic autonomic nervous system?

A. Reticular formation

B. Lateral horns cells of C8-L2 segments

C. Limbic system

D. X pair dorsal nucleus

E. Hypothalamus

4. What is the craniobulbar formation of the parasympathetic nervous system?

A. Gaulle's and Burdah's nuclei

B. Red nucleus

C. Caudatus nucleus

D. Limbic system

E. Salivatory nuclei

5 What is the function of the suprasegmental level of the autonomic nervous system?

A. Visual function

B. Memory substrate

C. Muscle tone support

D. Sensitive innervation

E. Coordination of movements

6. A patient has a heart rhythm disturbance, heat-like pain in his face, neck and arm. What is the preliminary diagnosis?

7. A patient has bradycardia (heart rhythm lowering), blood pressure lowering, asthmatic breath, and myosis. Name the attack that withholds these symptoms.

8. A patient has a tumor at S3-S5 level. What kind of urinary disturbance does the patient show?

9. A patient has a pale, dry skin, tachycardia, high blood pressure, tremor, fear of dying. Name the attack that withholds these symptoms.

10. Young woman has amenorrhea, diabetes insipidus, trophic disturbances, insomnia, What is the preliminary syndrome?

List of answers:

1. A. 2. B. 3. D. 4. E. 5. B. 6. Vegetative vessel dystonia. 7. Vago-insular attack. 8. Real urinary incontinence. 9. A sympathy-adrenal attack. 10. Hypothalamic syndrome.

6. CRANIAL NERVES AND SYNDROME'S OF ITS LESION

Cranial nerves that start from the brain innervate the skin, muscles, organs of the head and neck and some other organs of the thorax and abdominal cavity nerves. Ill, IV, VI, XI, XII are motor nerves, V, VII, IX, X are both motor and sensory nerves, I, II, VIII are sensory nerves, that support specific innervation of olfactory, optic and acoustic organs. Pairs I and II are brain derivatives, and they don't have nuclei in the brain stem. Other cranial nerves exit from the cranial stem or come into it, where their motor, sensory and vegetative nuclei are located. The nuclei of the pairs III and IV are disposed in the cerebral pedunculi, pairs V, VI, VII, VIII — in the pons tentorium, pairs IX, X, XI, XII — in the medulla oblongata.

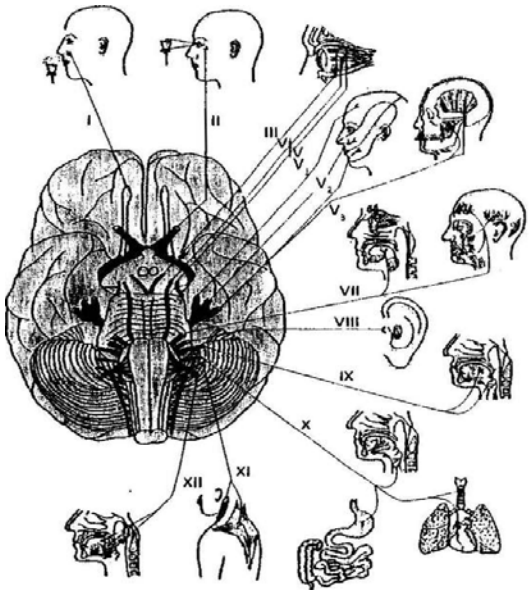
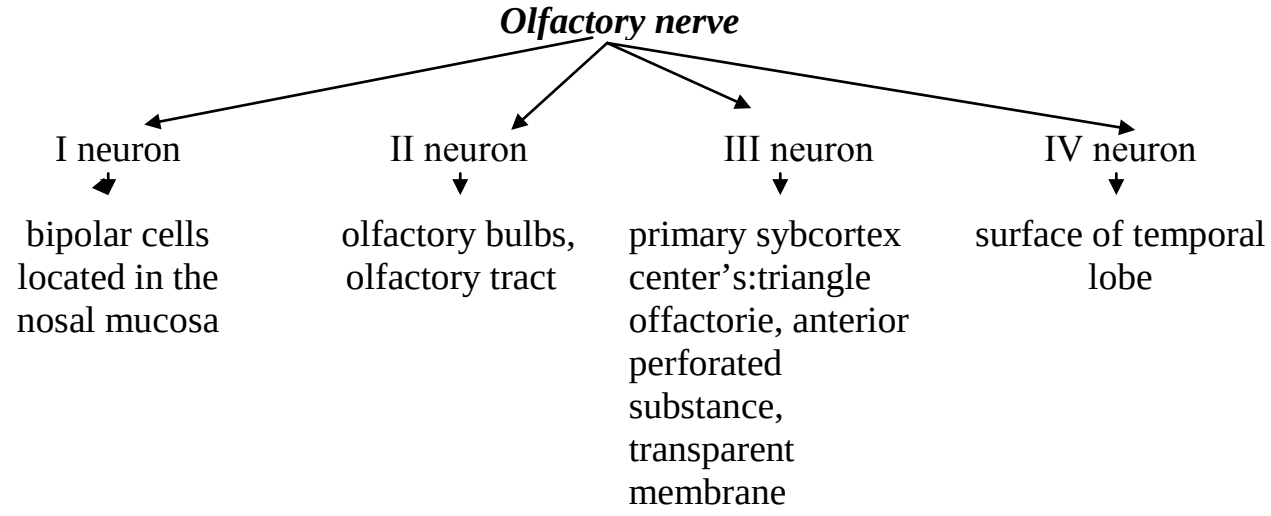


Fig. 5.1. Cranial nerves: I–XII pairs



Research method

suggest to smell aromatic separately by every nostril, closing here other

- mint drops
- butter carnations
- vanilla
- anise
- lavender
- almond water
- perfumeries

Symptoms of defeat I pair of cranial nerves and olfactory to the analyzer in general

- the anosmia loss of sharpness of smell
- the hyposmia decline of sharpness of smell
- the hyperosmia increase of sharpness of smell
- a parosmia is declension of smell

It is important to know that:

- possibility to recognize and identify smells testifies to the maintainance of function of crust center of sense of smell
- at the irritation of peripheral department olfactory to the analyzer (olfactory

filaments, olfactory way) there can be the phenomena of irritation in the kind of elementary smells

- processes on the basal surface of brain (front cranial fossula) can result in an one-sided loss or decline of smell
- processes in the area of primary olfactory centers result in the origin of bilateral loss or decline of smell
- one-sided processes in a cortex (bend of marine horse) more frequent all cause the easy displays of decline of smell only – anymore expressed on an opposite side
- processes in the temporal lobe of cerebrum can cause olfactory hallucinations (various difficult smells)

Visual nerve

Peripheral part

- roots and cones
- bipdar cells
- ganglion cells
- optic nerve
- optic chiasm
- optic tract

Central part

- sybcortex centers:
  - lateral geniculate body
  - upper hillocks
  - pillow of thalamus
- bunch of Gratsiole
- crust center of analyzer
  - spur furrow of cervical fate

Type of research

- acuity of visual
- field of vision the color
- ophthalmoscopy

Examination

Visual acuity

Special tables from 10 rows letters. Patient it is offered to name letters from most to the least from distance 5 meters, checking up the visual acuity for every eye separately.

Norm – the sharpness of sight takes place when an eye is distinguished by two points under the corner of 1° in the distance 5 meters. If the inspected distinguishes 10 lines of letters on a table, both the sharpness of sight is evened 1, if the first row sees only or – 0,1.

Feeling of color

Special colored tables.

Achromatopsia - is the complete ununderstanding of color.

Dischromatopsia - is recognition only of concrete color.

Daltonism - is the innate ununderstanding of color.

Visual field

Checked up for every eye separately by special to the perimeter.

Eye ground

Check up the state of vessels of retina, state of optic disk.

Symptoms of defeat of II pair of cranial nerves and visual to the analyzer in general.

Symptoms of violation of visual acuity:

Amaurosis is a complete loss of eyesight.

Amblyopia is a decline of visual acuity.

Defeat of retina and visile to the nerve result in amaurosis and amblyopia with the loss of direct photoharmose on the proper side.

Symptoms of violation of eyeshots:

A scotoma is a fall of separate area in one of eyeshot's.

Quadrant anopsia – one falling out of four quadrants of eyeshot on both eyes.

Homonymous a hemianopsia is a fall of onenominal parts of eyeshot (right or left).

Heteronymous a hemianopsia is a fall of opposite parts of eyeshot (binasal or bitemporal).

Symptoms of violation of the state of eye ground:

Changes of motion and caliber of vessels of retina.

Papilledema optic disk of visual nerve – at the increased of intracranial pressure

Simple or primary atrophy visile to the nerve.

Second atrophy of visual nerve – more frequent all predefined by the stagnant phenomena or neuritis visile to the nerve.

Retrobulbar neuritis – inflammation visile to the nerve without the damage of optic disk to the nerve.

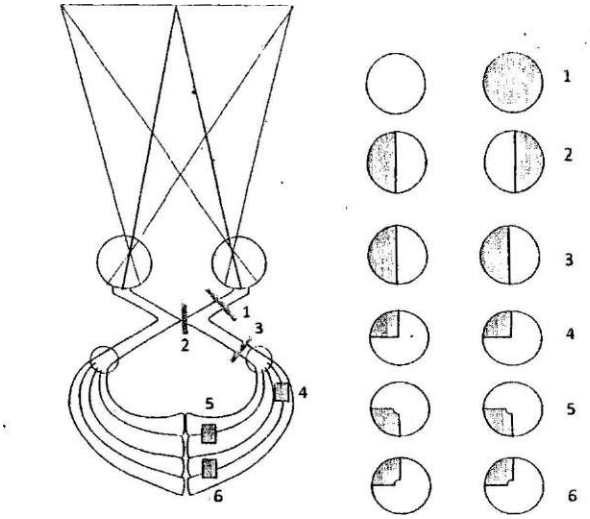


Fig. 5.2. Visual field impairment (explanations in the text)

Oculomotorius nerve

Types of examination of function of nerve:

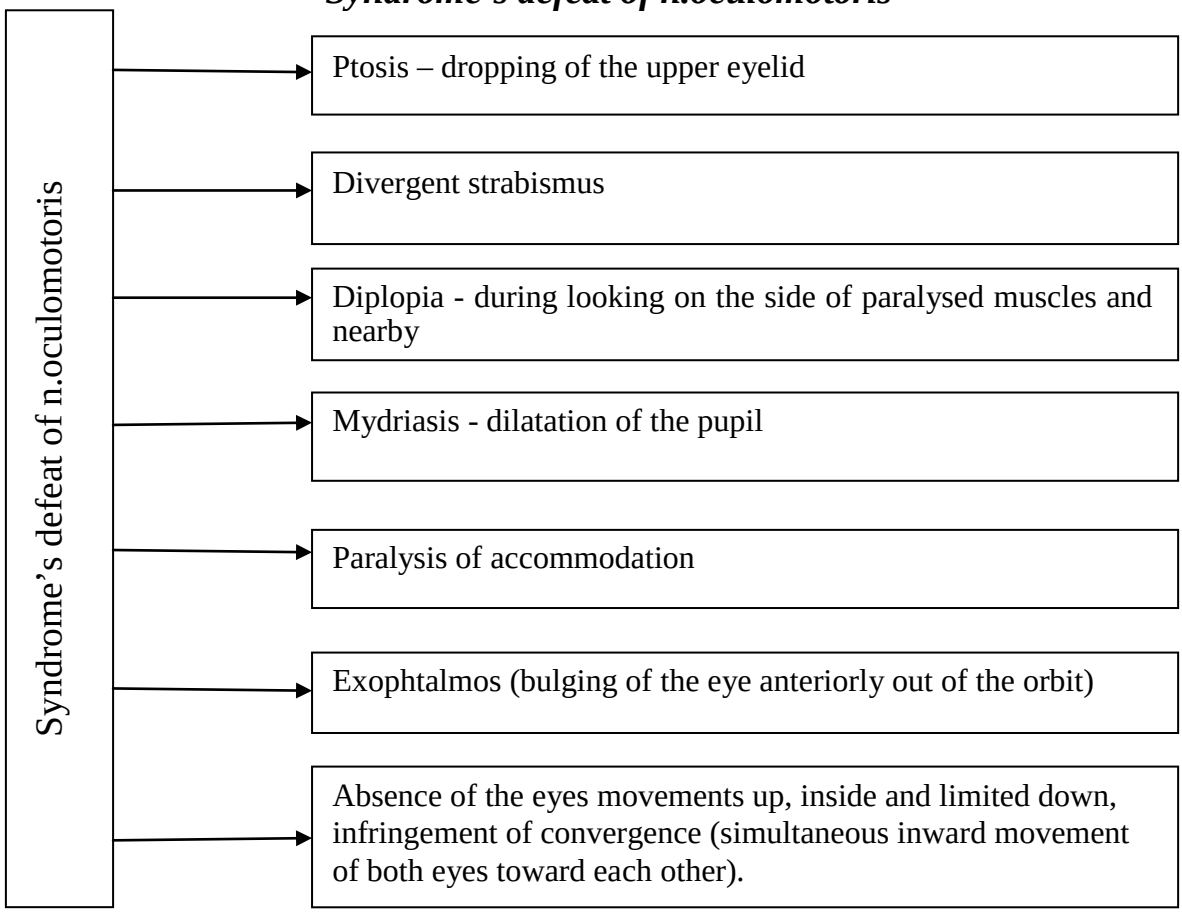
- ✓ position-finding of eyeballs at peace
- ✓ determination of width of eye slit
- ✓ determination of form of pupils
- ✓ estimation of size of pupils
- ✓ mobility of eyeballs
- ✓ fixation of look is at the extreme taking of eyeballs
- ✓ there is a photoharmose of pupils
- ✓ a reaction of pupils is on an accommodation
- ✓ a reaction of pupils is on convergence

Methods of examination of functions of nerve

- ✓ inspection of eyeballs – eyeballs in a norm are located on a middle line symmetric
- ✓ inspection of eye cracks – in a norm have an identical width
- ✓ determination of form of pupils – in a norm have the rounded form, even
- ✓ estimation of width of pupils – by a review
- ✓ volume of motions of eyeballs – suggest to watch a patient a look after a hammer, which is moved up, down, in sides

- ✓ fixation of look at the extreme taking of eyeballs - suggest to watch a patient a look after a hammer, which is fixed in the extreme adduction
- ✓ the papillary reaction of light:
  - direct – suggest to look a patient in distance, then a doctor closes eyes the hands inspected, which under hands remain opened. A doctor in turn subtracts the hands rapid motions from a person, looking after on the state pupils. Narrowing of pupils under the action of direct light name the direct reaction of puppills on light
  - associated is singing a friendly reaction is looked after at the opened eye in the moment of closing or illumination of the second eye.
- ✓ reaction of pupils on an accommodation – suggest to watch a patient after a hammer which is in the distance 50-60 see from a person. At a look in distance of pupil broaden, and at a look to the close located objects - narrow
- ✓ reaction of pupils on convergence – suggest to look a patient in distance, then to the tag of nose approach the hammer of и ask to look at him. There is bringing eyeballs over to the nose (convergence) and narrowing of pupils.

Syndrome's defeat of n.oculomotoris

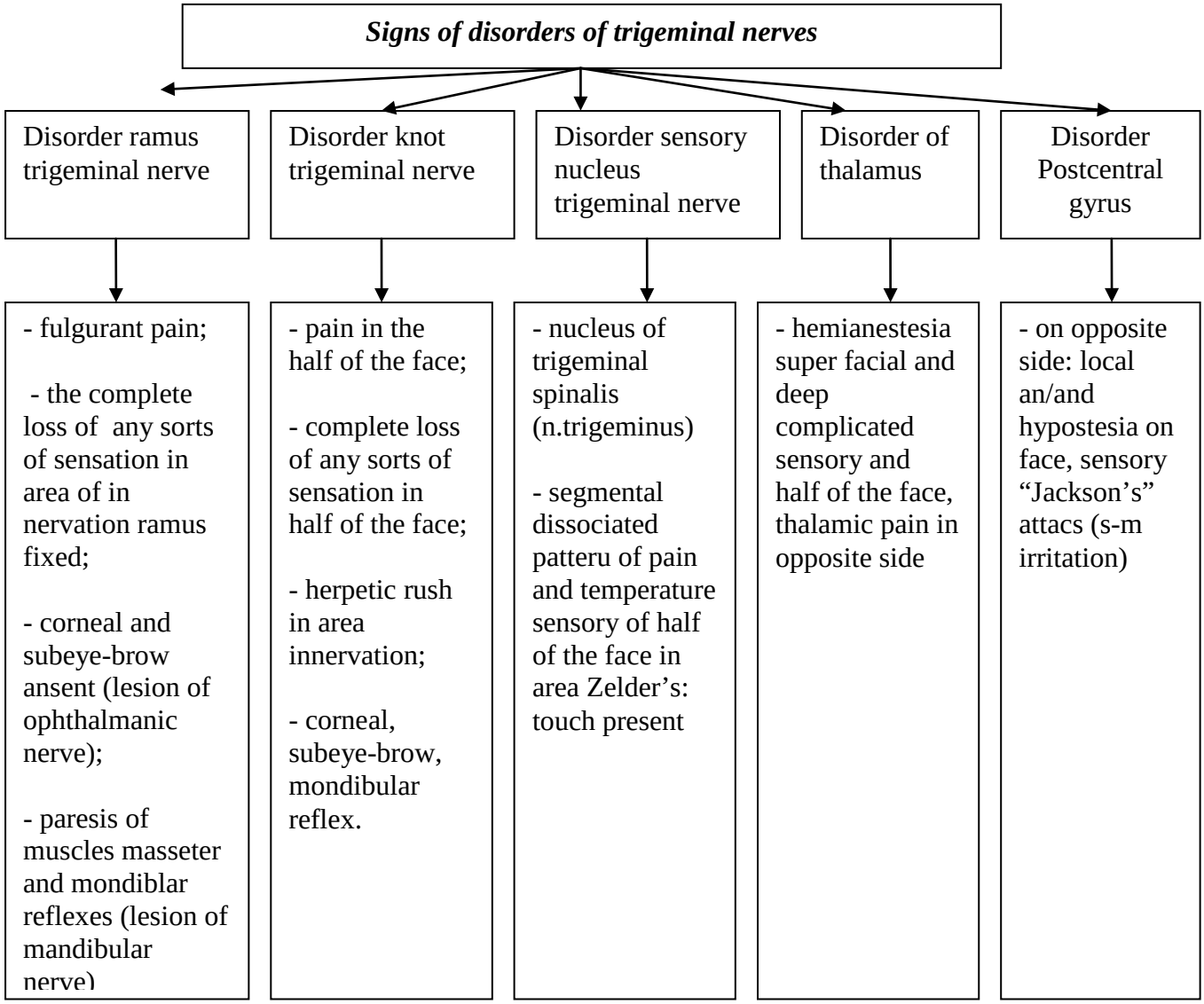


Trigeminal nerve (n. trigeminus)

Trigeminal nerve (pair V) is both a motor and sensory nerve. It consists of three divisions. Two first of them are sensory, the third is mixed, consists of motor and sensory fibres. Sensory fibres supply sensitivity of the face, cornea, sclera, conjunctiva, mucous membrane of the nose and nasal sinuses, oral cavity, tongue, teeth, dura mater. Motor fibers innervate the chewing muscles.

The 1^{st} neuron of the sensory tracts is located in the trigeminal ganglion (Gasser's). Their dendrites form three divisions: first — n. ophtalmicus, second — n. maxillaris, third — n. mandibulars. The last division also contains motor fibres fiom the motor nuclear of the trigeminal nerve in the pons tegmentum. Radicle of the n. trigeminus enters the brain from the lateral surface of the pons. In the pons deep and tactile sensitivity fibres ascend to the upper part of the pons and finish on the n. terrninalis (principalis), where the II neuron is located. Fibres of the superficial sensitivity finish in the nucleus longitudinalis, descending radicle (II neuron), that stretch from the pons to the spinal cord and supply superficial sensitivity of the segmental Zelder's zones. Axons of the second neuron make decussation, join to lemniscus medial and finish in the thalamus, where the third neurons of the sensory tract are located. From the thalamus the third neuron goes to the postcentral gyrus.

Trigeminal nerve examination consists of sensitivity examination of the parts that are innervated by it and chewing muscles function. Complaints of the face pain, pain during palpation of the exit points of the nerve can be identified. Painful, tactile and temperature sensitivities in the zones of nerve innervation and segmental Zelder's zones, corneal, supraorbital, conjunctival, mandibulary reflexes are examined. Chewing muscles atrophy, their tension during palpation and deviation of the mandible to one side when opening the mouth are identified.



Nerve thochlear

A type of examination is a volume of motion of eyeballs.

Examination – suggest looking a patient at a hammer which is moved to the down and outside.

Symptoms of defeat of nerve:

- peripheral paralysis – symptoms arise up on an opposite side, because the fibres of nerve do decussation in a front cerebral sail. At a hemilesion there are doublings of objects at a look downward, limitation of movement of eyeballs downward and outside, strabismus

- a central paralysis does not appear from bilateral cortico-nuclear connections

Nerve abducens

A type of examination is a volume of movement eyeballs at a look outside (right, left).

Examination method – suggest to look a patient at a hammer which is moved outside.

Symptoms of parafunction of nerve:

- peripheral paralysis – at a hemilesion there are doublings of objects at a look in sides, limitation of movements of eyeballs at a look outside, strabismus
- central paralysis does not appear from bilateral corticoconnections

Facial nerve (n. facialis)

The facial nerve (pair VII) gives innervation to mimic muscles. ***The intermediate nerve*** has gustatory, parasympathetic salivatory and lacrimatory fibers j in the facial nerve topographic composition. Gustatory fibers give innervation i to the anterior two thirds of the tongue. Secretory salivatory fibers innervate the submandibular and sublingual (Rivinus') glands. Secretory lacrimatory fibers give innervation to the lacrimal gland.

The facial nerve motor nucleus is situated in the lower part of pons Varolii. The nucleus upper part receives corticonuclear tract from the precentral gyrus of both hemispheres and innervates upper facial muscles. The nucleus lower part receives the corticonuclear tract from the contralateral precentral gyrus and innervates the lower facial muscles.

The facial nerve leaves the brain at its base as well as intermediate nerve fibers close to the vestibulocochlear nerve in the pontocerebellar angle. The intermediate nerve fibers are mostly situated in the medulla oblongata and are common with pair IX (the gustatory nucleus of the solitary tract, the upper salivatory nucleus).

Out of the brain the facial nerve together with the intermediate nerve fibers and auditory nerve go to the internal acustic meatus in the petrosal part of the temporal bone. Here the facial and intermediate nerves enter the facial nerve canal where they give the first branch — ***the greater petrosal nerve*** (n. petrosus major) that consists of

parasympathetic fibers and innervates the lacrimal gland. Involvement of this nerve leads to xerophthalmus (dry eye).

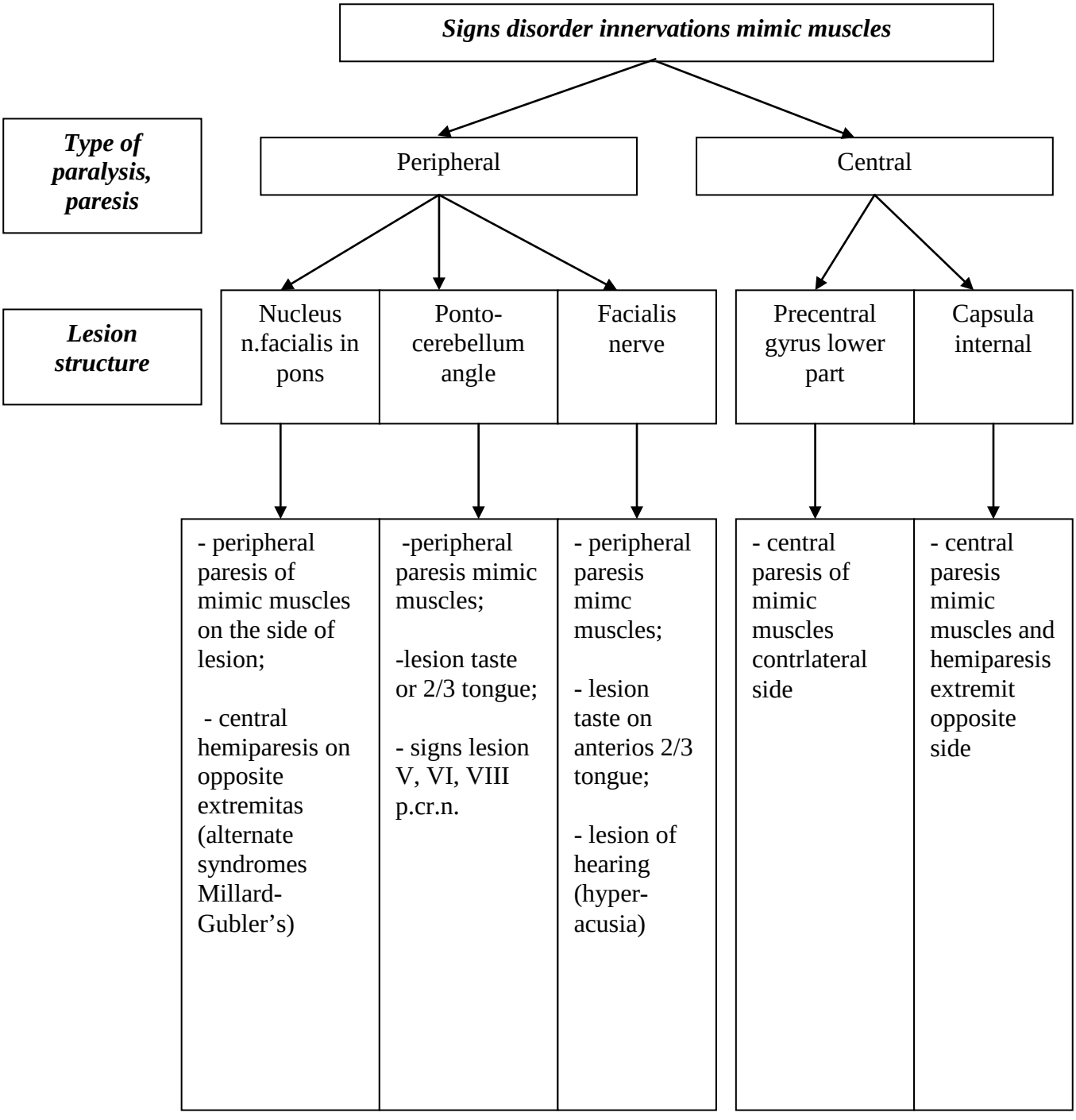
Lower goes ***the nerve to the stapedius muscle*** (n. stapedius) that gives motor fibers in the tympanic cavity. Disorders of this muscle innervations are manifested with hyperacusia — uncomfortable increased sound perception.

Still lower goes ***the cord of the tympanum*** (chorda tympani) that has gustatory fibers for the anterior two thirds of the tongue and salivatory — for the submandibular and sublingual glands.

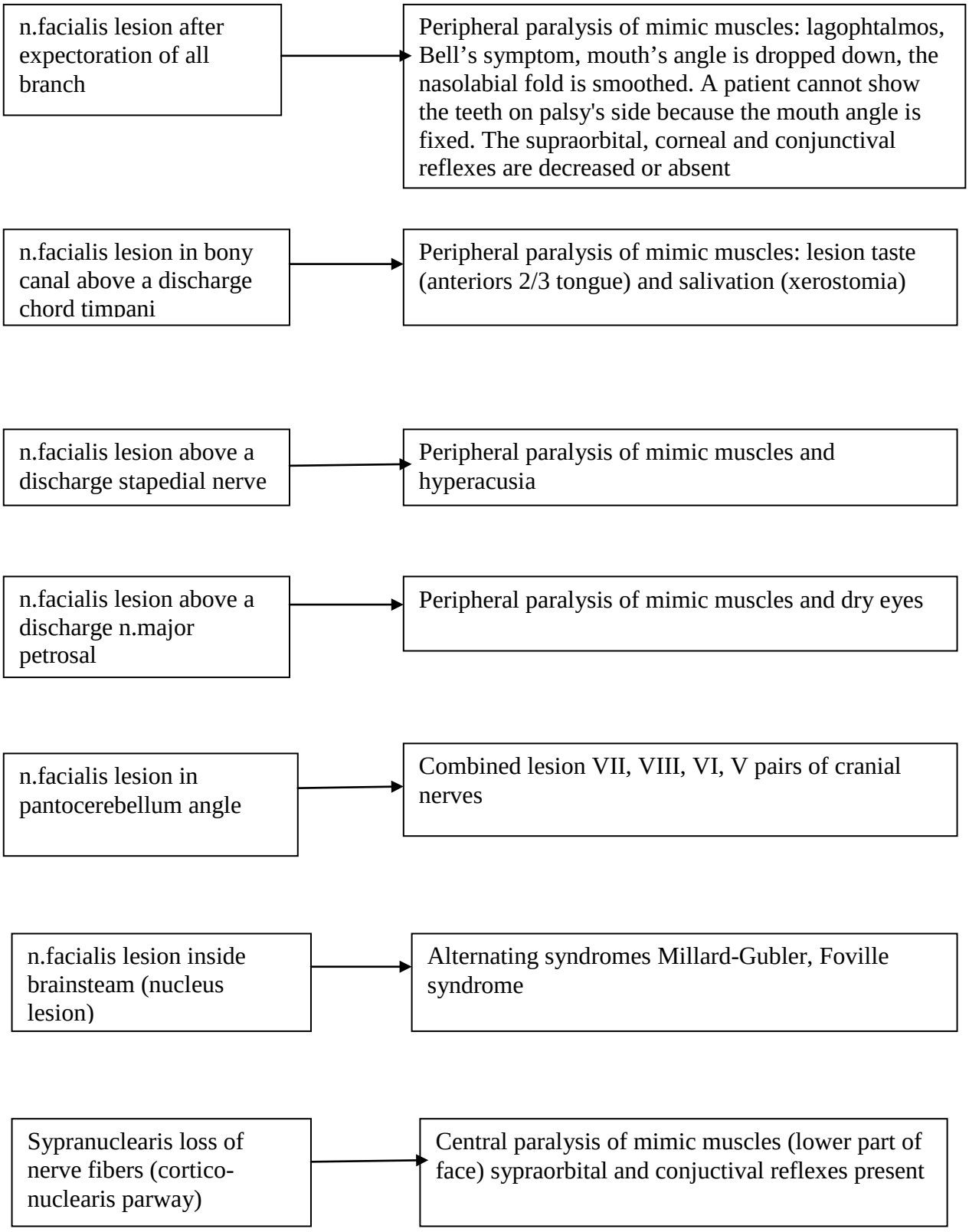
After the cord of the tympanum origin motor fibers of the facial nerve leave the skull through the stylomastoideus hole and is spread to the face as a 'greater goose claw'. The facial nerve innervates all mimic muscles (at the exception of muscle that lifts the upper eyelid).

While examining facial nerve function one defines face asymmetry, flatness of the forehead skin folds and nasolabial fold. The patient is asked to lift up the eyebrows, to frown, to close, the eyes, to wrinkle his nose, to show the teeth, to puff up his cheeks, to whistle, to blow. One considers the presence of watering eye or dryness of an eye conjunctiva, hyperacusia, examines taste on the anterior two thirds of the tongue.

Signs disorder innervations mimic muscles



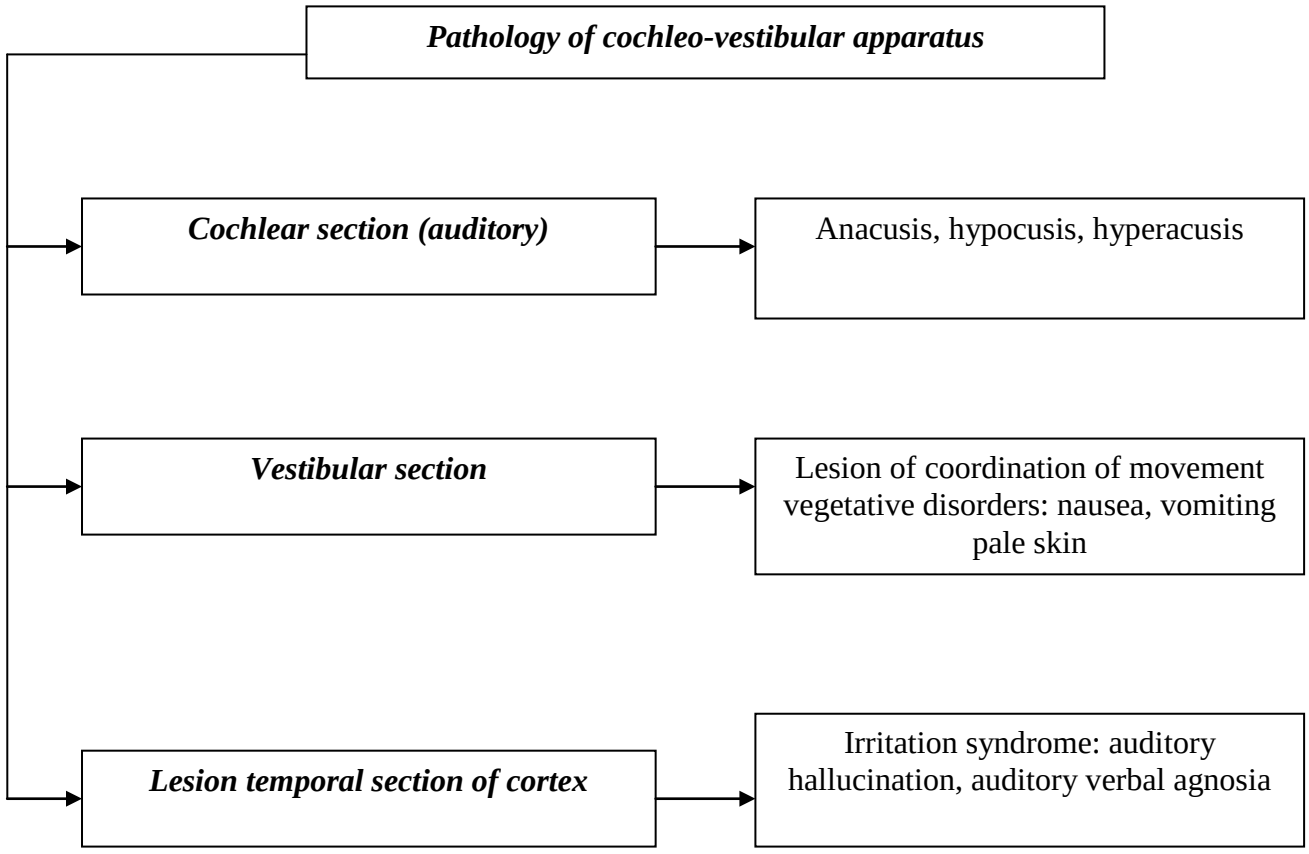
Syndromes of n.facialis defeat on different levels



Vestibulocochlear nerve (n. vestibulocochlear)

The vestibulocochlear nerve (pair VIII) is sensory, consists of two independent nerves — vestibular and cochlear that possess different functions.

	Cochlear nerve		
I neuron	Auditory receptors – hair cells in the organ of corticospinal knot	I neuron	Vestibular receptors in labyrinth. Vestibular knot (in the internal auditory meatus
Pathway	Cochlear nerve in the internal auditory meatus	Pathway	Vestibular nerve (in the internal auditory meatus). Radix vestibular nerve (cerebellopontine angle)
II neuron	Cochlear nucleus of the pons ventral and dorsal	II neuron	Nucleus of the pons: lateral, medial, upper, lower
III neuron Primary auditory cortex	Inferior knolls medial geniculate body Cortex auditory centress (midsection of upper temporal gyrus of both sides	Connection vestibular nucleus	Spinal cord cerebellum nucleus n.oculomotorius, nucleus n.vagusdorsal Reticular formation extrapyramidal system. Cortex of brain



Methods of cochlear nerve function examining include examining of hearing acuity, bone and air conduction. Hearing acuity is examined separately for each ear with the help of whisper and loud speech at the distance of 6-7 meters. Normally a healthy ear hears whisper at the distance of 6-7 meters, loud speech — at 20 meters. Hearing acuity is examined precisely with the help of audiography.

Patient's hearing could be decreased at the disorder of the sound-perceiving or sound-conducting apparatus in the middle ear. Tuning tests are held to define which of the system (sound-perceiving or sound-conducting) is Impaired. Bone and air conduction are usually examined neurologically with the help of tuning fork with frequency of vibration 128 per second.

Rynne test. The vibrating tuning fork is placed on the mastoid. After a patient stops to feel the vibration the tuning fork branches are moved to external acustic meatus at the distance of 1-2 cm. The healthy person hears a sound through the air almost twice longer than through the bone. In this case the test result is registered as positive. If the patient after the bone does not hear a sound through the air this is evident for sound-conducting apparatus impairment (otitis, otosclerosis etc.) and is registered as negative Rynne test.

Veber test. The vibrating tuning fork is placed in the middle of the patient's vertex. Normally a sound is felt with both ears or in the middle. In case of unilateral disorder of the sound-conducting apparatus bone conduction would be better than the air one that's why the patient feels a sound with the sick ear better. In the case of unilateral disorder of the sound-perceiving apparatus (the spiral organ, cochlear nerve) the tuning fork sound is felt better by the healthy ear.

Pathology of the auditory analyzer. There are such disorders as complete loss of hearing, deafness (anacusis), hearing impairment (hypacusis), increased perception (hyperacusis). A one-sided decrease or a hearing loss is possible only during pathology of the inner ear, cochlear nerve or its nuclei (in neurological clinic often because of cochlear nerve neuropathy or its neurinoma in the pontocerebellar angulus). Unilateral impairment of the lateral lemniscus, subcortical auditory centers or cortical part of the auditory analyzer doesn't produce perceptible hearing disturbances. If the pathological

process irritates the cortical part of the auditory analyzer auditory hallucinations occur which could be the aura of the generalized convulsive seizure.

Vestibular nerve (n. vestibularis) is a part of the vestibular analyzer that provides information perception and analysis of the head and body situation in the space. The vestibular nerve conducts impulses from the semicircular ducts and statoconic apparatus of the inner ear. The peripheral neuron of the vestibular analyzer is situated in the vestibular ganglion that is located in the inner ear. Its axons as a part of the vestibular nerve together with the cochlear nerve make their way through the internal acustic meatus toward the vestibular nuclei of the brainstem. These nuclei carry bodies of the second neurons of the vestibular analyzer whose axons go in different directions, providing connection of the vestibular apparatus with cerebellum, the oculomotor group of the nerves nuclei via the system of the medial longitudinal fascicle, with the anterior spinal cord horns, brainstem reticular formation, vagus nerve nucleus and other structures. Numerous connections of the vestibular analyzer explain the presence of different symptoms once it is impaired. The cortical part of the vestibular analyzer is situated in the cortex of the temporal lobe beside the auditory projection area.

Examining the vestibular analyzer's function includes checking the presence of spontaneous nystagmus, balance disturbances, performing coordination tests, defining excitability of the vestibular analyzer with the help of caloric and rotative tests, electronystagmography.

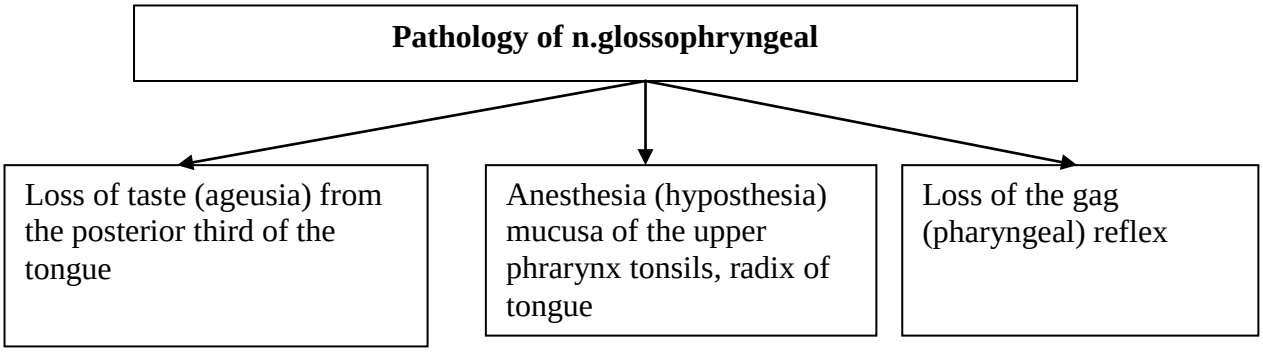
Vestibular analyzer pathology. Vestibular disorders appear as a result of vestibular analyzer injury in any level: because of inner ear diseases, once the vestibular nerve is impaired especially in the pontocerebellar angulus, in the braistem pathology, brain cortex lesions. The leading symptoms of vestibular function disturbances are systemic vertigo and nystagmus. *Vertigo* is a feeling of rotation of enclosing subjects in one direction.

Pathology IX, X, XI, XII cranial nerve. Bulbar and pseudobulbar syndromes.

Glossopharyngeal nerve (n. glossopharyngeus)

The glossopharyngeal nerve (pair IX) is mixed, contains somatic motor fibers, fibers of general and taste sensitivity, parasympathetic secretory fibers. The glossopharyngeal nerve has four nuclei — the motor double nucleus and the nucleus of general sensitivity common with the vagus nerve, also the taste nucleus common with the intermediate nerve and the inferior salivatory nucleus. The nerve leaves the brain in the area of the posterior lateral sulcus of the medulla oblongata behind the olive and the skull — via the jugular foramen.

Motor nerve fibers innervate just one pharyngeal muscle — stylopharyngeal. The nerve provides sensitive innervation of the posterior third of the tongue, the soft palate, throat, pharynx, anterior surface of epiglottis, also of the auditory tube and tympanic cavity. Taste nerve fibers perceive mostly bitter and salt taste irritations from the posterior third of the tongue. Parasympathetic secretory nerve fibers from the inferior salivatory nucleus innervate the parotid salivatory gland.



Clinical lesion: glossophryngeal neuralgia: paroximal pain of the section tonsils, radix tongue radiating to the ear, triggered by swallowing, conversation.

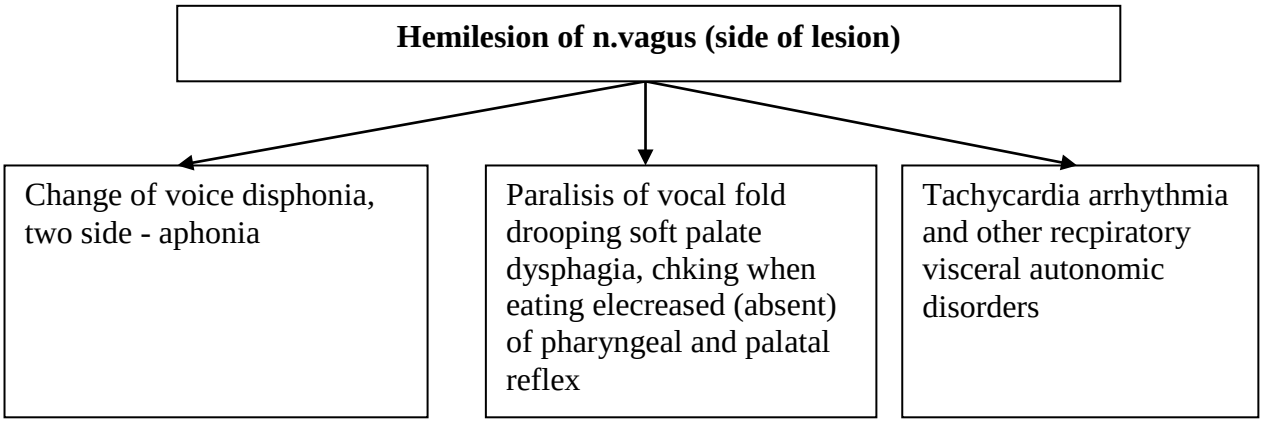
Vagus nerve

The vagus nerve (pair X) is mixed, the longest of all cranial nerves. It contains parasympathetic, somatic motor and sensitive fibers. The nerve has three nuclei, two of which: is the motor and sensitive — are common with the glossopharyngeal nerve; also

there's posterior parasympathetic nucleus. The vagus nerve goes out of the brain in the posterior lateral sulcus of the medulla oblongata and leaves the skull via the jugular foramen. It is situated between the carotid artery and jugular vein; the vagus nerve penetrates in the chest and enters the abdominal cavity via the esophageal foramen of the diaphragm giving numerous branches to innervate the internal organs. Sensitive fibers of the vagus nerve innervate the dura mater of the brain, the depth of the external acustic meatus, pharyngeal mucous membrane, mucous membrane of larynx, trachea, bronchi, lungs, digestive tract and other organs.

Motor somatic fibers of the vagus nerve innervate transversal striated muscles of the pharynx, soft palate, larynx and epiglottis. Motor parasympathetic fibers innervate nonstriated muscles of the trachea, bronchi, esophagus, stomach, small intestine and upper part of the large intestine; secretory fibers go to the stomach, pancreas, inhibitory fibers — to the heart, vasomotor ones — to vessels.

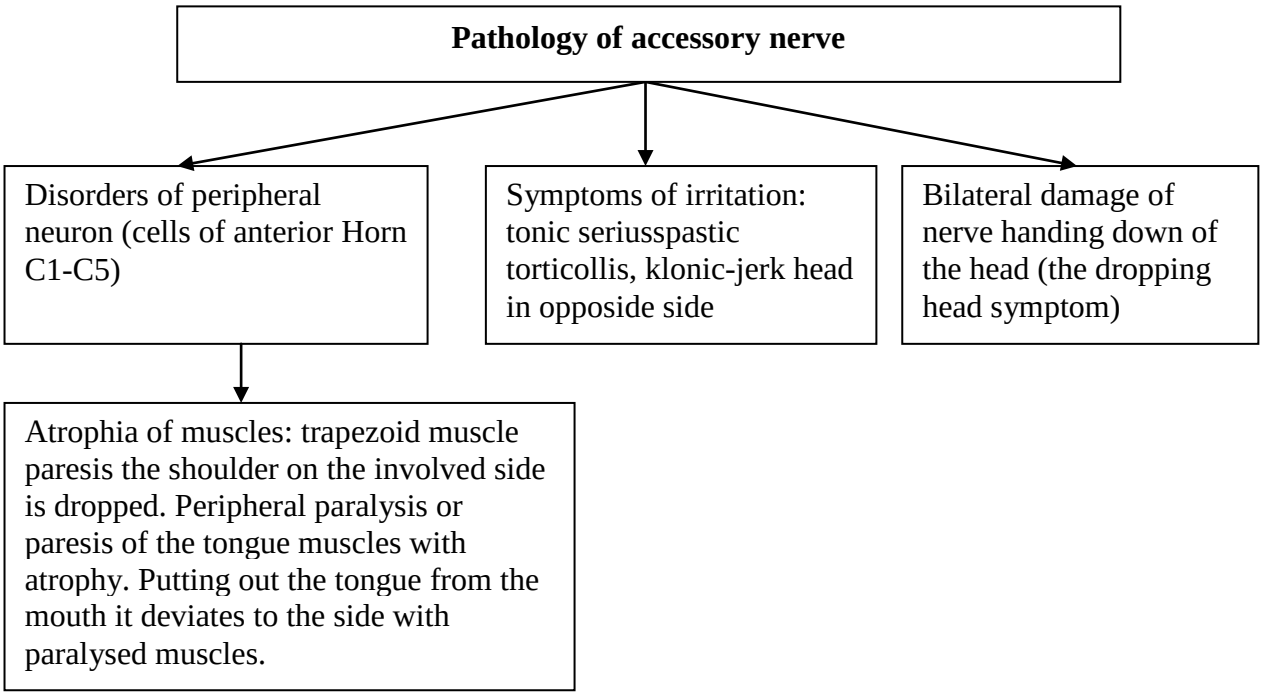
Bilateral lesion compatible of life: disorders of vital function – cessation ofbreathing and cardiac activity.



Examination of glossopharyngeal and vagus nerves function in neurological clinic is carried out usually together. One asks how patient swallows, if there is choking, if the liquid food hits the nose. One should pay attention to voice disturbances (hoarse, snuffling), soft palate drop; follow the mobility of the soft palate while articulating "a", whether the uvula deviates to the side. Examine the taste on the posterior third of the tongue, pharyngeal and palatal reflexes, pulse and breathing rates, blood pressure.

Accessory nerve (n. accessorius)

Accessory nerve (pair XI) is motor; innervates sternocleidomastoid and trapezoid muscles. Long motor nucleus of accessory nerve consists of two parts. The upper (cerebral) part is situated in the lower areas of the medulla oblongata, the lower (spinal) part of the nucleus is placed at the base ot the anterior horns of spinal cord upper cervical segments. The axons of the nucleus spinal part cells leave with thin radices on the lateral surface of the spinal cord and going up unite to the trunk that enters the posterior cranial fossa via the great occipital foramen. Here it joins fibers from the cerebral part of the accessory nerve nucleus that leave the brain in the posterior lateral sulcus of the medulla oblongata. The general nerve trunk leaves the brain via the jugular foramen and innervates muscles pointed.



Examination of accessory nerve function. While examining muscle function that are innervated with the accessory nerve a patient is asked to turn the head to the sides, to tilt it to the front, to lift up the shoulders (to shrug the shoulders), to move the scapulas toward the spine, to lift up the arm higher than the horizontal level.

Pathology. The damage to XI cranial nerves' pair manifests itself with paralysis or paresis of these muscles.

As a result:

1. Of trapezoid muscle paresis the shoulder on the involved side is dropped.

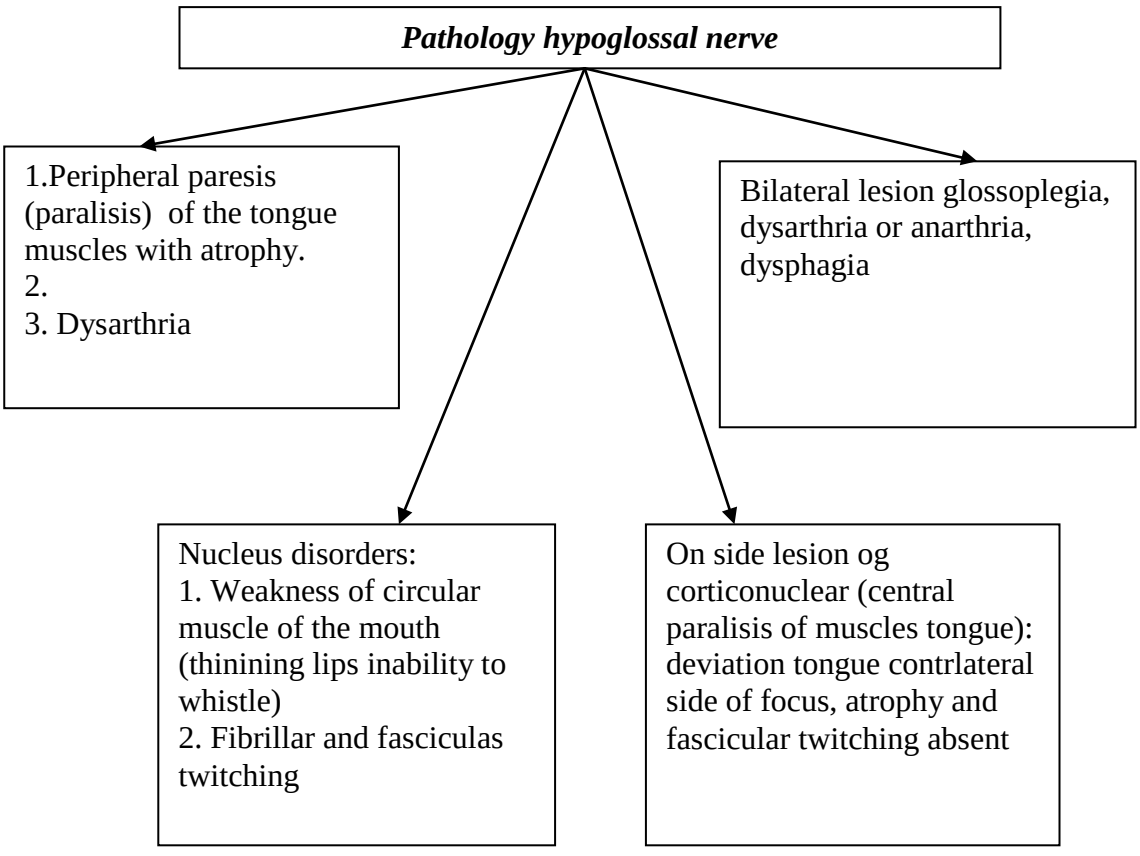
2. The inferior angle of the scapula deviates from the spine to the side and up ("*winged scapula''*).

3. Lifting up the upper extremity higher than the horizontal level is limited.

The weakness of the sternocleidomastoid muscle is shown as disturbed turn of the head to the healthy side, the muscle is poorly outlined. Bilateral damage of accessory nerve manifests itself as hanging down of the head *(the dropping head symptom).*

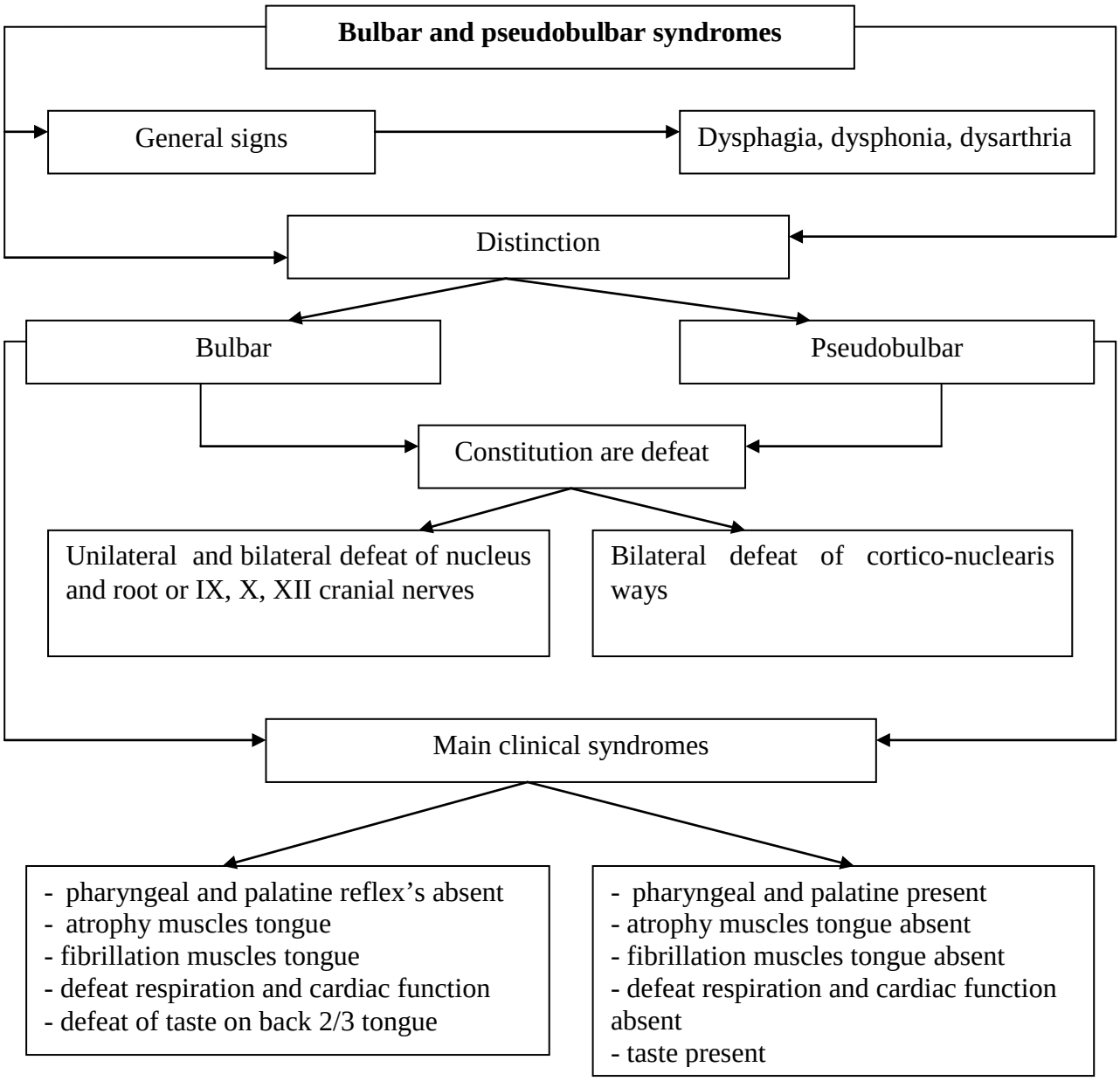
Hypoglossal nerve (n. hypoglossus)

The hypoglossal nerve (pair XII) is clearly motor; innervates tongue muscles. The nucleus of this nerve is situated in the lower area of the medulla oblongata. The nerve goes out of the brain in the anterior sulcus of the medulla oblongata between the pyramid and inferior olive; and leaves the skull via hypoglossal canal of the occipital bone.

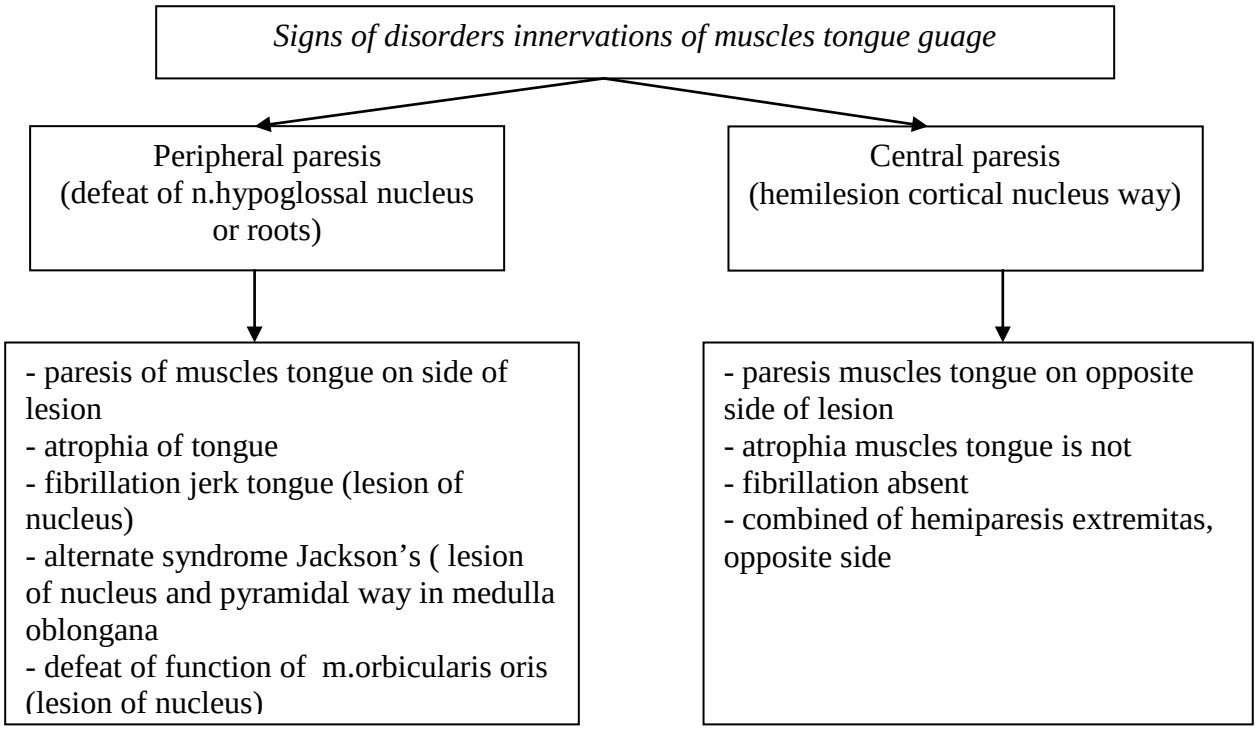


Examination of hypoglossal nerve function. While the patient puts out the tongue one examines whether it is deviated to either side from the medial line. Define the presence of atrophy and fibrillar muscular twitching. Examine the speech articulation.

Bulbar and pseudobulbar syndromes



Signs of disorders innervations of muscles tongue guage



Alternating syndromes

Syndrome name	Ipsilateral symptoms	Contralateral symptoms
Peduncle		
Weber	Diplopia, ptosis of the superior eyelid, divergent strabismus, exophthalmus, mydriasis, disturbances of accommodation and convergence the absence of pupillary light reflex	Central hemiparesis
Benedikt	Diplopia, ptosis of the superior eyelid, divergent strabismus, exophthalmus, mydriasis disturbances of accommodation and convergence the absence of pupillary light reflex	Choreoathetosis Hemiataxia
Claude	Lesion of oculomotour nerve	Cerebellum signs (intension hemitremor, hemiataxia, hyptonia of muscules
Fua		Cerebellum and extrapyramidal signs (intension hemitremor

		choreoathetosis
Pontine		
Foville	Diplopia convergent strabismus peripheral paresis of mimic muscles	Central hemiparesis
Millard - Gubler	Peripheral paralysis of mimic muscles	Central hemiparesis
Medullary		
Avellis	Palate paresis, vocal cord paresis	Central hemiparesis
Schmidt	Palate paresis, vocal cord paresis, trapezius and stemocleldo-mastoideus muscles peripheral paresis tongue peripheral paresis	Central hemiparesis
Jackson	tongue peripheral paresis	Central hemiparesis
Wallenberg - Zakchar-chenko	palate paresis vocal cord paresis Horner syndrome segmental loss of pain, and temperature sensation on the face hemiataxia	Hemianesthesia May be hemiparesis

Syndrome Argyll Robertson: the absence of the direct and consensual pupillary light reflex but presence of the pupillary reaction to convergence and accommodation (in the case of the neurosyphilis) or inverse to it (in the case of epidemic encephalitis).

Syndrome Foster- Kennedy: ipsilateral side – primary atrophy of n.opticus, contrlateral – congestion of n.opticus disk.

Syndrome Pti (irritation of sympathetic nerve): midriasis, exophtalmos.

Syndrome Cloude- Bernara- Gornera (*lesion of centrum ciliospinale)*: simpatico ptosis, miosis, enophtalmos, homolateral angidrosis of fase, hyperemia conjunctive and half of fase.

Toulouse-Hunt (III-V p.cr.n.): pain ophtalmiplegia; signs: one side: retro-periorbital pain, irradiating in temporal region disorders of n.oculomotorius (ptosis, diplopia, exophtalmos), odema of eyelids.

Syndrome lesion of pontocerebella angle: peripheral paresis of V-VIII pair nerves one side: loss of hearing and taste; vestibular ataxia in Pomberg test (balance disturbance in side of focus), pain and peripheral paresis of face, diplopia, convergent strabismus.

Meniere syndrome (lesion VIII p.cr.n): attacs of dissiness (lesion of coordition, nystagmus of side focus), vegetative lesion (hypergidrosis, nosia).

Test and typical test

1. What is characteristic for the defeat of trochlas nerve? What syndrome developed at a patient?

A. Outside cross-eye.

B. Amaurosis.

C. Diplopia at the look downward.

D. Diplopia at the look aside.

E. Enophthalmos.

2. At a patient with a stroke there is peripheral paresis of facial nerve on the side of focus in the trunk of brain, on opposite hemiplegia. What syndrome developed at a patient?

A. Schmidt's syndrome

B. Avellis's syndrome.

C. Millard-Gubler syndrome

D. Foville's syndrome.

E. Benedict's syndrome

3. Damage to what part of the visual analyzer is the cause of amaurosis?

A. Thalamus

B. Internal capsule

C. Optic tract

D. Optic nerve

E. Occipital lobe

4. Indicate the localization of the pathological center in the case of alternating Weber syndrome

A. Oculomotor nerve

B. Abducens nerve

C. Cerebri peduncle

D. Pons

E. Medulla

5. A patient has the Foville's syndrome. What pair of cranial nerves is involved in a pathological process?

6. At a patient converging cross-eye, dyplopia in a right side. Is a function, what nerve broken?

7. A patient has pathology of main vessels, cerebral atherosclerosis. After the physical loading at a patient hemianesthesia appeared on face, paresis of muscles of soft palate and vocal fold, the Horner's syndrome, cerebellum ataxia in extremities, contrlateral – hemianesthesia, hemyparesis. What syndrome developed at a patient?

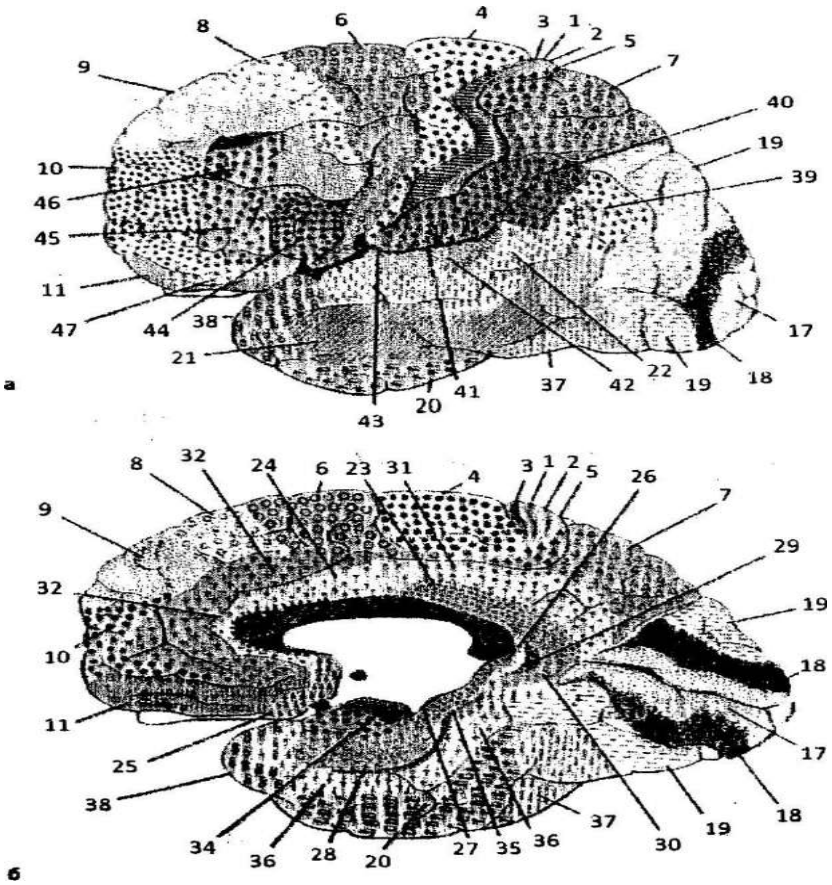
8. A patient has the peripheral paralysis of language, soft palate, vocal cords on the right; lateral - high tendon reflexes, pathological reflexes, hemyparesis in combination with hemianesthesia. What syndrome developed at a patient?

9. A patient has Syphilis. In neurological status: absence of direct and concord reaction of pupils on light at safety reactions on convergence and accommodation, anisocoria, deformation of pupil. What syndrome developed at a patient?

10. A patient has the Foville's syndrome. What pair of cranial nerves is involved in a pathological process?

List of answers: 1.C. 2.C. 3.D. 4.C. 5. VII, VI pair of cranial nerves. 6. Abduncens nerve on the right. 7. Wallenberg’s-Zakcharchenko syndrome. 8. Avelis’s syndrome. 9. Agyll-Robertson syndrome. 10. Cerebello-pontine angle syndrome.

7. THE BRAIN CORTEX. DISTURBANCES OF HIGHER CEREBRAL CORTEX



Brodmann's map of the brain cortex'

The *primary projection areas* are those that receive most of their sensory impulses directly from the thalamic relay nuclei:

- primary somatosensory cortex, *Brodman areas 1, 2, 3,*
- the visual, *area 17,*
- the auditory, *areas 41, 22,*
- the olfactory, *areas 27, 28.*
- primary motor cortex, *area 4*

Secondary projection areas:

- motor, *areas 6, 8*
- sensory, *areas 5, 7a,*
- visual, *area 18, 19,*
- auditory, *area 42*

Main tertiary projection areas: 7b, 9, 10, 37,39, 40, 45.

Higher cerebral functions

- speech and language
- reading and writing
- praxis and gnosis
- memory
- mentality
- consciousness

Methods of higher cerebral functions examining include defining a disturbances of consciousness — confusion, stupor, coma. The degree of consciousness impairment is estimated according to Glasgow Coma Scale (GGS). A doctor has to determine patients' perception of themselves and their environment, behavior and responses to external stimuli. In the normal state of consciousness, a patient is fully conscious, oriented and awake.

Examining a comprehension (receptive) and spontaneous (expressive) speech includes determination of motor (Broca's), sensory (Wernicke's) and amnestic aphasia.

While examining higher cerebral functions one defines counting, reading, writing, gnosis and praxis.

Disturbances of higher cerebral functions Speech and language disorders

Language disorders are termed *aphasias,* and involve language disturbances in comprehension, production, or both.

Speech disorders are termed:

- *dysarthria,* a disturbance in articulation,
- *dysphoria,* a disturbance in vocalization or phonation.

Patients with dysarthria or dysphonia retain their language ability despite their speech disturbance.

Description of the symptom is important in characterizing and differentiating speech and language disorders;

► difficulty in articulation or vocalization implies a speech disorder,

► whereas the inability to find words, comprehend, read, or write is indicative of a language disorder.

Differentiation of speech and language disorders has an important localizing value for underlying pathology within the nervous system and helps distinguish among different etiological processes.

Kinds of aphasias

Broca's aphasia (also called anterior, motor, or expressive aphasia) is characterized by the absence or severe impairment of a spontaneous speech, while comprehension is only mildly impaired.

Wernicke's aphasia (also called posterior, sensory, or receptive aphasia) is characterized by severe impairment of comprehension. A spontaneous speech remains fluent and normally paced, but *paragrammatism*, *paraphasia*, and *neologisms* make the patient's speech partially or totally incomprehensible (word salad, jargon aphasia).

Amnestic (anomic) aphasia. This type of aphasia is characterized by impaired naming and word-finding. A spontaneous speech is fluent but permeated with word-finding difficulty and paraphrasing. The ability to repeat, comprehends, and writes a word is essentially normal.

Global aphasia involves all aspects of language and severely impairs spoken communication. A patient cannot speak spontaneously or can only do so with a great effort, producing not more than fragments of words. Speech comprehension is usually absent; at best, patients may recognize a few words, including their own name.

Disturbances of higher cerebral functions

Apraxia. While some patients complain of a *loss of ability to perform skilled movements*, most do not recognize their disability or complains about it. The activities to be inquired about include the use of common bathroom tools such as toothbrushes, razors, combs, and brushes.

Agnosia — the inability to recognize a known object. May be visual, auditory, gustatory and olfactory agnosia.

Agraphia may be defined as a loss or impairment of the ability to produce written language, caused by brain dysfunction.

Acalculia — the inability to count.

Autotopagnosia — the inability to identify one's own extremities and other parts of the body.

Anosognosia — the inability to identify one's own defect, for example, hemyparalysis.

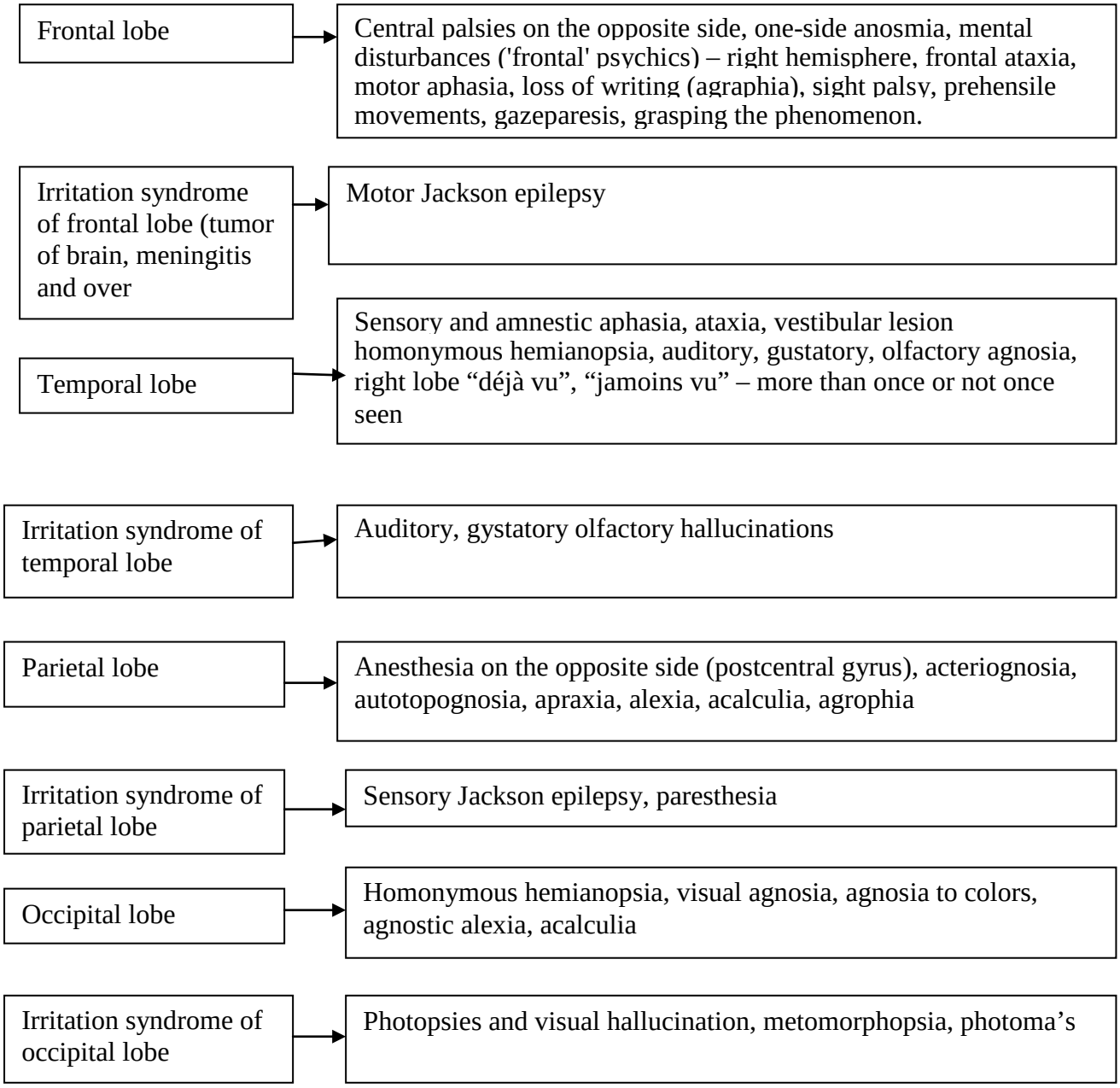
Syndromes of brain cortex

Right hemisphere

- central palsies on the opposite side,
- autotopagnosia,
- anosognosia,
- mental disturbances,
- parakinesia — continual movement in the right extremities.

Left hemisphere

- central palsies on the opposite side,
- aphasia,
- alexia,
- agraphia,
- acalculia,
- apraxia.



Test and typical tasks

1. At patient has ischemic stroke in right hemisphere. In neurologic status: homonumous hemianopsia. What is localization of focus defeat?

A. Frontal lobe.

B. Parietal lobe.

C. Occipital lobe.

D. Brainstem.

E. Temporal stake.

2. Patient grumbles about appearance before the eyes of asterisks, flashings sparks. What is the syndrome of irritation?

A. Sleep the state.

B. Parastesia.

C. Photopsia.

D. Metamorfopsia.

E. Metafotopsia.

3. The sensory aphasia is characterized by:

A. Loss of ability to name familiar subjects

B. Disturbance of speech comprehension

C. Disturbance of expressive speech

D. Speech dysarthria

E. disturbance of complex logically-grammatic structures comprehension

4. Where is the cortical part of the olfactory analyzer localized?

A. Frontal lobe

B. Occipital lobe

C. Anterior central gyrus

D. Temporal lobe

E. Postcentral gyrus

5. What pathology you will see when precentral gyrus is damaged?

A. Hemianesthesia

B. Tactile amnesia

C. Monoplegia

D. Hemianopsia

E. Olfactory agnosia

6. Name type of aphasia:

A. Motor

B. Dysarthria

C. Dysphonia

D. Alexia

E. Mutism

7. At patient has hemiparesis left-side, astesia, abasia, apatic frontal mental disordes. Positive grabbing phenomen on of Yanishevskiy. What is localization of pathology focus?

8. At patient is delivered in a neurologic clinic from a street. A contact with a patient is limited from an allolalia. In neurologic status: motor aphasia, alexia, paresis of look aside opposite a focus, right-side hemiparesis. What is localization of pathology focus?

9. A patient grumbles about the loss of usual skills (can not be independently dressed), can not read, write, consider, does not know objects by touch. Objectively: violation of all of types of sensitiveness on a right arm and right half of face, apraxia, agraphia, acalculia, lateral aphasia. What is localization of pathology focus?

10. Patient complaint about visual hallucinations as «lightning's», «sparkling stars». Where is lesion localized?

List of answers: 1. C. 2. C. 3. B. 4. D. 5. A. 6. A. 7. Frontal lobe on the right. 8. Frontal lobe on the left. 9. Parietal lobe on the left. 10. Visual illusions.

8. DISTURBANCES OF CONSCIOUSNESS

Different degree of weght

Degree of weght	Reaction on the pain, reflex	Visual optic signs	Position of extremities	Respiration	Cardio vascular	Level of affect
Spoor	Reaction of pain and reflexes present	Pupils narrows raction on light present	Voluntary movements present	Violation: Cheyne-Stoks often	Tachycardia Normotonia	Thalamus
Coma I	Reaction of pain and reflexes present	Pupils narrows raction on light absent	Absent	Central hyperventilation	Tachycardia Arterial hypotension	Midbrain
Coma II	Reaction of pain and reflexes absent	Pupils pinctate movents of the eyes and reaction of light absent	Rigidity of decerebration	Periodic apnea	Tachycardia Arterial hypotension	Pons
Coma III (atonic)	Reaction of pain and reflexes absent	Pupils narrow reaction of light absent	Atonia	Rare, superficial	Arterial hypotension (low 60 mm rt. arrhythmia	Medulla oblongata

Cerebral causes of persistent loss (coma) or disturbance of consciousness

Intracranial cause	Clinical features	Investigations
Epilepsy	Status epliepticus Convulsive movements Incontinence Tongue biting	EEG CT or MRI
Trauma Extradural haemorrhage	Evidence of head injury – laceration, bruising, blood or CSF from nose orear	Fracture on skull X-ray CT or MRI of head
Subdural haemorrhage	Past history of head injury several weeks previously: the elderly and alcoholics are prone to falls and tearing of penetrating veins	CT or MRI of head
Vascular Subarachnoid haemorrhage	History of explosive-onset headache Collapse Subhyaloid haemorrhage Pyrexia Neck stiffness, +ve Kernig's s sign Focal neurological signs if there is intracerebral extension of the blood	CT of head – should be perfomed within 48 hours to avoid false negative results Lumbar puncture (LP) – if CT is normal, an LP shoud be done between 12 hours and 1 week after the initial event – xanthochromic CSF

Intracerebral haemorrhage	Collapse, seizures, focal signs	CT or MRI head
Hypertensive encephalopathy	Hypertension, retinopathy, seizures, nephropathy	Investigation of secondary hypertension
Vertebrobasilar thromboembolism	History of TIAs or stroke, valvular or ischaemic heart disease, predisposition to thromboembolism	MRI head MRA vertebrobasilar system
Infecton Meningitis	Headache Pyrexia Neck stiffness, +ve Kernig's s sign	If there is no evidence of raised intracranial pressure, an LP should be done; otherwise CT should be perfomed first
Encephalitis	Clouding of consciousness, confusion, behavior and memory disturbance, headache, pyrexia, seizures, focal isgns	CT or MRI – focal or diffuse cerebral oedema EEG, LP
Cerebral abscess	Subacute onset Headache Usually focal symptoms and signs Often seizures Source of infection elsewhere – ears, sinuses, lungs, valvular heart disease	CT or MRI of head Blood cultures Microbiology from potential sourse of infection

Extracerebral causes of persistent loss (coma) or disturbance of consciousness

Intracranial cause	Clinical features	Investigations
Circulatory collaps Cardiac, e.g. arrhythmia, myocardial infarction	Hypotension, tachycardia, rhythm disturbance, cardic failure	EEG, cardiac enzymes, echocardiogram
Septicaemic chok	Rigors, pyrexia, vomiting, peripheral vasodilatation	Cultures – blood, sputum, urine, throat, stool
Hypovolaemia, e.g. blood loss, profuse diarrhoea	Melaena, haemetemesis, abdominal pain (ruptured aortic aneurysm)	
Hypotensive drugs		
Metabolic Hypo- or hypernatraemia	**Subacute onset** Muscle twitches, dehydration	Biochemical confirmation
Hypo- or hypercalcaemia	Carpopedal spasm Polyuria and/or polydipsia, abdominal pain, vomiting	
Hypo- or hyperglycaemia	Hemiplegia (reversible), seizure Dehydration, hyperventilation, ketotic fetor	
Hypo- or hyperthermia	Bradycardia, hypotension, hypoventilation, rigidity, cardiac arrest	Recal temperature
Uraemia	Sallow skin, uraemic fetor, pale conjunctivae, hypertension	Raised creatinine, acidosis, anaemia
Hepatic failure	Jaundice, signs of portal hypertension, GI bleed, sedative drugs, sepsis	Abnormal LFTs, raised ammonia, electrolyte imbalance

Hypoxia	Following respiratory failure, cardiorespiratory arrest; brain damage occurs after 4 mitutes of anoxia	
Hypercapnia	Bounding pulse, papilloedema, asterixis	Elevated pCO_2
Endocrine Adrenal crisis	Hypotension, abdominal pain, buccal and flexure pigmentation	Low Na^+, Ca^{2+}, glucose Raised K^+, urea, Synacthen test
Hypopitutarism	Pallor, hypogonadism, bitemporal hemianopia	Low pituitary and target hormones, CT/MRI of head
Myxoedema	Dry and coarse facies, hypotension, bradycardia, hypothermia	Clinical diagnosis confirmed by thyroid function tests; pituitary failure
Hypo- or hyperparathyroidism	As for hypo- and hyperparathyroidism	↓/↑ Ca^{2+}, ↓/↑ parathyroid hormone
Toxins Alcohol	Ethanolic fetor Wernicke's encephalopathy	Raised blood alcohol level Low thiamine
Carbon monoxide	Pink skin colour	Elevated carboxyhaemoglobin
Drugs Opiates	Slow respiratory rate, pin-poin pupils	Reversible with naloxone +ve blood and urine toxicology
Sedatives	Barbiturates – hypotension and hypothermia	+ve toxicology
Tricyclic antidepressante	Dilated pupils with sluggish responses, hypothermia	
Psychiatric Psychogenic coma	Eyelids resist opening: normal reflexes and plantar responses; corneal reflex cannot be suppressed; normal optokinetic and caloric responses	

Examination of the comatose patient

Examine the partient for:

- ✓ sings of head injury
- ✓ neck stiffness (if no evidence of cervical spine injury)
- ✓ respiratory pattern
- ✓ pupil responses
- ✓ ocular movement

- ✓ fundoscopic abnormalities
- ✓ limb posture and spontaneous movements
- ✓ reflexes and plantar responses
- ✓ assess Glasgow Coma Scale

Signs of head injury

Lacerations and bruising may be present occur over an underlying fracture. A basal sracture may be present with a normal skull X-ray. It is important to look for evidens of an anterior fossa fracture such as rhinorrhoea, bilateral periorbital haematoma, and subconjunctival haemorrhage. A fracture of the petrous bone may produce cerebrospinal fluid (CSF) or blood otorrhoea and be associated with Battle's sign (swelling and bruising over the mastoid process).

Investigations in the comatose patient

Immediate investigations include:

- ✓ temperature
- ✓ blood glucose
- ✓ electrolytes, calcium, urea, and creatinine
- ✓ full blood count and coagulation screen
- ✓ arterial blood gases
- ✓ blood culture and toxicology
- ✓ EEG and chest X-ray

Neurological investigations in selected cases include:

- ✓ brain imaging (CT or MRI)
- ✓ CSF examination
- ✓ EEG
- ✓ Angiography

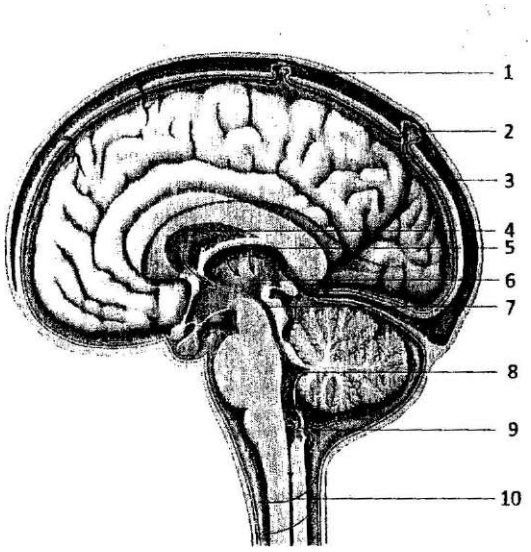
Prognosis of coma

In patient where a drug overdose or a reversible metabolic cause coma and there has not been a prolonged period of unsupported cardiorespiratory failure, then the prognosis can be excellent with appropriate critical care. Unfortunately, other causes have a poorer prognosis. In patient with a Glasgow Coma Scale score of 3 for more than 6 hours there is mortality of over 50% and, of the remainder, only a small minority will return to independent existence.

9. CEREBROSPINAL FLUID. MENINGEAL AND HYPERTENSIVE SYNDROMES

The brain and spinal cord are invested by three meningeal membranes called the dura mater, arachnoid, and pia mater. The ***subarachanold space*** lies between the arachnoid and pia mater. It is filled with cerebrospinal fluid. ***Ventricles*** are called the spaces which are filled with cerebrospinal fluid and protect the brain by cushioning it and supporting its weight. The two lateral ventricles extend across a large area of the brain. The third ventricle lies between two thalamic bodies. The fourth ventricle is located between the cerebellum and the pons.

Cerebrospinal fluid (CSF) is a clear, colourless liquid that surrounds the brain and spinal cord and fills the spaces in them. CSF arises from the blood and returns to it at a rate of about 500 ml a day. In the adult the total volume of CSF is about 150-200 ml. The circulation of CSF is completely replaced about every 4 hours. CSF is produced by the choroid plexus located in the lateral, third and fourth ventricles. From the lateral ventricles CSF drains through the two foramina of Monro, which are also known as the interventricular foramina, into the third ventricle, and then through the aqueduct of Sylvius into the fourth ventricle.



Ventricles and the direction of cerebrospinal fluids flow:

1 — superior sagittal sinus;

2 — arachnoid villi;

3 — subarachnoid space;

4 — lateral ventricles;

5 — choroid plexus;

6 — third ventricle;

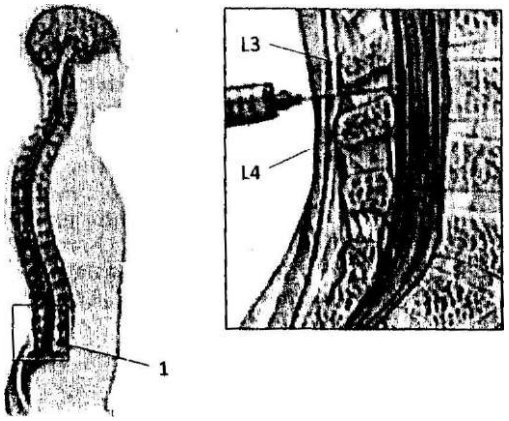
7 — aqueduct of Sylvius;

8 — fourth ventricle;

9 — the central canal of the spinal cord

From the fourth ventricle, the CSF flows into the subarachnoid space through the foramina of Magendie and Luschka to the 'basal cisterns' under the brain. After that the flow of CSF is mainly up and over the whole brain surface, partly flows down around the spinal cord. Completing the circuit back to the bloodstream, the fluid drains via the valve-like arachnoid villi (arachnoid granulations) into the sagittal sinus. Some of CSF is also taken up into veins around spinal nerve roots and into the lymphatic system. Physicians frequently perform lumbar puncture. ***Indications to lumbar punctures*** are suspected CNS infection, subarachnoid hemorrhage, central nervous system tumors. ***Contraindications to lumbar punctures*** are local skin infections over a given puncture site, raised intracranial pressure. Lumbar puncture is

performed by inserting a needle between the L3-L4 or L4-L5 below the lower end of the spinal cord (the spinal cord ends near L1-L2). Routine examination of CSF includes visual observation of color and clarity and commonly performed tests: a quantitative assessment of protein and glucose levels, cell counts and differential, microscopic examination and culture. Additional tests such as measuring pressure and a polymerase chain reaction also may be performed. Knowing which tests to order and how to interpret them allows physicians to use CSF as a key diagnostic tool in a variety of diseases.



Lumbar puncture: 1 — the spinal column

Pathology

Xanthochromia is most often caused by the presence of blood, but several other conditions should be considered. The presence of blood can be a reliable predictor of subarachnoid hemorrhage. An increase of white blood cells in CSF may occur in case of viral, bacterial, fungal, and parasitic meningitis. The white blood cells differential helps to distinguish these causes. For example, viral infection is usually associated with an increase in lymphocytes, while bacterial and fungal infections are associated with an increase in neutrophils. A low glucose level occurs in bacterial meningitis. Tuberculoses meningitis is characterized by an increase in lymphocytes, a decrease of glucose and chlorides in CSF. High total proteins levels in CSF are seen usually in tumors.

There are two kinds of quantitative indices dissociation in liquor: cells- protein and protein-cells dissociation. *Cells-protein dissociation* means that the cells count increases more than the protein level or proteins level is normal. Cells-protein dissociation occurs in meningitis. *Protein-cells dissociation* means that the protein level increases more than cells or the cells level is normal. Protein-cells dissociation is caused by tumor. An increase of lymphocytes in CSF is called lymphocyte pleocytosis, occurs in the case of serous meningitis. Raise of neutrophils — neutrophil pleocytosis, occurs in the case of purulent meningitis.

The cultural method is the gold standard for determining the causative organism in meningitis. However, a polymerase chain reaction is much faster and more sensitive in some cases.

CSF *hypertension syndrome* is caused by an increased intracranial pressure.

Causes of hypertension syndrome:

- ✓ a cerebral or extracerebral mass such as brain tumor, massive infarctions with edema, extensive traumatic contusion, parenchymal, subdural, or extradural hematoma or abscess;
- ✓ obstruction to the flow and absorption of CSF;
- ✓ any process that expands the volume of CSF (meningitis, subarachnoid hemorrhage);
- ✓ an increase in venous pressure due to cerebral venous sinus thrombosis;
- ✓ generalized brain swelling (ischemic-anoxic states).

The clinical manifestations of increased intracranial pressure in children and adults are a diffuse headache (very often at the night and in the morning), vomiting, nausea without diarrhea, dizziness, sleepiness, irritability, impaired consciousness and seizures..

Additional research data. A CSF hypertension can often be recognized by investigation of eye-ground. Papilloedema is usually present. If the pressure ' of the CSF is raised, on X-ray of the skull dilatation of the entrance into Turkish saddle, osteoporosis of its back may be seen. There is an enlargement of the ventricular system on MRl and CT.

Meningeal symptoms

They are a sign of irritation of the meningeal membranes, such as seen in meningitis, subarachnoid hemorrhages. IMeck stiffness, Kernig's signs and three Brudzinski's are termed meningeal signs.

Neck stiffness — inability to flex the neck forward passively due to an increased neck muscle tone and stiffness.

Kernig's sign is assessed when the patient is lying supine, with the hip and knee flexed to 90 degrees. In a patient with a positive Kernig's sign, there is the resistance to passive extension of the knee while the hip is flexed.

A positive *Brudzinski's upper sign* occurs when flexion of the neck causes involuntary flexion of the knee. Positive Brudzinski's middle sign — pressure on the pubic symphysis leads to the reflexive knee flexion. Positive Brudzinski's low sign — investigation of the Kernig's sign leads to involuntary knee flexion in the opposite leg.

Meningeal pose —a hyperextended posture when patient throws back the head and flex the legs.

General hypersthesia and *hyperesthesia of organs of senses* include photophobia (intolerance to a bright light) and phonophobia (intolerance to loud noises).

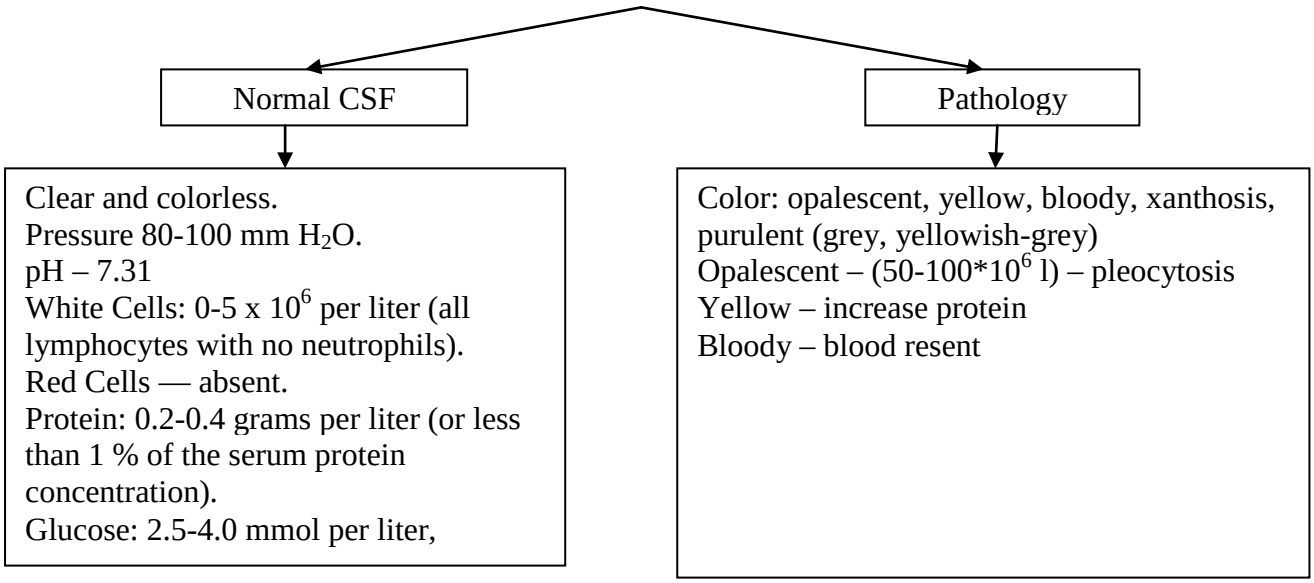
Reactive painful phenomena: painfulness when pressing eyeballs, points of outlet of branches of trigeminal, occipital nerves, cheek-bone Behterev's symptom.

Hydrocephalus

The term hydrocephalus is derived from the Greek words "hydro" meaning water and "cephalus" meaning head. When for any reason the volume of CSF increases within the head, the size of the ventricles also increases and it is known as hydrocephalus. The reasons for CSF excess may include: too much produce, too little reabsorption back into the venous system, blockage in flow.

Hydrocephalus may be congenital or acquired. Hydrocephalus may also be communicating or non-communicating. Communicating hydrocephalus occurs when the flow of CSF is blocked after it exits the ventricles, the CSF can flow between the

ventricles. Non-communicating hydrocephalus — also called "obstructive" hydrocephalus — occurs when the flow of CSF is blocked along one or more of the narrow passages connecting the ventricles.



Liquar syndrome:

- ✓ cell protein dissociation (neutrophil pleocytosis – purulent meningitis, myelitis: lumphocytes – serous meningitis, encephalitic and over)
- ✓ protein cells dissociation (polineuropathy, tumor)
- ✓ hemorrhagic syndrome: presence of blood of CSF
- ✓ CSF hypertension syndrome: increased intracranial pressure, tumor, encephalitis, leptomeningit, trauma
- ✓ CSF hypotension syndrome

Test and typical tasks

1. Indicate, which types of meningitis are characterized by neutrophilis pleocytosis in cerebrospinal fluid?

A. Herpetic

B. Syphilitic

C. Tuberculous

D. Meningococal

E. Enterovirus

2. Patient has enterovirus meningitis. What is identified liquar syndrome?

A. Cells-protein

B. Protein-cells

C. Hemorragic syndrome

D. Syndrome of cerebrospinal hypertensia

E. Syndrome of cerebrospinal hypotensia

3. Patient has tuberculosis meningitis, indicate of kind liquar.

A. Neutrophilis pleocytosis, increase of proten

B. Lymphocytes pleocytosis

C. Limphocytes pleositosis, decrease of glucose and chlorides

D. Increase proteins

4. Indicate, which types of meningitis are characterized by lymphocyte pleocytosis, a decrease of glucose and chlorides in cerebrospinal fluid:

A. Herpetic

B. Syphilitic

C. Tuberculous

D. Enterovirus

E. Parotitic

5. Cerebrospinal fluid returns back directly to the venous system by means of the:

A. Cerebral veins

B. Arachnoid villi

C. Choroid plexus

D. Cerebral aqueduct

E. Apertures in the third ventricle

6. Patient's CSF is turbid, cytosis is 1600 cells per 1 mm^3 (90 % of neutrophils), protein is 0.4 g/l; sugar - 1.2 millimole per litre. What is the name of such a ratio of cells and protein?

7. During examination at clinic, it was noted that the size of an infant's head was larger than normal expected for her age. Ultrasonography examination indicated obstruction of the cerebral aqueduct, enlargement of the lateral and third ventricles. What is the name of such pathology?

8. Results of the examination of CSF In a patient who had meningeal signs are following: 120 cells per mm^3 (mostly lymphocytes) and protein 0.99 g/l. What diagnosis can be suspected? How do we name similar cell-changes in CSF?

9. A patient suffers from pain in the back. Examination of CSF showed: protein 5.2 g/l, cells 5 per mm^3. How can we name changes in CSF? What disease can be suspected? '

10. A boy of 7 years old has acutely fallen ill. His temperature has raised up to 38.2 C he has vomited repeatedly and restlessly. His head is tossed back, the meningeal symptoms are present. No extremity paresis is observed. Describe meningeal symptoms. What does their appearance indicate?

List of answers: 1. 2. 3. 4.C. 5.B. 6. Cells — protein dissociation. 7. Non-communicating (obstructive) hydrocephalus. 8. Serous meningitis. Lymphocyte pleocytosis. 9. Protein — cells dissociation. Tumor of the spinal cord. 10. Neck stiffness, Kernig's and Brudzinski's signs. Irritation of the meningeal membranes.

10. ADDITION METHODS IN NEUROLOGY

Computer-Based Imaging

Computed tomography (CT) is a digital technique that produces cross-sectional anatomical images. Its physical basis is the differential absorption of roentgen rays by tissues of different density. Tissue density is expressed on the Hounsfield scale, named in honor of the inventor of CT. The scale runs from -1000 to +1000 Hounsfield units. The density of water is, by definition, 0 Hounsfield units. When CT images are acquired one section at a time, the roentgen ray tube has to be rotated through 360° for image acquisition, and then back to the starting position, and the patient table must then be moved forward, in preparation for the next image. An improved technique for image acquisition is known as helical (spiral) CT. In this technique, the roentgen ray tube continually, rotates in one direction, while the patient table continually moves forward at a constant speed. As a result, the examination time is reduced, and the images are sharper; normal and pathological anatomy are well demonstrated, and functional and dynamic CT studies can also be performed. The use of intravenous contrast material enhances the visibility on CT of blood, and of tissue in which there is a disruption of the blood-brain barrier. Contrast raises the sensitivity and specificity of CT for a wide range of pathological processes.

CT can be used in trauma, intracerebral hemorrhage, and shift of midline structures. Contrast enhancement is necessary for differentiation between stroke and neoplasms.

Advantages of CT. CT provides better visualization of bone and acute hemorrhage. A CT is generally sufficient in emergency situations. In the case of the clinical suspicion of an acute subarachnoid hemorrhage, the imaging study of choice is CT. For the elderly patients with focal neurological signs or confusion, a CT usually suffices to detect all potentially treatable conditions, such as hydrocephalus and chronic subdural hematoma. CT may be a necessary test in patients who cannot undergo magnetic resonance imaging scanning (e.g., claustrophobia, pacemaker, metallic stent, aneurysmal clips).

Disadvantages of CT: the exposure of the patient to ionizing radiation, fails to show infarcts for up to 24 h, lack of anatomic detail, fails to identify multiple sclerosis plaques,

CT angiography (CTA) — the basis for selective imaging of blood vessels: if one digitally suppresses other structures with high roentgen-ray absorption, such as bone, and then reformats the sectional images of the vasculature in three dimensions, a projectional image results, resembling the images of conventional angiography. CTA is a suitable technique for aneurysm screening, and for the demonstration of stenosis of the carotid artery or other vessels.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is based on the theory of nuclear magnetic resonance, discovered by Bloch and Purcell in 1946. The visible physical entities In MRI are the protons of hydrogen nuclei, which are richly abundant in all tissues containing water, proteins, lipids, and other biological macromol- ecules. Protons rotate around their own axis, and therefore have a small magnetic field. Protons' rotation in the transverse plane of the external magnetic field emits electromagnetic radiation, which can be detected with a radio antenna or coil. This radiation constitutes the magnetic resonance (MR) signal. MRl provides much better tissue contrast than conventional roentgenography or CT. MRl gives excellent anatomic details and shows virtually all structural lesions. The MR image is a planar (tomographic) map of the MR signal as it varies in intensity across different types of tissue.

Tumors and other tissues with .high free-water content appear dark on T1-weighted and bright on T2-weighted or proton density images. Cerebrospinal fluid consists largely of free water, it is thus very dark on T1- weighted images and very bright on T2-weighted and proton density images. T1-weighted images are more suitable than T2-weighted images for the demonstration of necrosis and cystic change within a tumor, or for the demonstration of subacute hemorrhage. Diffusion weighted imaging (DWI) is extremely valuable to identify early stroke signs. Perfusion weighted imaging (PWI) is helpful in demonstrating area at risk in stroke (ischemic penumbra). Gradient

echo is helpful for hemorrhage, both old and new. T2 weighted image shows edema and white-matter lesions well. FLAIR sequences are useful for evaluation of multiple sclerosis. Contrast enhancement with gadolinium will better show neoplasms and multiple sclerosis plaques. MRl is a good noninvasive method for spinal cord lesions or herniated disks in cervical or lumbar regions.

Advantages of MRl. MRl provides better tissue visualization than CT and thus allows the diagnosis of many conditions affecting the brain parenchyma that are not usually visible on CT, including microangiopathy, axqnal injury in head trauma, the plaques of multiple sclerosis, encephalitises, and other conditions.

MRl is *absolutely conlraindicated* in the presence of cardiac pacemakers, neurostimulators, cochlear implants, ferromagnetic aneurysm clips, and other ferromagnetic foreign bodies in tissues that are susceptible to injury.

Magnetic resonance angiography (MRA) is the initial imaging of choice; 2-D time of flight shows extracranial circulation (vertebral and carotid bifurcations); 3-D time of flight demonstrates intracranial circulation; contrast-enhanced studies will show the aortic arch and aortic branches.

Ultrasonography

Ultrasonography — neurologically relevant diagnostic techniques using ultrasound include Transcranial Doppler (TCD) and duplex-ultrasonography of the intra- and extracranial arteries.

This technique is based on the effect first described by Christian Doppler in 1843. Doppler ultrasound, a special application of ultrasound, measures the direction and speed of blood cells when they move through vessels. The movement of blood cells causes a change in pitch of the reflected sound waves (called the Doppler effect). A computer collects and processes the sounds and creates graphs or color pictures that represent the flow of blood through the blood vessels.

In recent years, the capabilities of ultrasound flow imaging have been increased enormously. Color flow imaging is now commonplace and facilities such as 'power' or

'energy' Doppler provide new ways of imaging flow in **duplex-ultrasonography.** Color flow imaging can be used to identify vessels, requiring examination, to identify the presence and direction of flow, to highlight circulation anomalies, throughout the entire color flow image, and to provide beam/vessel angle correction for velocity measurements. Pulsed wave Doppler is used to provide analysis of the flow at specific sites in the vessel by investigation. When using color flow imaging with pulsed wave Doppler, the color flow/B-mode image is frozen while the pulsed wave Doppler is activated. Recently, some manufacturers have produced concurrent color flow imaging and pulsed wave Doppler, sometimes referred to **triplex-ultrasonograpliy.**

When these modes are used simultaneously, the performance of each is decreased. Because transducer elements are employed in three modes (B-mode, color flow and pulsed wave Doppler), the frame rate is decreased, the color flow box is reduced in size and the available pulse repetition frequency is reduced, leading to increased susceptibility to aliasing. Power Doppler is also referred to energy Doppler, amplitude Doppler and Doppler angiography. The magnitude of the color flow output is displayed rather than the Doppler frequency signal.

A Doppler ultrasound may help to diagnose many conditions, including arterial occlusion or stenosis by blood clots, decreased blood circulation, aneurysms and venous insufficiency.

Angiography

Angiography is still considered the "gold standard" for identifying blood vessel stenosis and aneurysms. Cerebral angiography was invented by the Portuguese neurologist and neurosurgeon Egas Moniz, who performed the first carotid angiogram in 1927. Cerebral angiography is mainly used for the diagnosis or exclusion of the following: stenosis and occlusion of intra- and extracranial blood vessels, cerebral venous and venous sinus thrombosis, ruptured and unruptured cerebral aneurysms, specific arterial abnormalities including dissection and fibromuscular dysplasia, or irregular caliber and mycotic aneurysms in inflammatory and infectious conditions, arteriovenous malformations and fistulae, brain tumors (characterization of blood

supply). In addition, angiography is part of the interventional treatment (by the neuroradiologists) of aneurysms, arteriovenous malformations and fistulae, arterial stenosis and vasospasm. It is also a part of the technique of intraarterial thrombolysis, and of mechanical extraction of intravascular thrombi.

Electroencephalography

Electroencephalography (EEG) is a dynamic test of electrical activity of the brain, the most important diagnostic study in epileptology, provides information about the function, rather than the structure of the brain. EEG is performed by placing 20 electrodes on the scalp,(the standard EEG is recorded through mounted electrodes on the scalp according to the 10-20 system), amplifying the activity and displaying it on a monitor or paper. EEG is obtained either as a bipolar recording (in which potential differences are measured between the scalp electrodes) or as a monopolar recording (in which the difference is measured between each scalp electrode and a reference electrode). Brain waves are summations of excitatory and inhibitory potentials that are projected through the reticular nucleus of the thalamus to the cerebral cortex.

The normal EEG changes in the early years of life but becomes standardized during adolescence. Normal rhythm in adults in awake with closed eyes, resting state is *alpha* (8-13 Hz), disappears by opening eyes or in a drowsy state. *Beta* activity (14- 30 Hz) may be seen in the frontal areas. *Theta* rhythm (4-7 Hz) is normal during drowsiness or sleep, abnormal in alert. A small amount of theta activity may be present in the awake. *Delta* rhythm (less than 4 Hz) is normal during drowsiness or sleep, abnormal in alert. Delta activity is always abnormal in awake patient. Slow waves (delta, theta) may emanate from the white matter and represent a disruption of neural pathways. *Spike, sharp waves*— pathological EEG patterns, represent polarization shifts of neuron groups in the cortex. Hyperventilation, photostimulation and sleep deprivation are all provocative methods by which focal disturbances of brain function are more visible.

EEG is useful in the nonspecific diagnostic of diseases with focal structural lesions: encephalitis, tumors, strokes, brain abscesses. These diseases show focal slow activity. Focal temporal lobe discharges occur in herpes simplex encephalitis. EEG helps to

recognize coma, dementia and brain death. Electrocerebral silence is seen in brain death. Generalized slowing (mostly delta and theta activities) is seen in metabolic encephalopathy, postseizure states, dementias, encephalitis or anoxia.

The method should be evaluated in cases of epilepsy or any altered state of consciousness. EEG is usually recorded between seizures (interictal) but sometimes — during seizures (ictal). The diagnosis of epilepsy is clinical and the normal interictal EEG does not exclude a diagnosis of epilepsy. Spike and sharp waves may be focal (partial epilepsy) or generalized (primary generalized epilepsy). Sleep deprivation moves up patients into the superficial stage of sleep, in which the likelihood of a paroxysmal electrical disturbance is the greatest.

Evoked potentials

Evoked potentials (EP) are potentials, recorded from the projection cortex zones following stimulation of appropriate sensory receptors.

Visual EP. Electrodes, which are placed over the occipital lobes, will record visual stimuli (usually alternating checkerboard flash patterns are used in each eye as the stimulus). Latency and amplitude changes may occur in optic neuritis or multiple sclerosis.

Brainstem-auditoiy EP. Electrodes, which are placed over the vertex, will record cerebral potentials following auditory signals (usually a click). It can help to distinguish lesions of the auditory nerve (e.g., acoustic neuroma) from brainstem lesions (e.g., stroke) by the shape and distribution of the evoked potentials.

Somatosensory EP. Electrodes, which are placed over the parietal lobes, will record stimulation of the peripheral nerves. Commonly they are used during spinal surgery to determine if there has been an interruption in nerve or spinal transmission.

Electromyography

Electromyography (EMG) is an electrical recording of muscle activity that helps in the diagnosis of neuromuscular diseases. Muscles are stimulated by signals from lower motor neurons. This stimulation causes electrical activity in the muscle, which in turn

causes contraction. This electrical activity is detected by a needle electrode inserted into the muscle and connected to a recording device. The interpretation of EMG results requires analysis of the onset, duration, amplitude, and other characteristics of the EMG patterns. Muscle tissue normally shows no EM6 activity at rest or when moved passively by the examiner. EMG activity is decreased in long-standing muscle disorders where muscle tissue is replaced by fibrous tissue or fat.

EMG is performed more often to help to diagnose different diseases causing muscle weakness: muscular dystrophy, congenital, mitochondrial, metabolic myopathies, myotonias, peripheral neuropathies, radiculopathies, nerve lesions, amyotrophic lateral sclerosis, polyneuropathies, spinal muscular atrophy, Guillain-Barre syndrome, ataxias, and myasthenia.

Test and typical tasks

1. Name normal rhythm in adults in the awake with closed eyes.

A. Spikes

B. Alpha rhythm

C. Sharp waves

D. Delta rhythm

E. Theta rhythm

2. Electromyography is the additional method for diagnostic of:

A. Epilepsy

B. Brain tumors

C. Myopathies

D. Meningitis

E. Stroke

3. Indicate the pathology where the encephalography complexes "spike - wave" appear.

A. Epilepsy

B. Meningitis

C. Syringobulbia

D. Multiple sclerosis

E. Poliomyelitis

4. What additional method provides information about the function of the brain?

A. Computed tomography

B. Magnetic resonance imaging

C. Electromyography

D. Encephalography

E. Duplex ultrasonography

5. What additional method is used for the detection of stenosis or occlusion of the cerebral vessels?

A. Encephalography

B. Magnetic resonance imaging of the brain

C. Computed tomography of the brain

D. Craniography

E. Duplex ultrasonography

6. A 30-years-old woman is presented with a 1 week history of double vision, weakness in legs, dizziness. Four years ago she had an episode of the right eye's vision lost for a few weeks. On examination she has nystagmus, a pale right optic disc, increased reflexes in her legs, her gait is ataxic. Which disease can be suspected? What additional diagnostic method can prove the diagnosis?

7. A 52-years-old man is presented with a history of progressive difficulty of walking over the last 4 months. He has noticed weakness, stiffness in his right leg. During the last 2 weeks there were three occasions, when his right leg has been jerking repeatedly for about 5 min. On examination - increased tone and reflexes in the right leg with an extensor plantar response. Sensation is normal. Which disease can be suspected? What additional diagnostic method is the most important in this case?

8. A 9-years-old patient has short sudden periods of consciousness loss without seizures lasting only few seconds with a blank stare and an interruption of ongoing activity. These attacks occur many times during the day. The patient doesn't remember attacks. Neurologic and cognitive examination results are normal. His diagnosis is absence form of epilepsy. What additional diagnostic method is the most important in this case? What can be seen in it?

9. A 18-years-old patient complains of weakness in distal parts of the extremities. On examination he has a decrease of the power and reflexes in his legs and arms and sensory disorders in distal parts of the extremities. His father has the same disease. What additional diagnostic method can prove the diagnosis?

10. A 14-years-old patient has been suffering from partial epilepsy for 3 years. What pathological EEG patterns may be seen in this case?

List of answers: 1. b. 2. c. 3. a. 4. d. 5. e. 6. Multiple sclerosis. MRI of the brain. 7. Cerebral tumour. MRI or CT of the brain. 8. EEG. During the seizure generalized three per second spike-and-wave discharges will register. 9. Electromyography. 10. Focal spike or spike-wave discharges.

DYSFUNCTION OF THE PELVIC ORGANS

<table>
<tr><th colspan="5">Urination damage</th></tr>
<tr><th>Components of an act</th><th>Influence on innervated structures</th><th>Phisiological function</th><th>Level</th><th>Clinical symptom</th></tr>
<tr><td>Involuntary reflex</td><td colspan="4"></td></tr>
<tr><td rowspan="3">- the spinal center of paresympathetic innervation of the bladder (S2-S4).</td><td rowspan="3">- relaxation of the external sphincter;
- the cotraction of the detrusor.</td><td rowspan="3">An act of urinaion</td><td>Spinal cone</td><td>True incontinence</td></tr>
<tr><td>Sacral segments (lesion)</td><td>Urinary retention</td></tr>
<tr><td>Sacral segments (irritation)</td><td>Paradoxical ishuria: the bladder is full, urine is excreted by drops.</td></tr>
<tr><td rowspan="2">- spinal center sympathetic innervation of the bladder (cells of Jacobs in lateal horns Th12-L2</td><td rowspan="2">- contraction of internal sphincter;
- relaxation of muscle detrusor</td><td rowspan="2"></td><td>Autonomic preganglion neurons</td><td>Tru incontinence</td></tr>
<tr><td>Lession of cervical and thoracic segments from both sides</td><td>Urinary retentiom</td></tr>
<tr><td>Voluntary
Paracentral</td><td>The motor cortical region of urination and defecation.</td><td>The voluntary control of act of urination (inhibition or strengthening urge)</td><td>Hypothalamus</td><td>True incontinence</td></tr>
<tr><td>Precentral gyrus</td><td>The sensetivity cortical regionof urination.</td><td>Feeling of filling the bladder</td><td>Bilateral violation of communication with cortical centers</td><td>Loss of urge, urine through the urehra, there is no arbitary management of the act.
In the behinnig the retention of urine, excitability the segmental apparatus of spinal cord periodic incontinence.
With complete loss of arbitrary control – the phenomenon of an autonomous urinary bladder (reflex emptying) – intermittent incontinence.</td></tr>
</table>

BASIC TOPICAL NEUROLOGY SYNDROME

With disease of the peripheral nervous system.	Neuritic. Polyneuritic. Plexal type. Redicular.
With disease spinal cord	Segmetal-dissociated (dorsal horns cord and front grey soldering). Anterior horn Lateral horn. Conduction posterior column. Conduction lateral. Conduction column combined. Half transversal of spinal cord (syndrom Brown-Sequard) Total lesion of the diameter of the spinal cord.
Witg diseases of the brai and membranes	1. Brain stem: - bulbar syndrome; - alternating 2. Pseudobulbar. 3. Thalamic. 4. Hypothalamic. 5. Extrapyramidal. 6. Internal capsule. 7. Cortical: - frontal; -parietal; - temporal; - occipital; - combination. 8. Meningeal 9. Hypo- and hypertensive.

A QUESTION IS FOR THE VERBAL QUESTIONING

1. Types of sensory disorders.
2. Tolosa-Hunt syndrome.
3. Thalamic syndrome Dejerine-Russi, clinical characteristics
4. Symptom lesions of the central motor neuron.
5. Levels circuit arcs deep and superficial reflexes of the lower extremities.
6. Syndrome defeat the cerebellopontine angle.
7. Facial nerve, syndromes defeat of different levels.
8. Syndromes lesions of peripheral motor neuron.
9. Syndromes lesions of the internal capsule.
10. Syndrome of Brown-Sequard, his clinical picture.
11. Symptom lesions of the frontal lobe.
12. Bulbar syndrome, clinical characteristics.
13. Syndromes lesions of the upper cervical spinal cord segments.
14. Alternating syndrome midbrain.
15. Symptom lesions of cervical thickening.
16. Pseudobulbar syndrome, clinical characteristics.
17. Syndrome lesions of the temporal lobe.
18. Syndrome defeat cauda equina.
19. Syndrome parietal lobe lesions.
20. Alternating syndromes of the pons (brain stem).
21. Characteristics of hyperkinetic syndrome, hyperkinetic types.
22. Alternating syndromes of medulla oblongata (Schmidt, Jackson Avelisa).
23. Syndrome lesions of the lumbosacral spinal cord.
24. The functions of the cerebellum, symptoms defeat of cerebellum.
25. The syndrome of lesion of the occipital lobe.
26. Characteristics hyperkinetic-hypertensive syndrome (Parkinsonism). Clinical manifestations.
27. Syndromes defeat vestibulo-cochlear nerve.

28. Violation of higher functions of brain (gnosis, praxis, speech).
29. Syndromes of lesions of the oculomotor, abducens and trochlear nerves.
30. Foster-Kennedy syndrome.
31. The syndrome of lesion of the trigeminal nerve.
32. Types of ataxias, their clinical and anatomical characteristics.
33. Hypothalamic syndrome, clinical characteristics.
34. Alternating-Zakharchenko Wallenberg syndrome.
35. Syndrome lesions of the anterior horns.
36. Syndrome Klodt-Bernard-Horner, clinical characteristics.
37. Syndromes of lesions of the optic nerve.
38. Syndrome lesions of the thoracic spine.
39. Syndromes defeat postcentral and precentral gyrus.
40. Syndrome meningeal irritation.

GLOSSARY

Accomodation – the increase in thickness of the lens needed to focus a near external object on the retina

Ageusia – loss of the sense of taste (gustation)

Agnosia – lack of the sensory – perceptional ability to recognize objects: visual, auditory and tactile agnosia

Agrafia – defeat of write

Akinesia – absence of loss of the power of voluntary motion seen in Parkinson's disease

Alexia – visual aphasia, word or text blindness, loss of the ability to grasp the meaning of written or printed words

Alternating hemiparesis – an ipsilateral cranial nerve palsy and a conralateral hemiparesis of extremities

Amnesia – disturbance or loss of memory

Amyatrophy – muscle wasting or atrophy (eg. ALS)

Anesthesia – loss of sensation

Analgesia – insensibility to painful stimuli

Anisocoria – pupils that are unequal in size

Anosmia – loss of the sense of smell (olfactory anesthesia)

Aphonis – loss of the voice

Apraxia – a disorder of voluntary movement

Areflexia – absence of reflex

Astereognosis – tactile amnesia

Ataxia – incoordination

Athetosis – slow, writing, involuntary movements seen in Huntington's disease

Autotopagnosia – the inability to recognize any part of the body, seen with lesion ob the parietal lobe

Babinski's sign – extension of the great toe in response to plantar stimulation pathology reflex (lesion of pyramidal tract)

Ballism – dyskinesia resulting from damage to the subthalamic nucleus

Bell's palsi – facial nerve paralysis

Chorea – irregular, spasmodic, purposeless, involuntary movements of the limbs and facial muscles, seen in Huntington's disease

Choreoathetosis – abnormal movements of the body of combined choreic and athetoid patterns

Diplopia – double vision

Diplegia – paralisis of both sides of the body

Dysartria – disturbance of articulation caused by paralysis (eg.Vagus nerve paralisis)

Dysesthesia – impairment of sensation

Dyskinesia – movement disorders (lesion of extrapyramidal system)

Dysphagia – difficulty in swallowing

Dysphonia – difficulty in speaking, hoarseness

Enophthamus – recession of the eyeball within the otbit

Epilepsy – a chronic disorders characterized by paroxysmal brain dysfunction caused by excessive neuronal discharge (seizure)

Extrapyramidal (motor) system – system including the striatum caudate nucleus and putamen, globus palling, subthalamic nucleus and substantia nigra

Fasciculations – visible twitching of muscle fibers seen in lower (peripheral) neuron disease

Flaccid paralysis – a complete loss of muscle power or tone resulting from a lower motor neuron

Global aphasia – difficulty with comprehension, repetition and speech

Hemiballism – dyskinesia resulting from damage to the subthalamic nucleus; consists of violent flinging movements of the contralateral extremities

Hemiparesis – slight paralysis affecting one side of the body; seen in stroke involving the internal capsule

Hemiplegia – paralysis of one side of the body

Hydrosis – sweating, perspiration, diaphoresis

Horner's syndrome – oculosympathetic paralysis consisting of miosis, hemianhydrosis, mild ptosis and apparent enophthalmos

Hypacusis – hearing impairment

Hypalgesia – decreased sensibility to pain

Hyperacusis – abnormal acuteness of hearing the result of a facial nerve paralysis (e.g. Bell's palsy)

Hypokinesia – diminished or slow movement; seen in Parkinsosn's disease

Intention tremor – a tremor that accurs when a voluntary movement is made; a cerebral tremor

Kerning sign – subject lies on back with thigh flexed to a right angle, then tries to extend leg. The movement is imposible with meningitis

Kinesthesia – the sensory perception of movement the muscular sense; it is mediated by the dorsal column – medial lemniscuses system

Lhermitte sign – flexing the head result in electric like snocks extending down the spine

Macrographis – megalographia; large hand writing seen in cerebellar disease

Micrographia – small hand writing seen in Parkinsonism

Millard-Gubler syndrome – an alternating faciad hemiparesis, an ipsilateral seventh nerve palsy and a contralateral hemiparesis

Myopathy – disease of the muscle

Neuralgia – nerve pain

Nystagmus – to-and-fro oscillations of the eyeballs; it is named after the fast component; ocular dystaxia as seen in cerebellar disease

Papilledema – choked disk; edema of the optic disk; caused by increased intracranial pressure (e.g. tumor, epi-or subdural hematoma)

Paraplegia – paralysis of both lower extremities

Pill-rolling tremor – a tremor at rest seen in Parkinson's disease

Pseudobulbar palsy – pseudobulbar supranuclear palsy; an upper motor neuron syndrome resulting from bilateral lesion that interrupts the corticobullar tracts; symptoms include difficulties with articulation, mastication and deglutition; results from repeated bilateral vascular lesions

Psychosis – a severe mental thought disorder

Ptosis – drooping of the upper eyelid; seen in Horner's syndrome and oculomotor nerve paralysis

Quadrantonopsia – loss vision in one quadrant of the visual field of one or both eyes

Quadriplegia – tetraplegia; paralysis of all four limbs

Retrobulbar neuritis – optic neuritis frequently caused by demyelinative disease multiple sclerosis

Rigidity – increased muscle tone in both extensors and flexors

Romberg sign – subject stands with feet together, when subject closes his eyes he loses his balance; this a sign of dorsal column ataxia

Scanning speech – scanning dysarthia; word are broken up into syllables; typical of a cerebellar disorders

Scotoma – a blind spot in the visual field

Strabismus – lack of parallelism of the visual axes of the eyes; squint, heterotropia

Syringomyelia – cavitations of the cervical spinal cord result in bilateral loss of pain and temperature sensation and wasting of the intrinsic muscles of the hand

Tabes dorsalis – locomotor ataxia, progressive demyelination and sclerosis of the dorsal columns and dorsal roots seen neurosyphils

Tactile agnosia – inability to recognize objects by touch

Tremor – an involuntary, rhythmic, oscillatory movement

Vertigo – a sensation of whirling motion due to disease of the vestibular system

Visual agnosia – inability to recognize objects by sight

Wernicke's aphasia – difficulty in comprehending spoken language, also called receptive, posterior, sensory, or fluent aphasia

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