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

NEUROLOGY IN TABLE
(Special neurology)

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

Zaporozhye
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Authors:

O. A. Kozyolkin – professor of Neurology, the Head of Department of Neurology;

I. V. Vizir – docent of Neurology, Department of Neurology;

M. V. Sikorskay - docent of Neurology, Department of Neurology.

Reviewers:

E. V. Usachova – Head of the Department of childrens infectious diseases, Professor;

O. V. Kraydashenko – Head of the Department of clinical pharmacology, pharmacy, pharmacotherapy and cosmetology, Professor.

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Kozyolkin O. A.

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CONTENTS

THEMA: CEREBRAL VASCULAR DISEASES. SLOWLY PROGRESSING AND TRANSIENT DISTURBANCES OF CEREBRAL BLOOD CIRCULATION. BRAIN STROKE

Cerebrovascular disease (CVD), including stroke, is the third leading cause of death in Ukraine and the leading cause of disability among senior people. Cerebrovascular disease occurs when the blood vessels supplying the brain with oxygenated blood are damaged or their function is compromised. If the blood flow is severely restricted, depriving the brain of adequate oxygen even briefly, a stroke can occur. It has been estimated that every 45 seconds, one person suffers from a stroke, often with debilitating consequences or even death. One of four men and one of five women over the age of 45 would suffer a stroke.

Anatomy of the cerebral vascular system

Four arteries supply the brain almost exclusively: two internal carotids and two vertebral arteries. The contributions of blood flow to the brain of these systems in the adult human brain are approximately three fourths of the total for the carotids, and one fourth for the vertebrals. These vessels originate from branches stemming out of the aortic arch. Internal carotid and vertebrobasilar arterial systems connect at the base of the brain by arterial anastomosis and form Circle of Willis. The arrangement of the brain's arteries into the Circle of Willis creates redundancies or collaterals in the cerebral circulation. If one part of the circle becomes blocked or stenosed or one of the arteries supplying the circle is blocked or narrowed, the blood flow from other blood vessels can often preserve cerebral perfusion well enough to avoid symptoms of ischemia.

Etiology of cerebrovascular disease

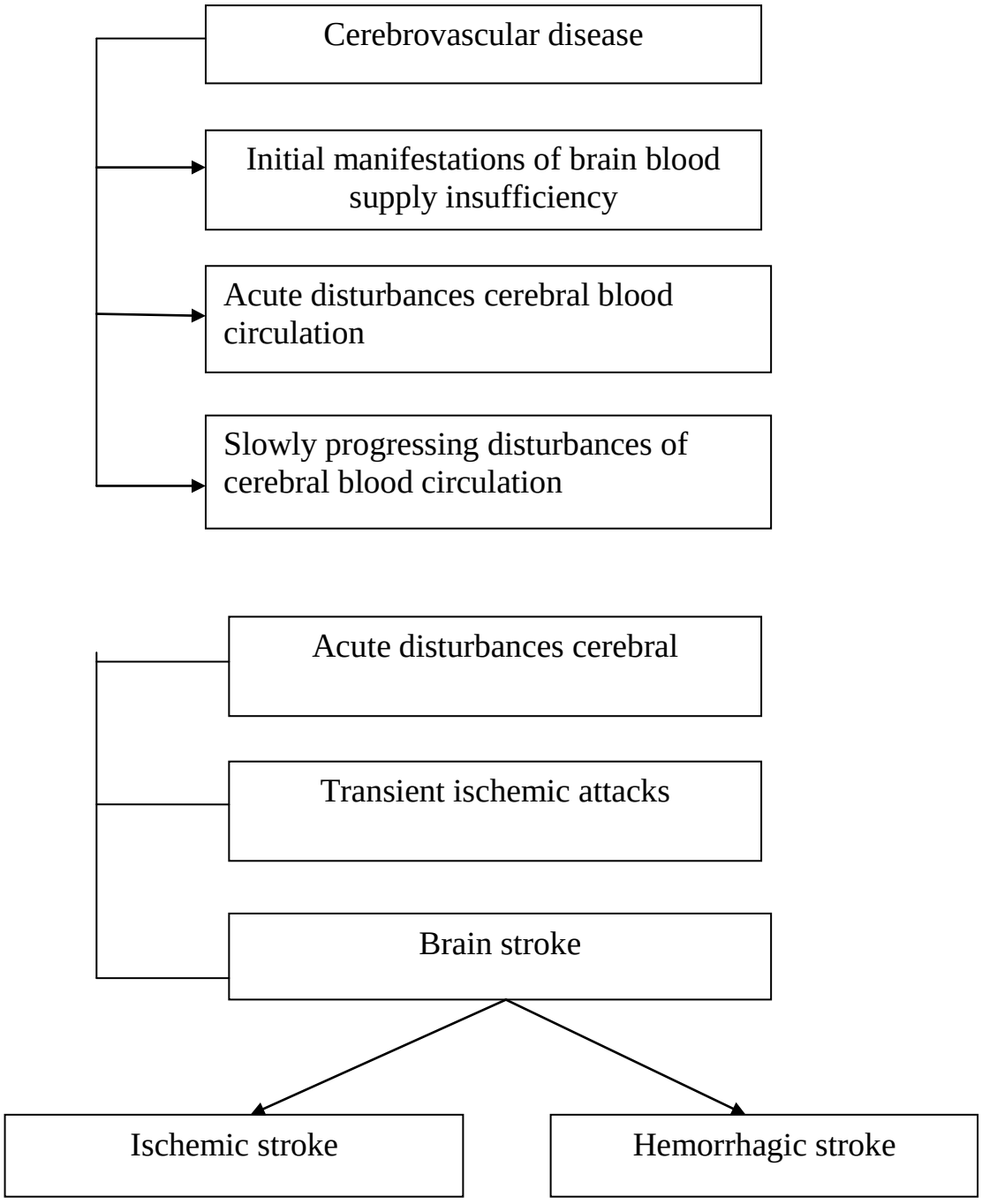
Atherosclerosis, arterial hypertension (> 140 mm Hg systolic, > 90 mm Hg diastolic), a combination of atherosclerosis and arterial hypertension, vasculitis, diabetes mellitus, blood diseases, elevated plasma fibrinogen, degenera tive changes in the upper cervical spinal cord, heart and vascular pathology (atrial fibrillation, valvular heart disease, mitral valve prolapse, myocardial infarction, carotid stenosis), obesity.

Risk Factors for cerebrovascular disease

The risk of stroke increases with *age* and is higher in *men* than in *women* at any age. Risk factors of CVD include: hyperlipoproteinemia (total cholesterol > 5.0 mmol/l, Low-density lipoprotein (LDL) > 3 mmol/l, High-density lipoprotein

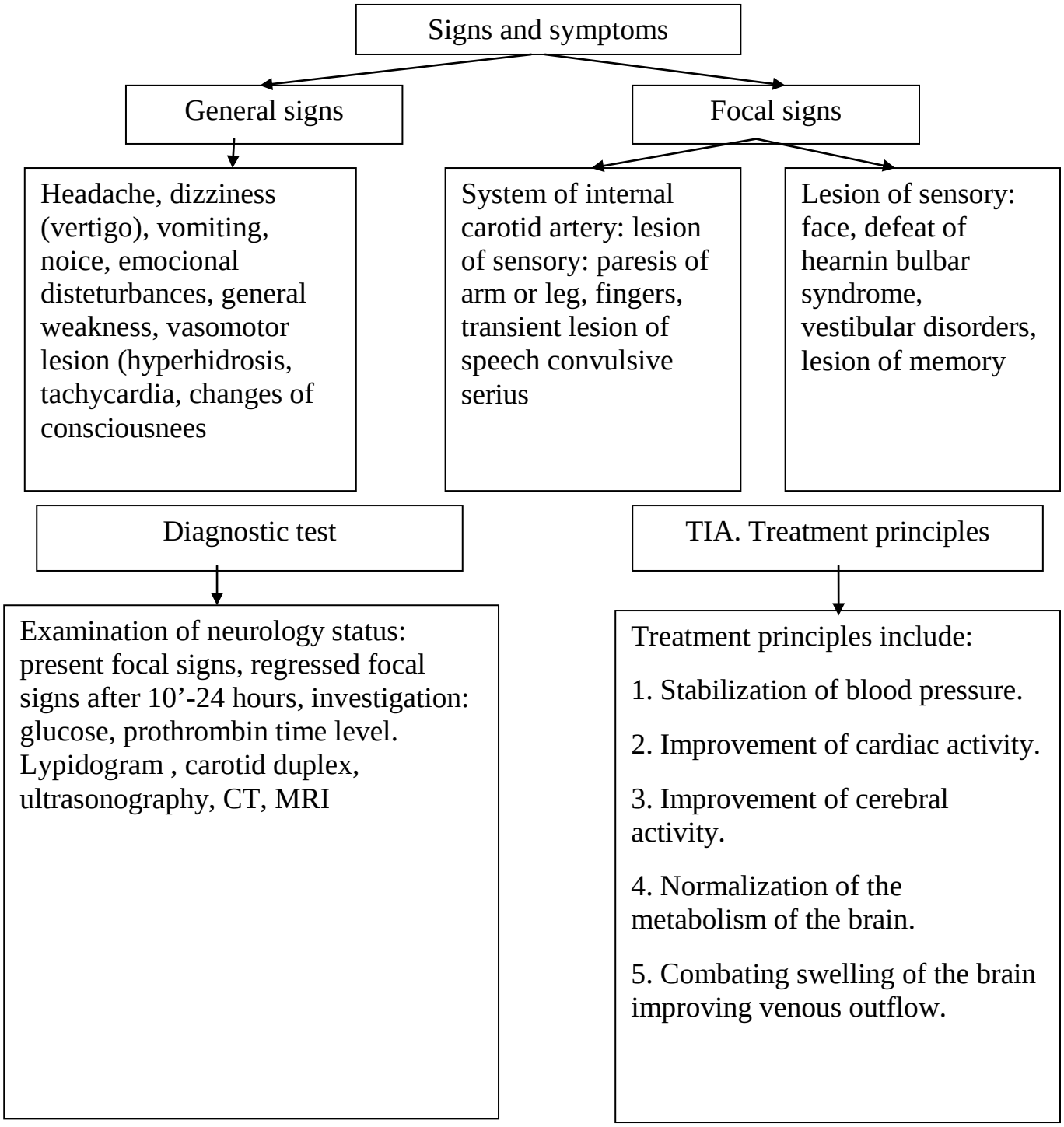
(HDL) < 0.9-1.2 minol/l), cigarette smoking, alcohol abuse (> 60 g of alcohol per day in men, > 40 g in women), drug abuse (amphetamines, heroin, cocaine), sedentary lifestyle.

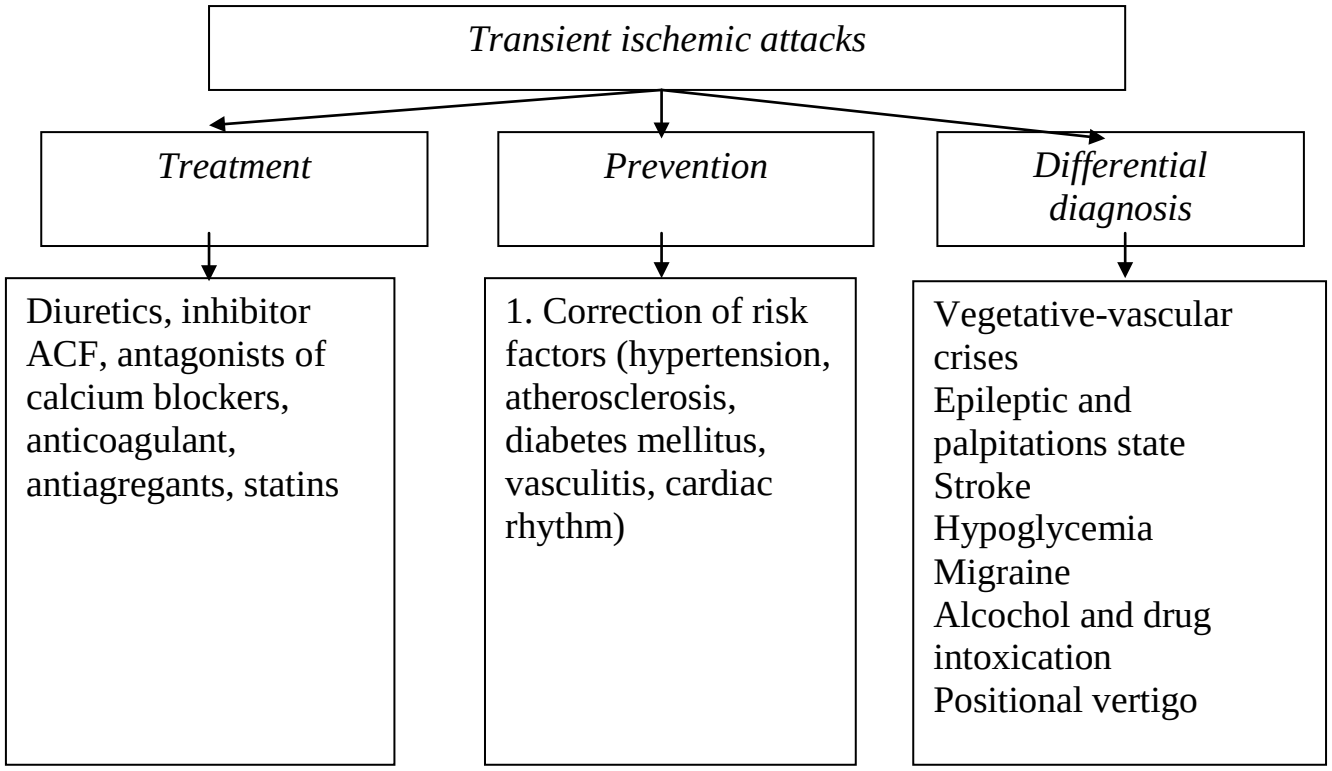
Classification of cerebrovascular disease

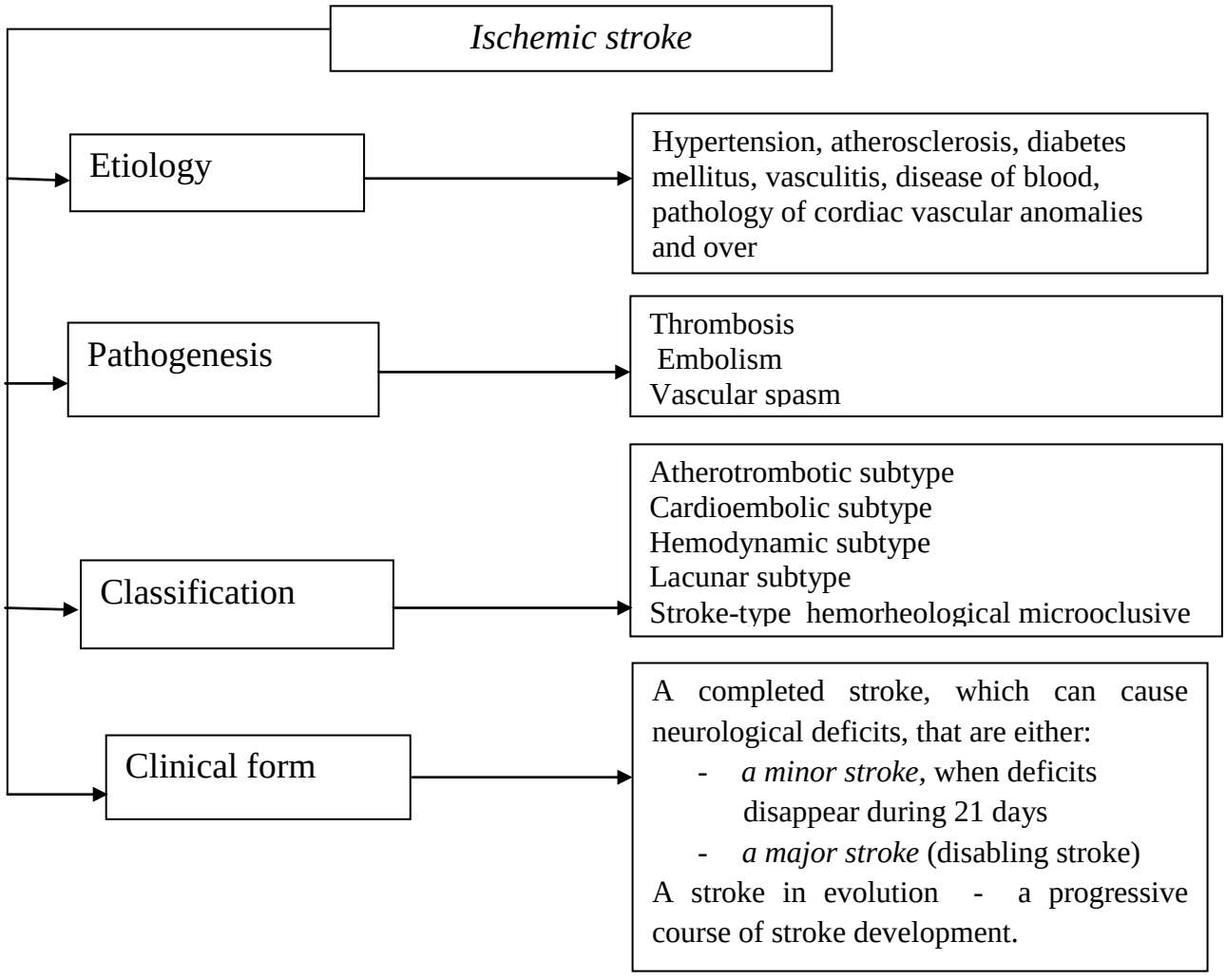


Transient ischemic attacks (TIA)

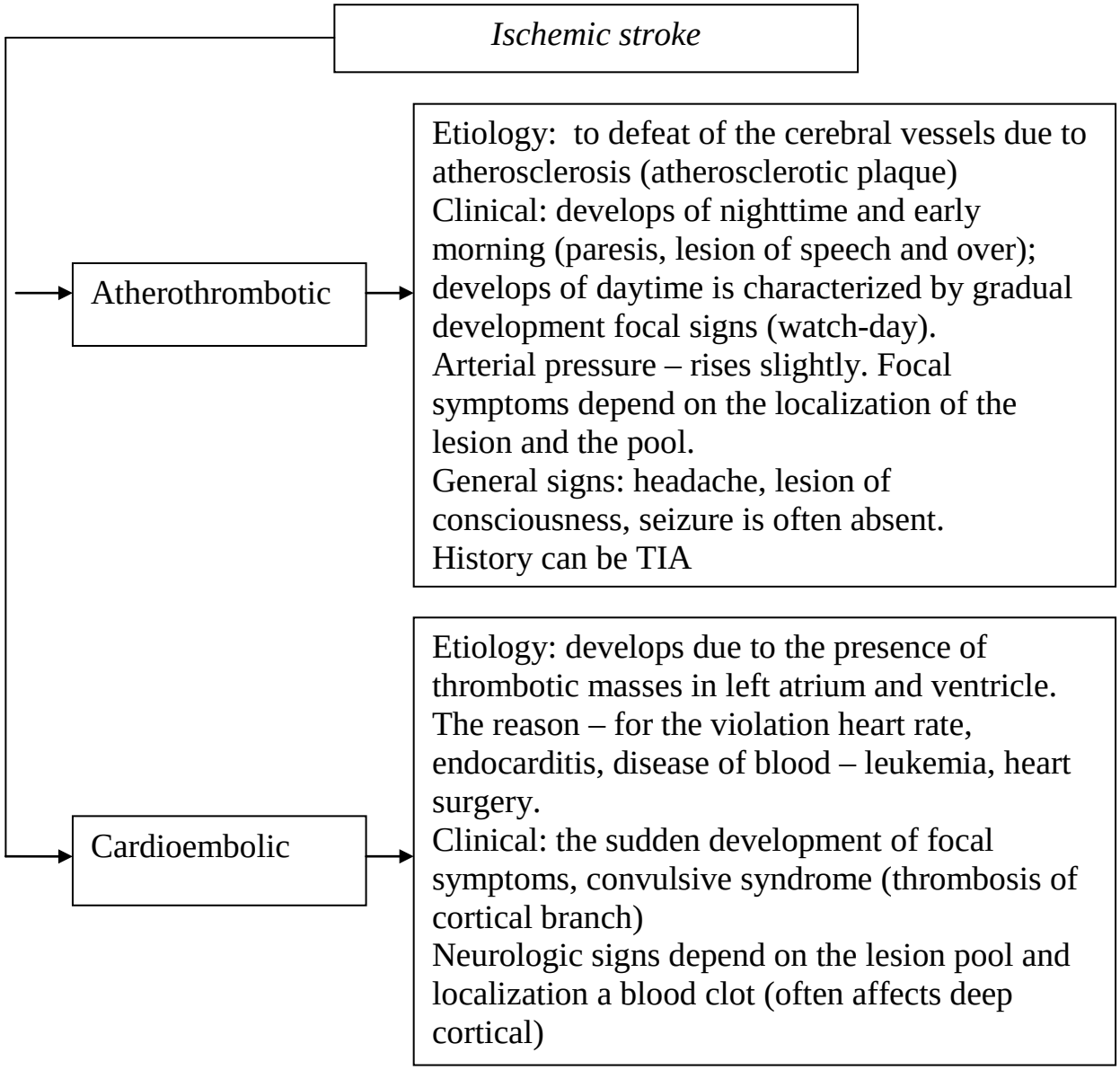
A transient ischemic attack is defined as *acute focal neurological deficit lasting less than 24 hours.* Attacks are usually much shorter, most episodes clearing within 1 hour, only 5 % last longer than 12 hours. Miller Fisher first described the phenomenology of TIAs as "prodromal fleeting attacks of paralysis, numbness, tingling, speechlessness, unilateral blindness or dizziness," which preceded cerebral infarction in patients with the occlusion of the internal carotid artery (ICA).

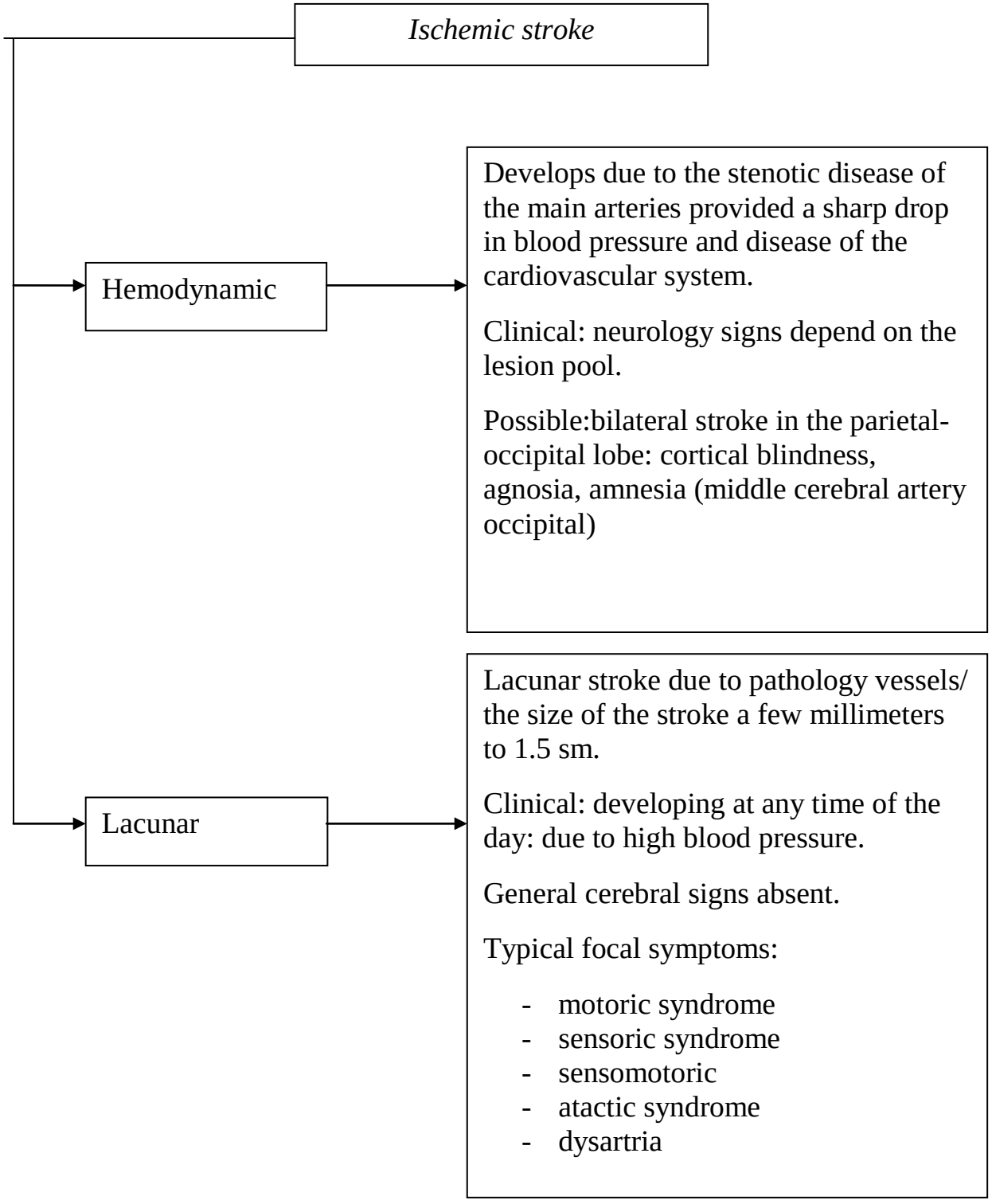


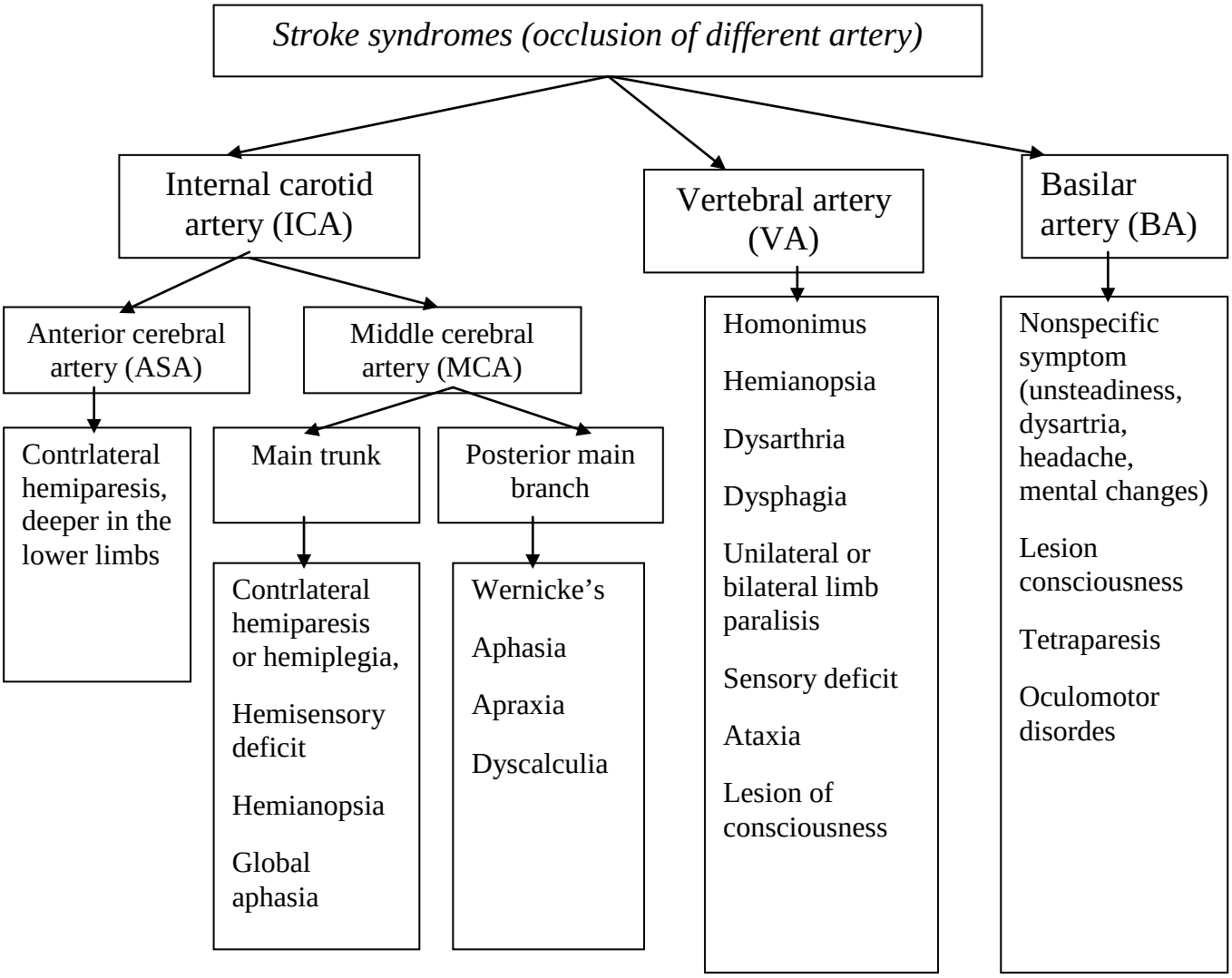

Transient ischemic attacks
Treatment
Prevention
Differential diagnosis
Diuretics, inhibitor ACF, antagonists of calcium blockers, anticoagulant, antiagregants, statins
1. Correction of risk factors (hypertension, atherosclerosis, diabetes mellitus, vasculitis, cardiac rhythm)
Vegetative-vascular crises
Epileptic and palpitations state
Stroke
Hypoglycemia
Migraine
Alcochol and drug intoxication
Positional vertigo


Ischemic stroke
Etiology
Hypertension, atherosclerosis, diabetes mellitus, vasculitis, disease of blood, pathology of cordiac vascular anomalies and over
Pathogenesis
Thrombosis
Embolism
Vascular spasm
Classification
Atherotrombotic subtype
Cardioembolic subtype
Hemodynamic subtype
Lacunar subtype
Stroke-type hemorheological microocclusive
Clinical form
A completed stroke, which can cause neurological deficits, that are either:
- a minor stroke, when deficits disappear during 21 days
- a major stroke (disabling stroke)
A stroke in evolution - a progressive course of stroke development.

Subtype of ischemic stroke




Ischemic stroke
Hemodynamic
Develops due to the stenotic disease of the main arteries provided a sharp drop in blood pressure and disease of the cardiovascular system.
Clinical: neurology signs depend on the lesion pool.
Possible:bilateral stroke in the parietal-occipital lobe: cortical blindness, agnosia, amnesia (middle cerebral artery occipital)
Lacunar
Lacunar stroke due to pathology vessels/ the size of the stroke a few millimeters to 1.5 sm.
Clinical: developing at any time of the day: due to high blood pressure.
General cerebral signs absent.
Typical focal symptoms:
- motoric syndrome
- sensoric syndrome
- sensomotoric
- atactic syndrome
- dysartria



Ischemic stroke diagnostic

1. Brain Imaging: CT or MRI

2. ECG

3. Laboratory test: complete blood count and platelet count, prothrombin time or INR, partial thrombi time (PTT), serum electrolytes, blood glucose, C-reactive protein (CRP) or sedimentation rate, hepatic and renal chemical analysis

4. Extracranial and transcranial Duplex/Doppler ultrasound.

5. MRA or CTA

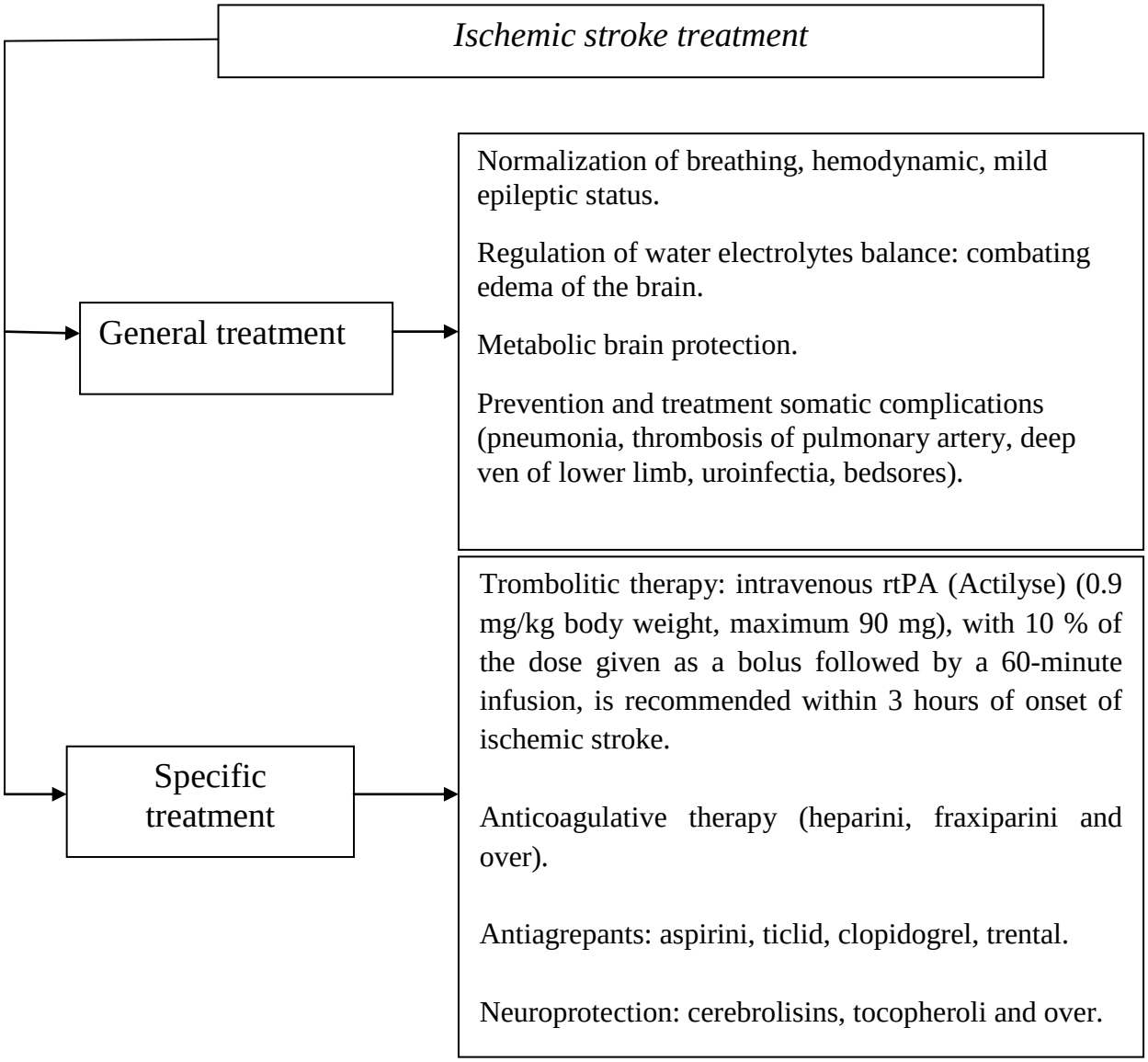
6. Diffusion and perfusion MR or perfusion CT

7. Echocardiography (transthoracic and/or transoesophageal)

8. Chest X-ray

9. Pulse oximetry and arterial blood gas analysis

10. Lumbar puncture
11. EEG
12. Toxicology screen

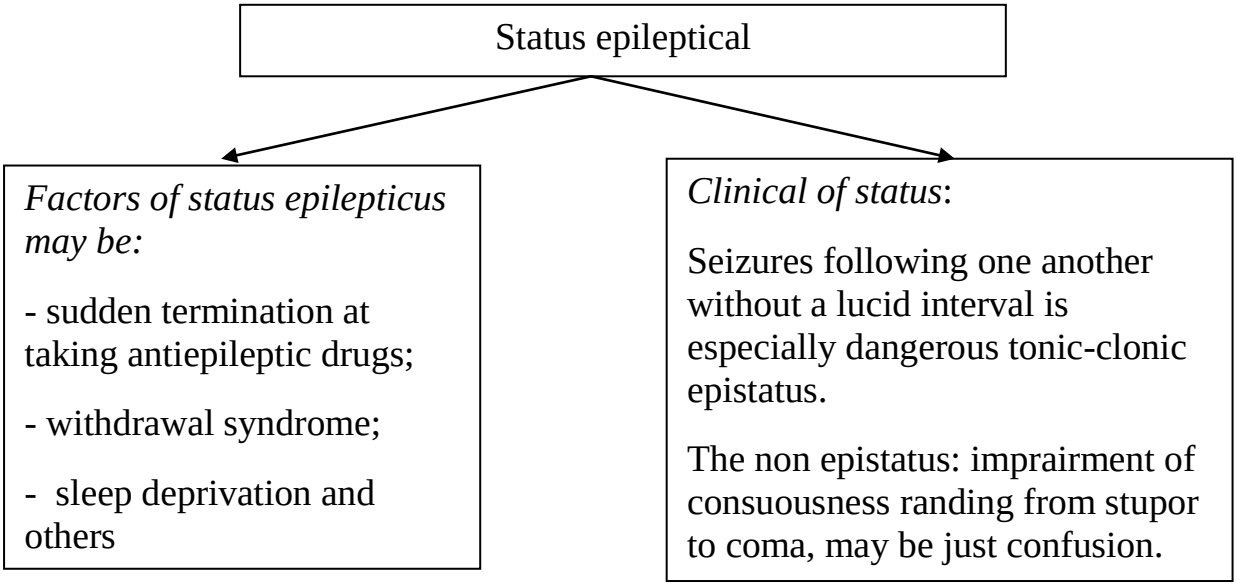


Differential diagnosis

1. Hemorrhagic stroke.
2. Tumor brain.
3. Metabolic encephalopatia.

THEMA: EPILEPSY AND NON-EPILEPTIC PAROXYSMAL STATES

Etiology	1. Idiopathic epilepsy ussually with age-related onset tends to appear during childhood or adolescence/ there is often a family histiry of epilepsy. 2. Symptomatic epilepsy forms result from organic brain damage: traumatic brain injury, stroke, meningitis, multiple sclerosis, alcoholism (one in ten alcogolic suffers from seizures), drug addiction and many other reasons. 3. Crypyogenic form is epilepsy with undetected, hidden etiology.		
Pathogenesis	The basic of occurrence of spontaneous seizures is local or generalized cortical neuronal membrane instability associated with inherited or aacquired secondary features of metabolic processes.		
Classification of epileptic seizures	*Generalized seizures:* - tonic-clonic (grand mal seizures); - absences (typical for children); - myoclonic; - akinetic	*Partial (focal):* Simple partial at: - motor epylepsy; - sensory epylepsy; - visual; auditory; olfactory; guctatory; hallucination; - mental symptoms. Complex partial attacks (with the disturbance of consciousness)/	*Secondary generalization:* - start with partial attacks a go into a seazure generalized
Diagnosis	Clinical feature, EEG, MRI of brain (KT-sczn), TV-EEG-monitoring		
Differential diagnosis	Paroxysmal nono-epileptic states.		
Principle treatment	Anticonvulsant therapy: Differention Contimuty Complexity Individuality		



Treatment

In intensive care unit: diazepam (1-2 times administration).

Control of brain edema: manitol.

Symptomatic therapy: corticosteroids, cardiovascular drugs, heperin in DIC syndrom.

In the absense of the effect of thiopental anesthesia and over.

Paroxysmal non-epileptic states (the absence of the sourse of epileptic activity)

Convulsant	*Non-convulsant*
Febrile and toxic seizures: - hypertethermia (febrile) convulsions are typical for children temperature more than 38^0C; - seizures of infections origin (toxic) associated with toxic-infections effects on the nervous system (meningitis, encephalitic).	Autonomic paroxysms (crises) - autonomic dysfunction sympathy-adrenal; - vagoinsular crisis; - mixed
Spasmophillia (infantile tetany). Occurs as a result of high peripheral neuromuscular excitability.	Syncope. - neurogenic (reflex syncope vasovagal); - cardiac syncope (paroxysmal

	supraventricular tachycardia, acute coronary syndrome and over); -in violation of blood homeostasis and metabolism of the brain; - migrane
Psychogenic nonepileptic seizures earlier known as hystercal paroxysms.	

Differential diagnosis of seizures and psychogenic seizures

Seizures	*Psychogenic seizures*
Start at any age	Does not occur in early childhood
Occurs in any conditions, even at night	In the presence of the observer, doesn't occur at night
During the attack injury, bite of tongue is possible	Traumatic injuries are absent, but the tongue can be bitten
Attack is intermittent	The long-term attack
Stereotyped synchronous movements	A variety of chaotic motion. Often accompanied by weeping, and morning
There is no resistance when doctor is trying to open the eyes of patient	Obvious resistance
Possible involuntary urination	No urination disorders
Often amnesia	No amnesia
Mydriasis with the lack of reaction of pupils to light	The reaction of pupils to light is preserved

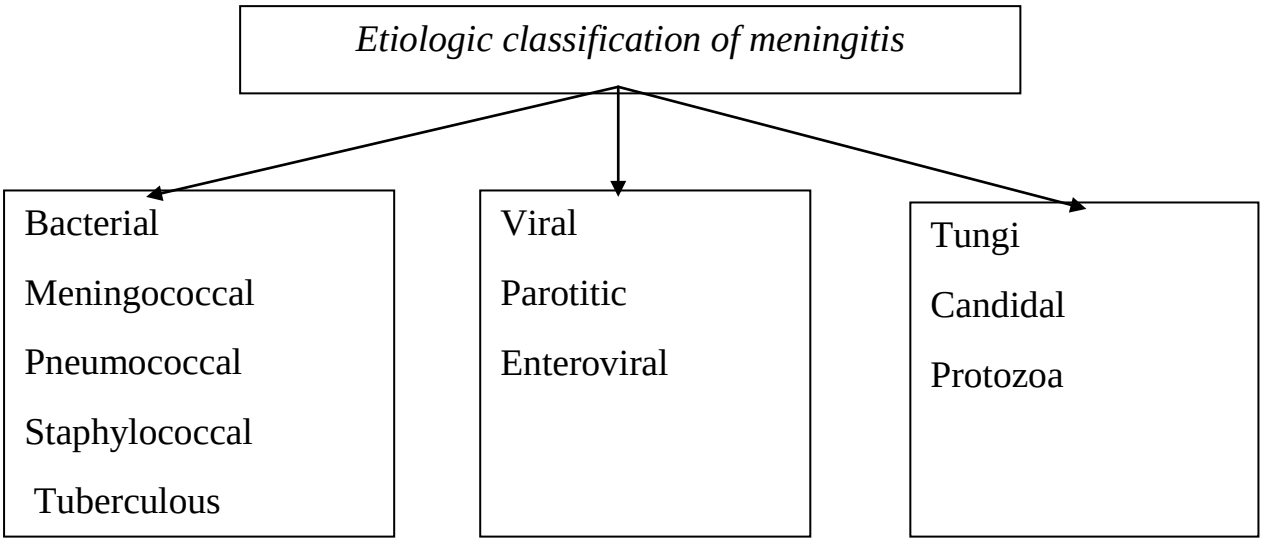
Differential diagnosis of neurogenic syncope and seixures

Neurogenic syncope	*Seizures*
Extremal factors (fear, long vertical position)	There s no extremal factor
Starts gradually	Begins with aura or arises suddenly
Falling slowly, there may be some clonic jerking	Falling is rapid
After syncope the codition worsens	Sleep or good condition after attack
Does not occur in a horizontal position,during sleep	Occurs during sleep
During the attack blood pressure decreases, bradycardia, pallor, sweating	Increased blood pressure, tachycardia, flushing of the skin
Epileptic activity on EEG is not detected	Epileptic activity on EEG is detected

THEMA: INFECTIOUS DISEASES OF THE CENTRAL NERVOUS SYSTEM

Meningitis

Meningitis is an acute infectious disease primarily affecting soft membranes of the brain and spinal cord. Meningitis is usually primarily diagnosed by a general practitioner.



Classification. According to the etiologic classification, there are the following types of meningitis: bacterial (meningococcal, pneumococcal, staphylococcal, tuberculous etc.); viral (parotitic, enteroviral, etc.); caused by tungi (candidal) and protozoal.

It is practically important to divide meningitis into purulent and serous meningitis depending on the nature of inflammation in the membranes and contents of cerebrospinal fluid. In case of purulent meningitis it is neutrophilic pleocytosis that is predominantly found in cerebrospinal fluid, in case of serous — lymphocytic pleotsytosis. This classification is widely used in clinical practice.

Depending on the pathogenesis, meningitis is classified into primary and secondary ones. Primary meningitis develops without previous general infection or infectious lesion of any organ. Meningococcal and enteroviral meningitis belongs to primary meningitis. Secondary meningitis occurs as a complication of general or local infectious disease. In this case, the pathogen crosses the blood-brain barrier and causes meningitis. Tuberculous, staphylococcal, pneumococcal meningitis and other types of meningitis occur in such a way.

According to the clinical classification, as for the course of the disease there are such types of meningitis: fulminant, acute, subacute, chronic, and as for the gravity — very severe, severe, moderate and light.

There are three ways of meninges infecting: contact (perineural and lymphogenous) spread of the pathogen onto the meninges in case of purulent

processes in the areas of paranasal sinuses, the middle ear, osteomyelitis of the skull, direct infection of cerebrospinal fluid due to open brain or spinal injuries, hematogenous spread of the pathogen that causes secondary purulent meningitis.

Clinical presentation of various forms of acute meningitis has much in common. Meningitis can be suspected of basing on the combination of such manifestations:

- syndrome of infectious disease;
- meningeal syndrome;
- syndrome of inflammatory changes in cerebrospinal fluid.

General infectious symptoms of meningitis are various. This can be fever, general fatigue, aching pain in muscles, inflammatory changes in peripheral blood: leukocytosis with a shift of the formula to the left, an increased erythrocyte sedimentation rate (ESR).

In case of purulent meningitis general infectious symptoms are acutely expressed in the first hours and days of the disease. In case of tuberculous meningitis they are expressed not acutely, gradually increasing. In patients with viral meningitis general infectious symptoms most definitely appear in the first days of the disease, but rapidly disappear.

Meningeal syndrome is a complex of symptoms caused by irritation or inflammation process in the meninges. It is observed in all types of meningitis and consists of general cerebral and meningeal symptoms. General cerebral symptoms include: headache, vomiting, psychomotor agitation periodically changed by weakness, impaired consciousness, and seizures. Headaches and vomiting in a combination with fever constitute pathognomonic triad of primary manifestations of meningitis. Observing these symptoms, a doctor of any speciality should suspect meningitis and check the presence of actually meningeal symptoms.

Actually meningeal symptoms are divided into general hyperesthesia and hypersensitivity of the sense organs, reactive pain phenomena and tonic muscle tension. Manifestations of tonic muscle tension include a stiff neck, Kernig's and Brudzinski's signs.

The syndrome of inflammatory changes in cerebrospinal fluid is crucial in diagnosing meningitis. In case of even a slight suspicion of meningitis a lumbar puncture and cerebrospinal fluid analysis have to be done. According to the results of analysis of cerebrospinal fluid, a conclusion about the clinical form of meningitis can be made.

In patients with meningitis spinal fluid flows under high pressure and has various coloring: serous meningitis gives a transparent opalescent colour, purulent— cloudy, yellowish-green one. In case of purulent meningitis pleocytosis is

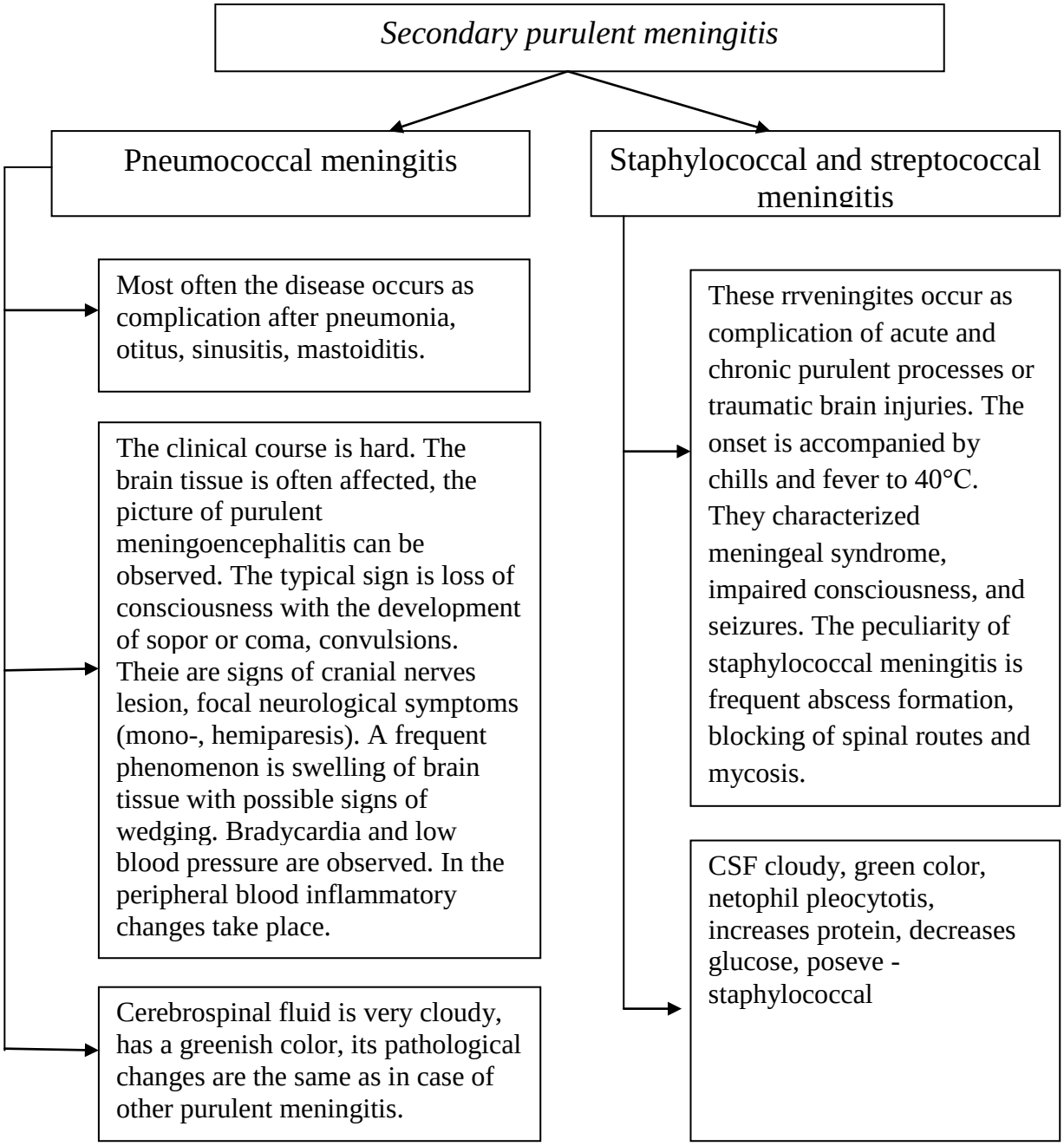
pronounced — thousands or tens of thousands of cells in 1 mcl, mainly neutrophils. In case of serous meningitis pleocytosis is lymphocytic, tens or hundreds of cells in 1 mcl. If a decrease of cytosis accompanied increasing of protein content, this may means encapsulation of the inflammation and the formation of brain abscess.

The analysis of glucose in the contents of cerebrospinal fluid also has a great importance. Its marked decrease is typical for tuberculous meningitis, but is also observed in case of cronic or subacute purulent meningitis and ' meningitis caused by fungi.

The results of cerebrospinal fluid analysis, its cellular composition, protein and glucose level are decisive for the diagnosis and etiotropic therapy prescription. The final etiologic diagnosis is made according to the results of bacteriological, serological and virological analysis of cerebrospinal fluid. The inoculation of pathogen in vital environments is also used to determine their sensitivity to antibiotics. Immunological express methods ensure more rapid diagnostics of meningitis etiology.

Epidemic cerebrospinal meningitis

Clinical presentation	Incubation period – 2-6 days. Disease acutely fever to 38-40^{0}C, a sharp headache with irradiation to the neck and back, vomiting meningeal signs positive, face hemorrahic rash. Neurological examination does not show any focal symptoms (in case of a hard course of the disease the signs of III, IV, VII, VIII pair of cranial nerves lesion are possible).
Diagnostic	Cerebro-spinal fluid flows under the high pressure and has cloudy, gray or yellowish-green coloring. Netrophilic pleocytosis is found (up to tens of thousands of cells in 1 mcl), a high level of protein (up to 1-16 g/l). In the smears of cerebrospinal fluid meningococcus is found in 80 % of cases. It can be seen in blood smears or washout from the posterior pharyngeal wall too.
Differential diagnostics	Other forms of meningitis. Meningism at general infections (that means the absence of CSF changes in case of meningeal syndrome). Subarachnoid hemorrhage (general-infection syndrome is absent, there is no blood in CSF).
Prognosis	The disease lasts from 2 to 6 weeks. Functional changes of CNS: - Asthenic syndrome - mild disorders of mental development - focal symptoms such as paresis of extremities - hydrocephalus, seizures, decreased vision, hearing loss, arachnoiditis and over



Treatment

I. Etiotropic:

1. Antibiot ics: penicillinum (ampicillinum) in dose 300-400 mg per i/v or i/m; cephalosporines, ceftriakson, cefotoxim and over 1 g 4 times per day i/v or i/m; aminoglycozides.

The most effective are combinations of different antibiotics.

2. Sulphanilamide.

3. The treatment of secondary purulent meningitis includes treatment of the source of infection (inflammation process in lungs, middle ear and nose).

II. Pathogenetic treatment includes:

1. Treatment of intoxication (reosorbilact or reopopolyglucin)
2. Correction of cardiovascular and respiratory disturbances.
3. Struggle with cerebral edema (diuretics: mannitol, laziks and dexamethasone).
4. Heparimem doses 5000-20000 U (prevention of disseminated intravascular coagulation).
5. Symptomatic treatment

Serous meningitis

Serous meningitis most often has viral etiology. Its pathogens can be enteroviruses, viruses of lymphocytic choriomeningitis, simple herpes or herpes zoster, Epstein-Barr virus, epidemic parotiditis, tick-borne encephalitis. All of them run with a serous inflammation of the soft cerebral membrane and are accompanied by lymphocytic pleocytosis in cerebrospinal fluid.

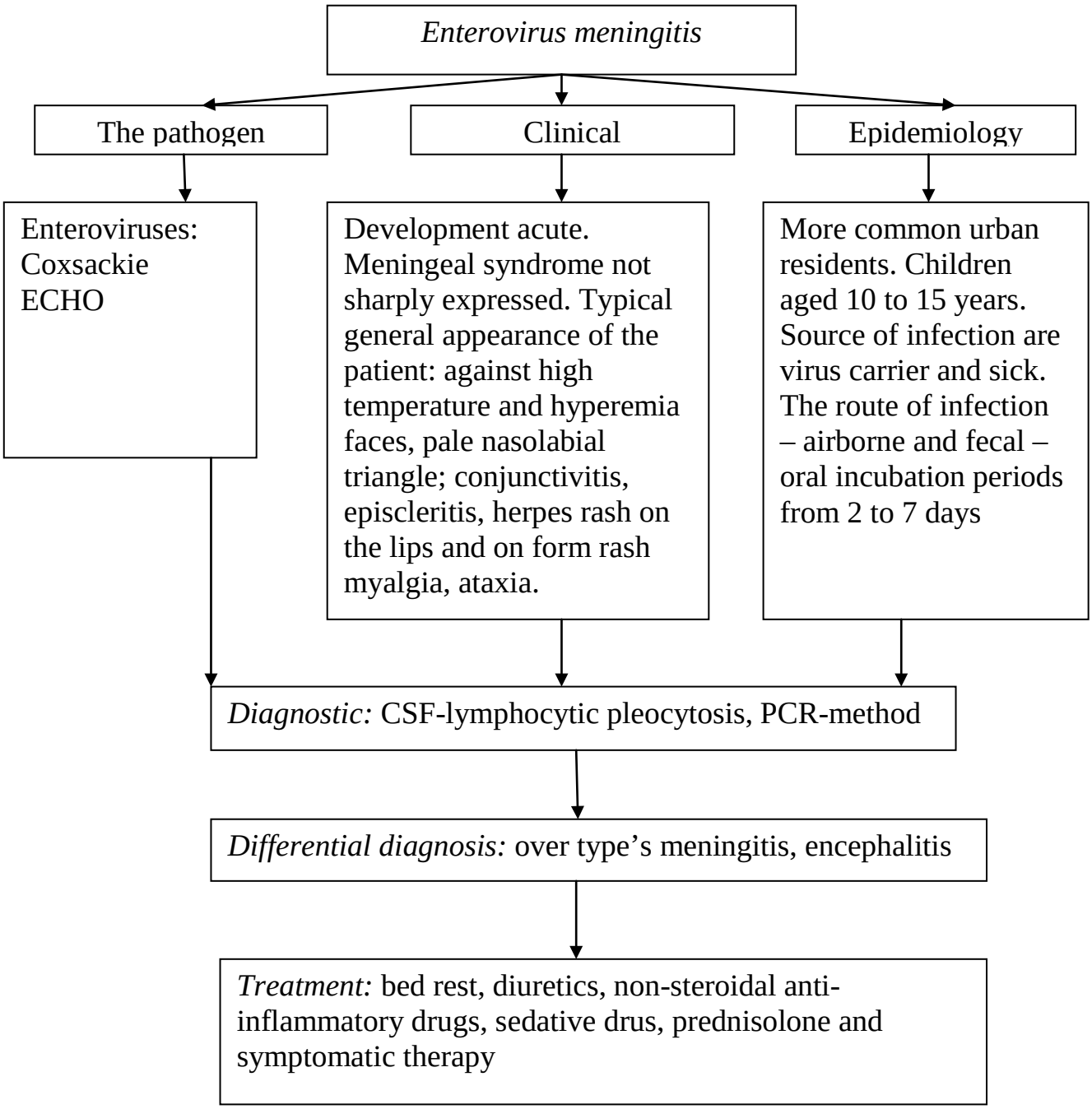
Tuberculosis meningitis

It occurs ill patients with hematogenic disseminated tuberculosis in case of the presence of primary tuberculous nidus in the lungs or lymph nodes. People of all ages may get sick, but the disease mostly affects children aged 2-7 and elderly people as well as patients with immunodeficiency (including AIDS, alcoholism, drug abuse, poor nutrition). The typical sign of tuberculous meningitis is the formation of miliary tubercles in the meninges and sero-fibrinous exudate in the subarachnoid space. The process is almost always localized on the basal surface of the brain, because the cranial nerves are accustomed to pathological procces. The substance of the brain itself also often suffers.

Clinical signs. Symptoms of the disease usually develop gradually. Development of meningeal syndrome is preceded by prodromal period. Its duration can take up to 2-4 weeks. During this period, a patient becomes weak, sleepy, and apathetic, he may have subfebril temperature. He quickly gets tired, loses appetite and weight, has a recurrent headache. The intensity of these symptoms increases with time, vomiting occurs. Gradually, signs of irritation of the meninges appear: a stiff neck and long back muscles, Kernig's, Brudzinski's signs. The body temperature increases up to 38-39 °C. With time the pathological process involves cranial nerves: oculomotor, facial, less frequently — visual and vestibulocochlear ones. Vegetative disturbances are often observed: excessive sweating, changes in pulse rate and blood pressure, hypothalamic disorders. There are also focal neurological symptoms: pathological foot signs, central mono- or hemipa-resis. The patient's condition gradually worsens, deafening proceeds, consciousness impairs, seizures appear. Patients in bed have a characteristic meningeal posture: the head is thrown to the back; lower limbs are bent at the knee joints.

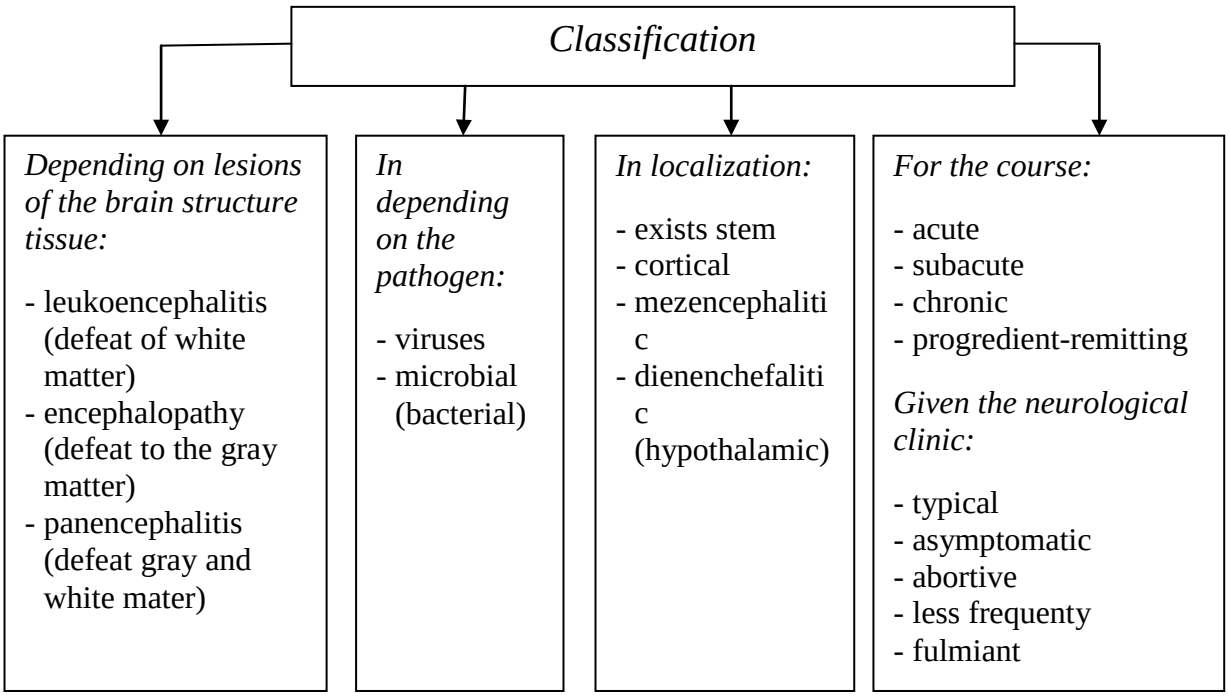
The X-ray investigation of the lungs must be carried out. Spinal fluid is colorless with a pearl shade and (lows under high pressure. Lymphocytic pleocytosis is found (100-500 cells in 1 mm^3). The amount of glucose (up to 1-2 minol/l) and chlorides (up to 90-100 mmol/l) decreases, protein content (up to 5-10 g/l) increases. After some hours a delicate fibrous membrane is formed in a tube with cerebrospinal fluid. A pathogen can be detected there.

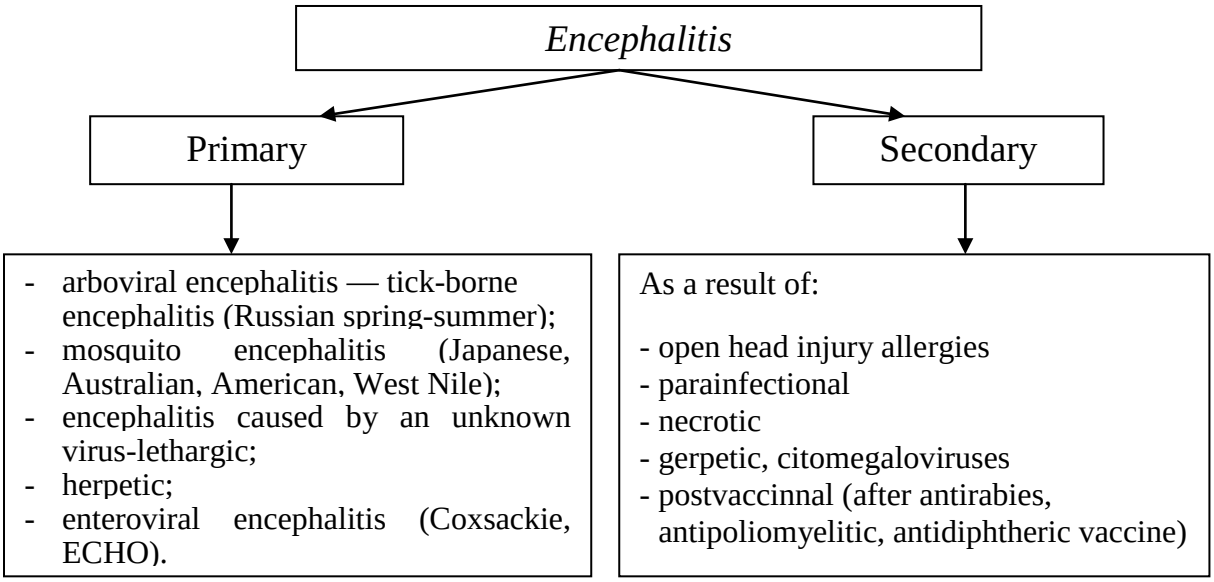
Treatment. The course of tuberculous meningitis is lengthy. Mortality reaches 10 %, mainly among children and the elderly. In the treatment combination of three tuberculostatic drugs is used at least: isoniazid (300-600 mg/day), rifampicin (450-600 mg), pyrazinamide (1.5-3 g/day). They all have side effects, the main one is hepatotoxicity. In case of effective therapy after 2-3 months pyrazinamide is revoked and treatment with isoniazid and rifampicin continues for 10-12 or more months.

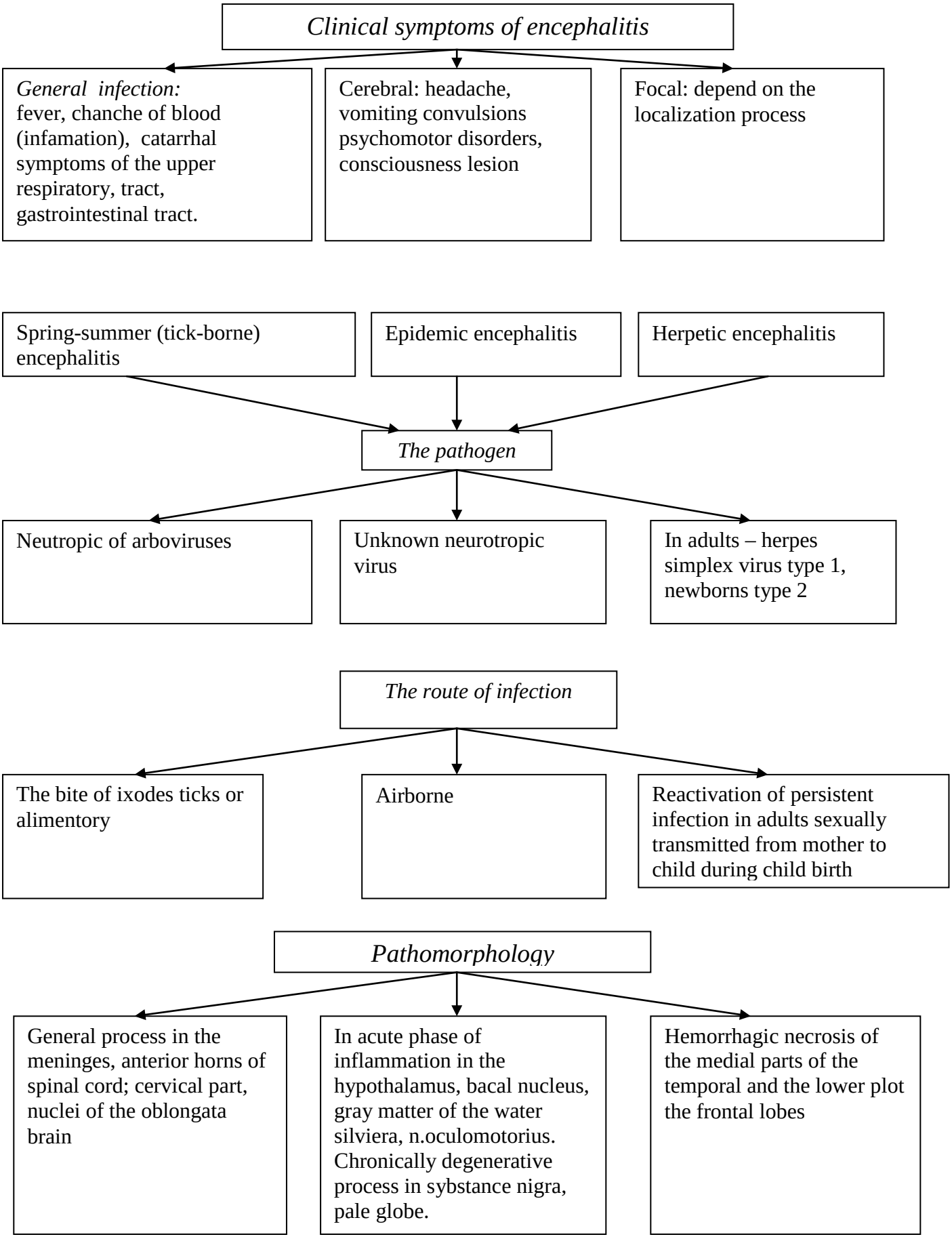

Enterovirus meningitis
The pathogen
Clinical
Epidemiology
Enteroviruses:
Coxsackie
ECHO
Development acute. Meningeal syndrome not sharply expressed. Typical general appearance of the patient: against high temperature and hyperemia faces, pale nasolabial triangle; conjunctivitis, episcleritis, herpes rash on the lips and on form rash myalgia, ataxia.
More common urban residents. Children aged 10 to 15 years. Source of infection are virus carrier and sick. The route of infection – airborne and fecal – oral incubation periods from 2 to 7 days
Diagnostic: CSF-lymphocytic pleocytosis, PCR-method
Differential diagnosis: over type's meningitis, encephalitis
Treatment: bed rest, diuretics, non-steroidal anti-inflammatory drugs, sedative drus, prednisolone and symptomatic therapy

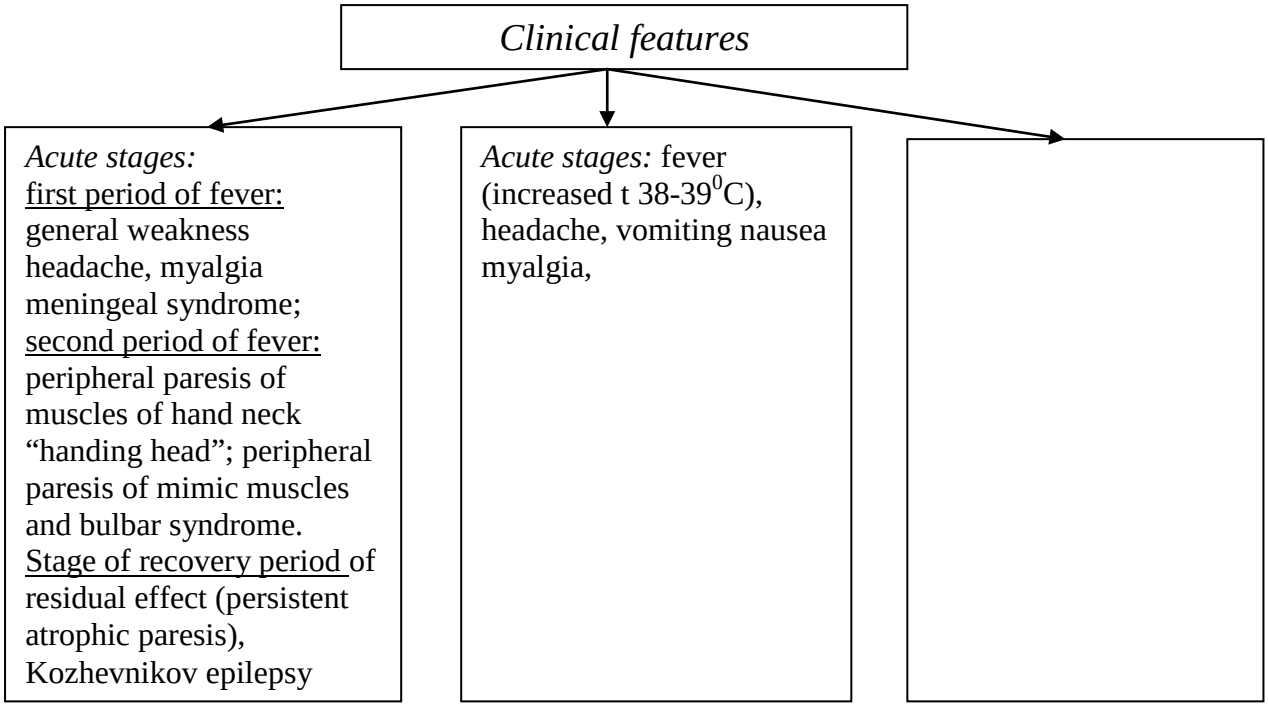
Encephalitis

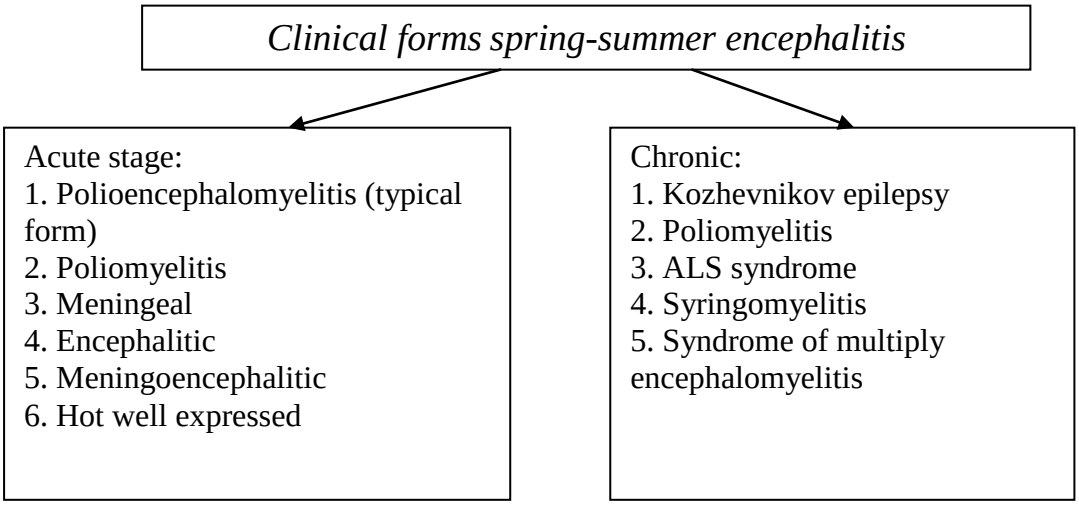
Encephalitis is an inflammatory lesion of the brain tissue of infectious or infectious-allergic origin.

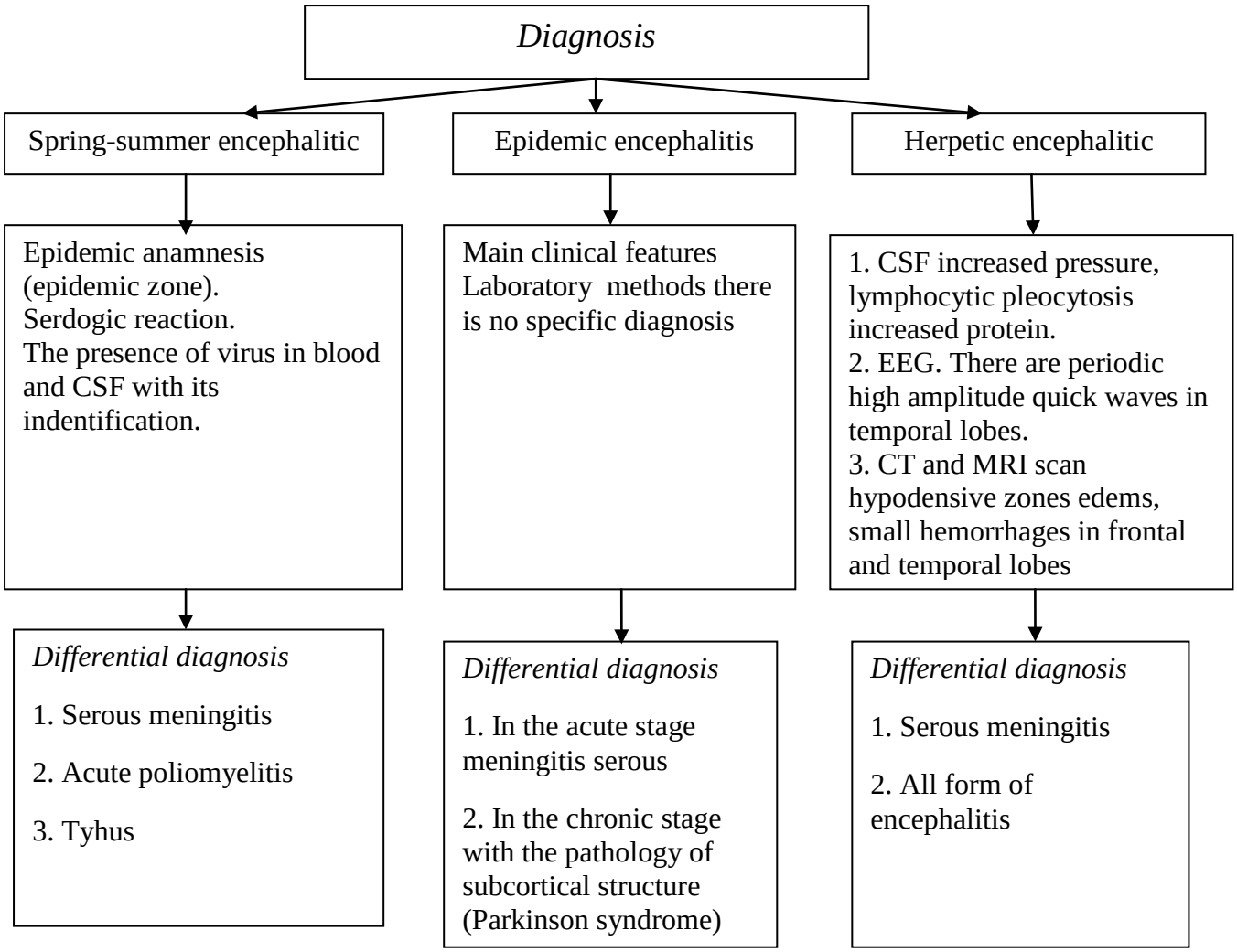


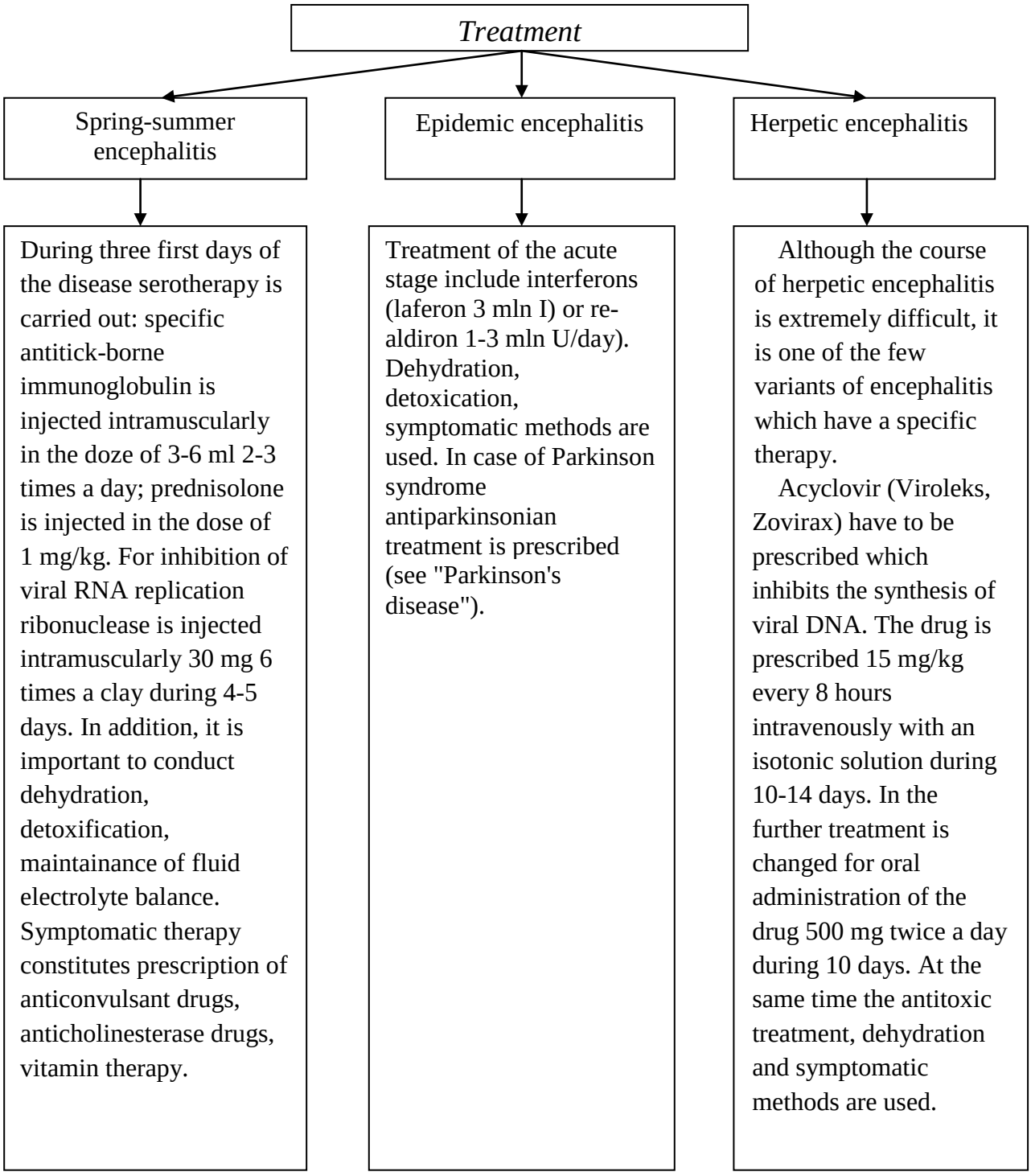



Clinical symptoms of encephalitis
General infection: fever, chanche of blood (infamation), catarrhal symptoms of the upper respiratory, tract, gastrointestinal tract.
Cerebral: headache, vomiting convulsions psychomotor disorders, consciousness lesion
Focal: depend on the localization process
Spring-summer (tick-borne) encephalitis
Epidemic encephalitis
Herpetic encephalitis
The pathogen
Neutropic of arboviruses
Unknown neurotropic virus
In adults – herpes simplex virus type 1, newborns type 2
The route of infection
The bite of ixodes ticks or alimentory
Airborne
Reactivation of persistent infection in adults sexually transmitted from mother to child during child birth
Pathomorphology
General process in the meninges, anterior horns of spinal cord; cervical part, nuclei of the oblongata brain
In acute phase of inflammation in the hypothalamus, bacal nucleus, gray matter of the water silviera, n.oculomotorius. Chronically degenerative process in sybstance nigra, pale globe.
Hemorrhagic necrosis of the medial parts of the temporal and the lower plot the frontal lobes


Clinical features
Acute stages:
first period of fever:
general weakness
headache, myalgia
meningeal syndrome;
second period of fever:
peripheral paresis of
muscles of hand neck
“handing head”; peripheral
paresis of mimic muscles
and bulbar syndrome.
Stage of recovery period of
residual effect (persistent
atrophic paresis),
Kozhevnikov epilepsy
Acute stages: fever
(increased t 38-39^{0}C),
headache, vomiting nausea
myalgia,


Clinical forms spring-summer encephalitis
Acute stage:
1. Polioencephalomyelitis (typical form)
2. Poliomyelitis
3. Meningeal
4. Encephalitic
5. Meningoencephalitic
6. Hot well expressed
Chronic:
1. Kozhevnikov epilepsy
2. Poliomyelitis
3. ALS syndrome
4. Syringomyelitis
5. Syndrome of multiply encephalomyelitis


Diagnosis
Spring-summer encephalitic
Epidemic encephalitis
Herpetic encephalitic
Epidemic anamnesis (epidemic zone).
Serdogic reaction.
The presence of virus in blood and CSF with its indentification.
Main clinical features
Laboratory methods there is no specific diagnosis
1. CSF increased pressure, lymphocytic pleocytosis increased protein.
2. EEG. There are periodic high amplitude quick waves in temporal lobes.
3. CT and MRI scan hypodensive zones edems, small hemorrhages in frontal and temporal lobes
Differential diagnosis
1. Serous meningitis
2. Acute poliomyelitis
3. Tyhus
Differential diagnosis
1. In the acute stage meningitis serous
2. In the chronic stage with the pathology of subcortical structure (Parkinson syndrome)
Differential diagnosis
1. Serous meningitis
2. All form of encephalitis

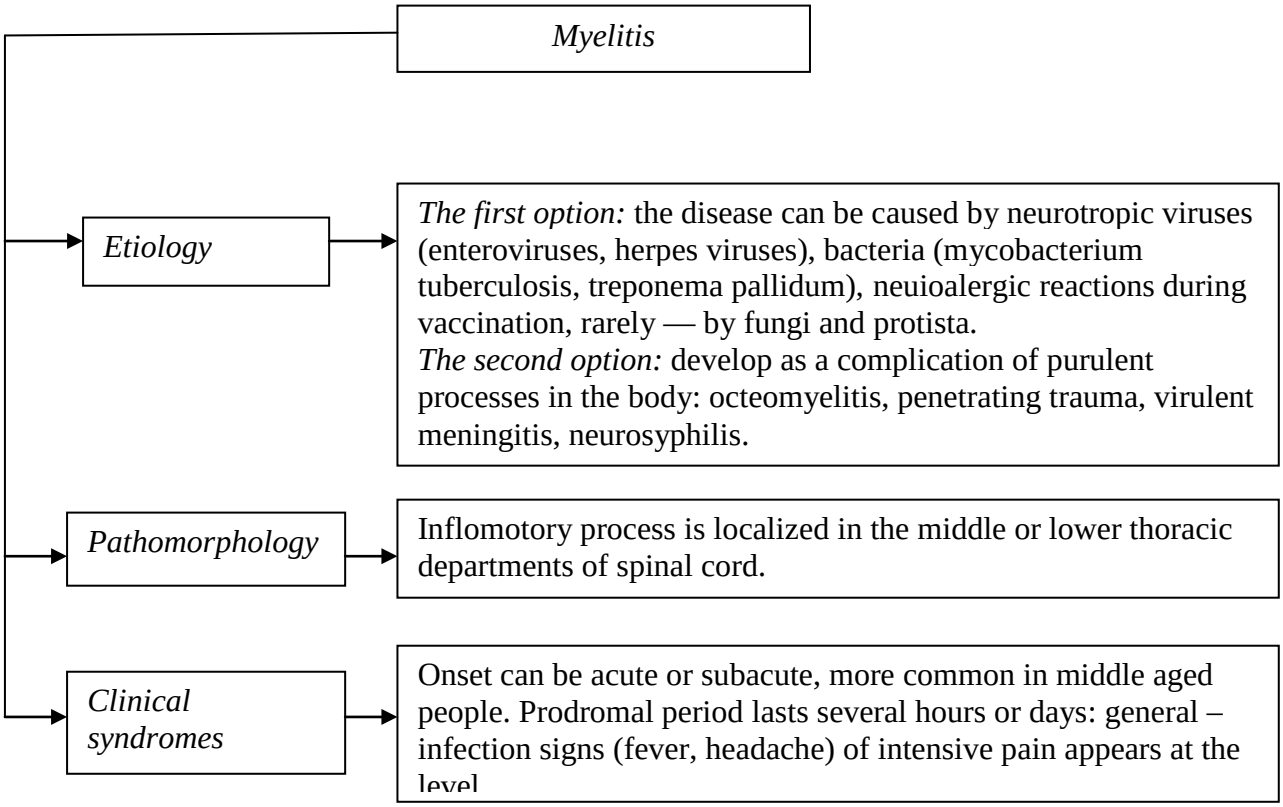

Treatment
Spring-summer encephalitis
Epidemic encephalitis
Herpetic encephalitis
During three first days of the disease serotherapy is carried out: specific antitick-borne immunoglobulin is injected intramuscularly in the doze of 3-6 ml 2-3 times a day; prednisolone is injected in the dose of 1 mg/kg. For inhibition of viral RNA replication ribonuclease is injected intramuscularly 30 mg 6 times a clay during 4-5 days. In addition, it is important to conduct dehydration, detoxification, maintainance of fluid electrolyte balance. Symptomatic therapy constitutes prescription of anticonvulsant drugs, anticholinesterase drugs, vitamin therapy.
Treatment of the acute stage include interferons (laferon 3 mln I) or re-aldiron 1-3 mln U/day). Dehydration, detoxication, symptomatic methods are used. In case of Parkinson syndrome antiparkinsonian treatment is prescribed (see "Parkinson's disease").
Although the course of herpetic encephalitis is extremely difficult, it is one of the few variants of encephalitis which have a specific therapy.
Acyclovir (Viroleks, Zovirax) have to be prescribed which inhibits the synthesis of viral DNA. The drug is prescribed 15 mg/kg every 8 hours intravenously with an isotonic solution during 10-14 days. In the further treatment is changed for oral administration of the drug 500 mg twice a day during 10 days. At the same time the antitoxic treatment, dehydration and symptomatic methods are used.

Secondary encephalitis

Form	Flu encephalitis	Measles encephalitis
Etiology	Viruses A_1, A_2, A_3, B	Severe complications of measles
Pathomorphology	Trombovascular diapeditic and focal hemorrhage perivascular infiltrates, focal lesions of the brain	Fibrous swelling of the walls of the vessels of the brain. Formation prevenslik foci of demyelination, predominantly the white matter of the brain, spinal cord
Main clinical forms	Hemorrhagic flu-like encephalitis. Influenzaencephalopathy syndrome with asthenic, vasculas autonomic syndromes intracranial hypertension	Encephamyelitis measles encephalopathy
Diagnosis	Clinical symptoms of serological and virological studies, in CSF – lymphocytic pleocytosis increased pressure, the blood, protein content increased	
Differential diagnosis	With serious meningitis and encephalitis of other etiology stroke (hemorrhagic form)	All form of encephalitis
Principles of treatment	Gammo-globulin,corticosteroids, diureticks implantation: etamzilat and over	Treatment of measles neuroprotection, L-Dopa immunosuppressants. When hyperkinesis – haloperidol, phenibut and over

Acute myelitis

Myelitis — inflammation of the spinal cord, usually exciting the white and gray matter. Inflammation, limited in several segments, referred to as cross- myelitis. In diffuse myelitis inflammation is localized at several levels of the spinal cord. The disease can be caused by neurotropic viruses (enteroviruses, herpes viruses), Bacteria (mycobacterium tuberculosis, treponema pallidum), neuioalergic reactions during vaccination, rarely — by fungi and protista. Clinical syndrome of acute transverse myelitis can be the first manifestation of multiple sclerosis. Subacute necrotizing myelitis usually occurs as paraneoplastic syndrome. Almost half the cases can not determine the cause of the disease. Frequently myelitis inflammations are localized in the lower part of spinal cord's thoracic section.



Poliomyelitis

Etiology	Three strain are virus 1, 2, 3 types
Ways of infection	Air-borne, alimentary ways
Forms	Asymptomatic, abortive, paralitic

Clinic of paralytic poliomyelitis

STAGES

Incubation period (10-14 days)	*Prodromal period* (1-3 days)	*Preperalitic period* (1-3 days)	*Paralytic period* (7-10 days)	*Restorative period* (2 year)	*Rezidual period*
Incubation period	Fever, information ostium, gastro-instinal disorders	High temperature, sleepiness', meningo-radicular symptoms, cramp pain	Perephiral paresis and paraplegia, spinal bulbar ponds, encephalitlc	Compensa-tion motion in paresis muscular	Persistent after effect paresis atrophy, deformations of extremities skeleton

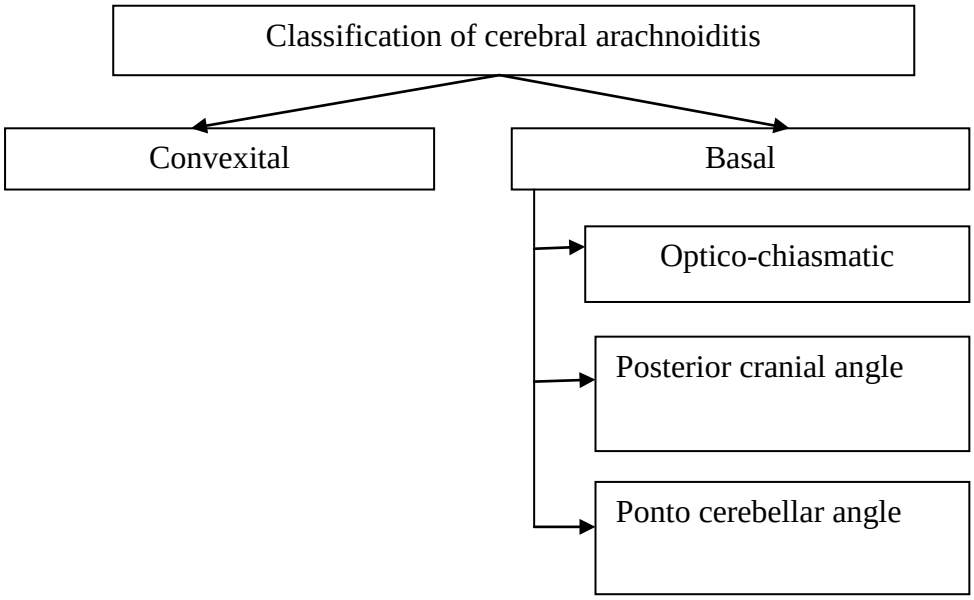
Diagnostic	Presence of virus in nasopharyngeal smear and fecal:, defection antibody in serum blood
Prevention	Isolation of patient on 6 weeks. Child (has contact of silks) – gamaglobullini, vaccination of plans
Treatment	Symptomatic, antibiotics of prevention on pneumonia, restorative period - physiotherapy, residual period – operative treatment.

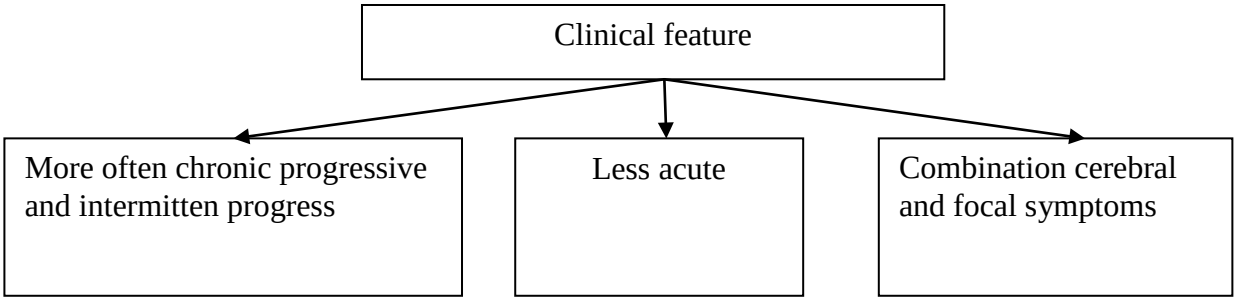
Arachnoiditis

The arachnoiditis a chronic serous inflammatory disease of sarachnoid and partly soft shell-tunic progressive hyperplasia.

Etiology and pathogenesis: flu, sinusitis, otitis, tonsilitis, general infections (mostly child), carried before meningitis, craniocerebral trauma and others.

Pathomorphology: thickening of the meninges, adhesions, cysts with liquid content.





- Constant headache diffuse or local (forehead, back of the head). The intensity of the pain increases in the morning.
- characteristic symptom of jump: get a headache when jumping.
- Nausea
- Vomiting
- Dizziness
- Apathy or irritability, tearfulness
- General weakness
- Rapid fatigability
- Sleep disturbance
- Can be epileptic attacks of different species

Focal symptoms depend on localization of process

Convexital arachnoiditis – is symptoms of irrstation, singht sign of focal symptoms.

- focal epileptic attacks (often)
- generalized epileptic seizurs (rarely)
- asymmetry of superficial and deep reflexes
- can reduced abdominal and plantar reflexes
- the presence of pathological reflex
- light paresis of the limbs

Basal araachnoiditis charactirized by combination of cerebral and focal symptoms (focal symptoms – signe of cranial nerve.

Optico-chiasmatic:

- headache in the area of forehead eye sockets, bridge of the nose
- the decrease in visual acuity
- loss of visual fields
- concentric narrowing of visual fieds
- congestion of the optic nerve
- anosmia (changes of sense of smell)
- vegetative disorders
- hypothalamic disorders

Arachnoiditis of pontocerebellum angle:

- headache in a cervical area
- shooting pain is in the face
- tinnitus, hearing loss
- dizziness of system character
- sometimes vomiting, ataxia

At a neurological inspection discover:

- signs of defeat of cranial nerves:
  -V c.n. trigeminal neuralgia

  -VI c.n. squit

  -VII c.n. is peripheral paresis of mimic muscles

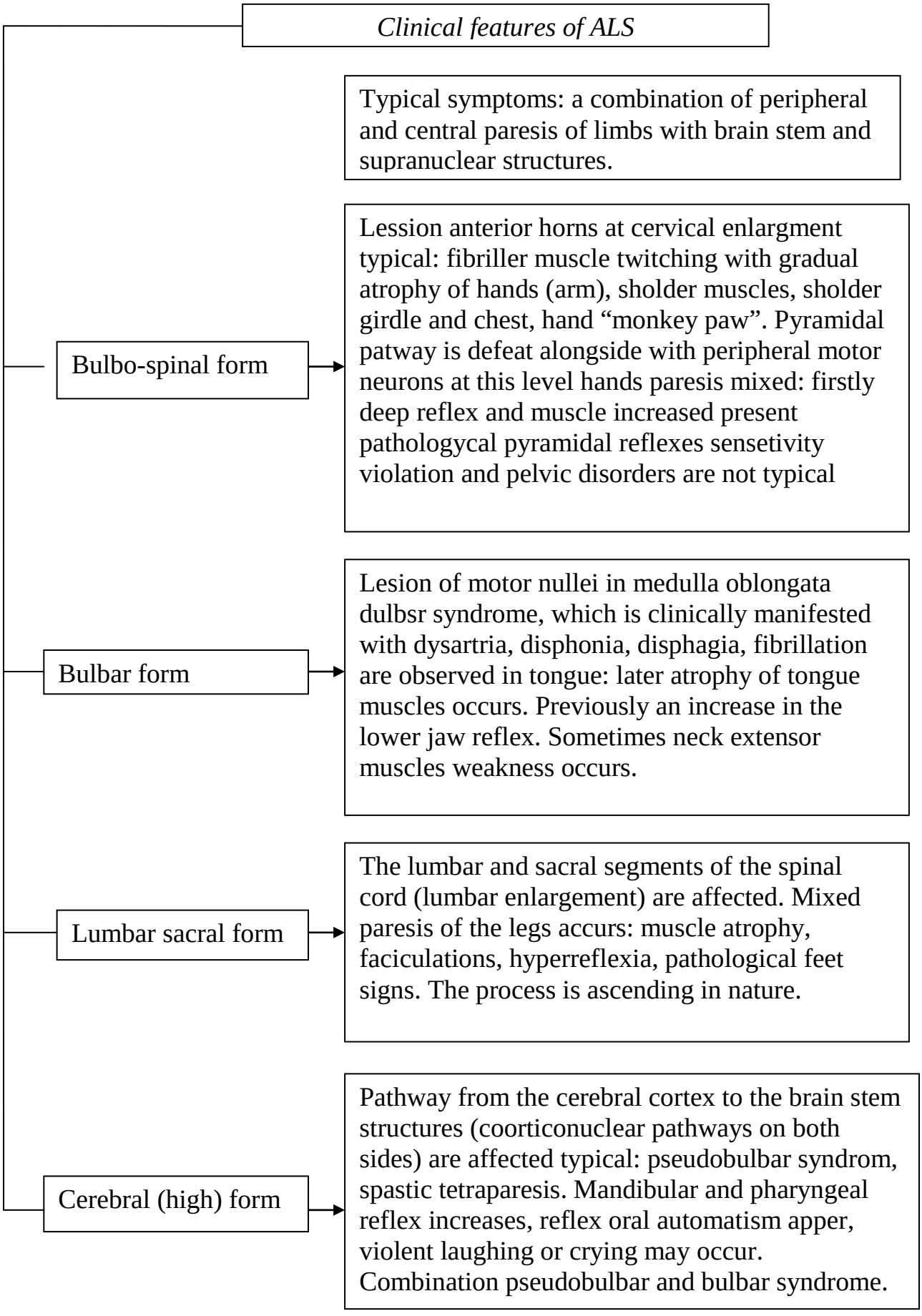
  -VIII c.n. is a decline of ear

- to the cerebellum disorders
  - ataxia
  - bends or falls toward side of defeat
  - nystagmus
- light pyramidal violations
  - on opposite side focal sings

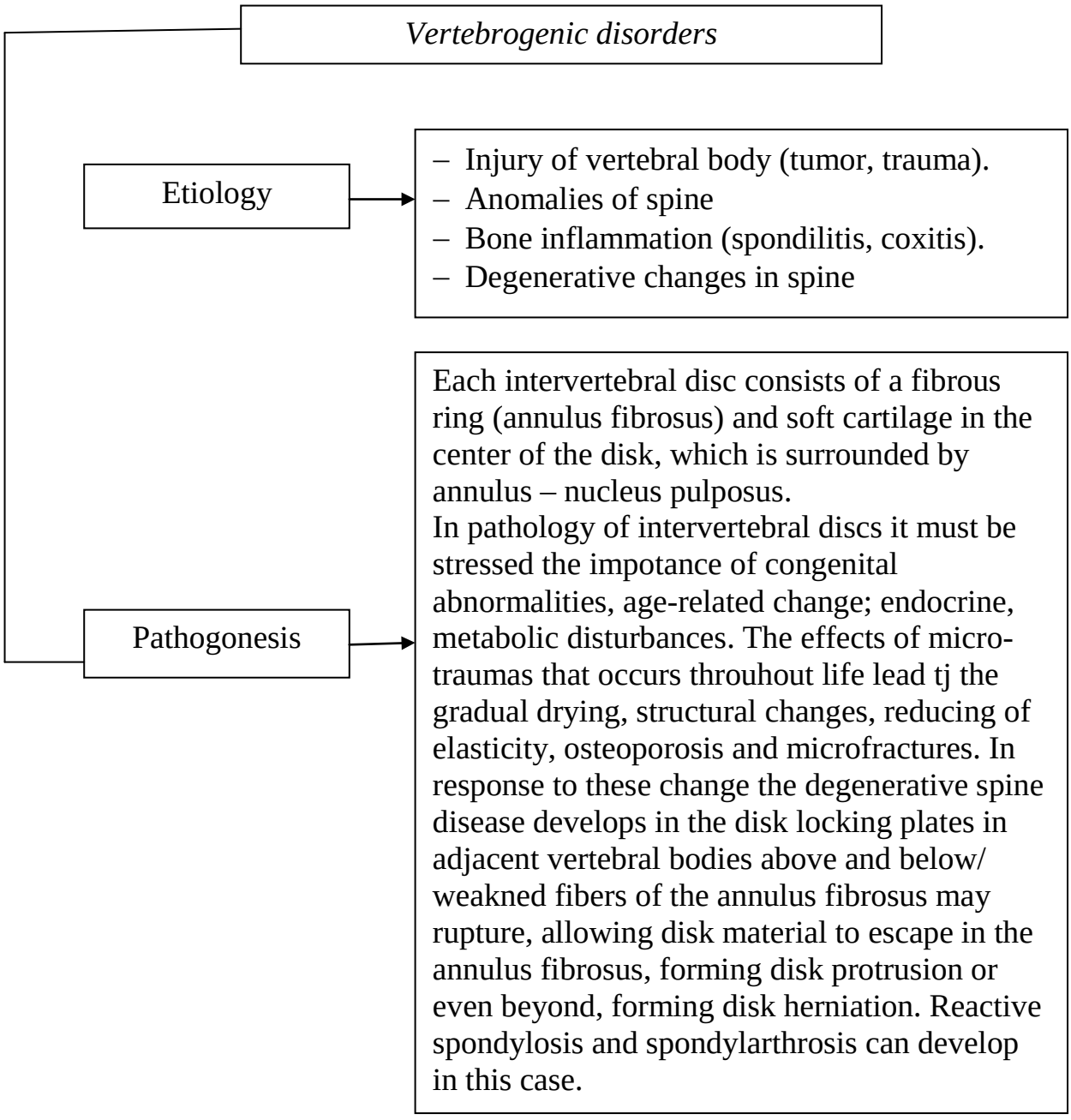
THEMA: AMYOTROPHIC LATERAL SCLEROSIS (ALS)

Progressive neurodegerative disorders, the hallmark of which is the destruction of central and peripheral motor neurorons. There are sporadic and hereditary amyotrophic lateral sclerosis. Basically, the disease affect people of 50-70 years old.

Etiology	Sporadic ALS cases is unknown. In 5 percent cases, there is an hereditary form. Exitoxic peripheral neurons and central neurons and central neurons damage, due to increased function of glutamate receptors.
Pathomorphologi	Degenerative chages of anterior horn cells of spinal cord (redgion cervical, lumbar segments), brain stem (nucleus VII, IX, X, XI, XII pair of cranial nerves and nucleor paths) and pyramidal al tracts of localization in lateral columns of spinal cord.
Main clinical form	Cerebral (high). Bulbar. Bulbar-spinal. Bulbar-sacral
Diagnostic	Main neurological symptoms: a combination of signs of spastic and flaccid paresis, fasiculations of muscules a fibrillation. Prevalence of paresis over atrophy. Additions methods: EMG
Differential diagnosis	Tumor brain, spinal cord. Syringomyelia. Spondilogenic cervical myelopathy.
Treatment	Slowdown of disease progression therapy is the antiglutamate drugs. The main one is riluzde (Rilutek). Symptomatic treatment. M-anticholinergics: acetylcholinesterase (dysartria, disphagia). Carbamazepine, Clonozepam, Baclofen (involuntary limbs lerking). Phenytoin or carbomazepin (muscle spash and pain in hands and feet) and over.


Clinical features of ALS
Typical symptoms: a combination of peripheral and central paresis of limbs with brain stem and supranuclear structures.
Bulbo-spinal form
Lession anterior horns at cervical enlargment typical: fibriller muscle twitching with gradual atrophy of hands (arm), sholder muscles, sholder girdle and chest, hand “monkey paw”. Pyramidal patway is defeat alongside with peripheral motor neurons at this level hands paresis mixed: firstly deep reflex and muscle increased present pathologycal pyramidal reflexes sensetivity violation and pelvic disorders are not typical
Bulbar form
Lesion of motor nullei in medulla oblongata dulbsr syndrome, which is clinically manifested with dysartria, disphonia, disphagia, fibrillation are observed in tongue: later atrophy of tongue muscles occurs. Previously an increase in the lower jaw reflex. Sometimes neck extensor muscles weakness occurs.
Lumbar sacral form
The lumbar and sacral segments of the spinal cord (lumbar enlargement) are affected. Mixed paresis of the legs accurs: muscle atrophy, faciculations, hyperreflexia, pathological feet signs. The process is ascending in nature.
Cerebral (high) form
Pathway from the cerebral cortex to the brain stem structures (coorticonuclear pathways on both sides) are affected typical: pseudobulbar syndrom, spastic tetraparesis. Mandibular and pharyngeal reflex increases, reflex oral automatism apper, violent laughing or crying may occur. Combination pseudobulbar and bulbar syndrome.

THEMA: VERTEBROGENIC DISORDERS OF THE PERIFERAL NERVOUS SYSTEM



Classification of vertebrogenic neurology is syndromes depending on the level of lesion

Cervical level

I. Reflex syndromes.

1. Cervicalgia
2. Cervicocranialgia ofvertebral artery.

3. Cervicobrahialgia.

II. Compressive radicular syndromes radiculopathy lesion (vertebral lesion of roots).

III. Vascular radicular spinal syndromes (radiculoishemia, radiculomyeloishemia, myeloishemia).

Toracic level

Reflex syndrome: toracalgia with muscular tonic, autonotic – visceral or neurodystrophic manifestations (syndromes scapular rib, cardialgia andd over).

Compressive radicular syndrome.

Lumbosacral level

Reflex syndromes with muscular tonic, vasomotor and thropic disorders.

1. Lumbago
2. Lumbalgia
3. Lumbalishalgia

Compressive vascular radicular (radiculopathy).

Compressive vascular radicular – spinal syndrome (radiculoischemia, radiculomyeloischemia, myeloischemia).

Nerve stretch test

Vertebrogenic disorders are often characterized by pain while palpating the paravertebral region and by positive stretch tests:

- *Lasegue's sign (straight Leg rise).* Patient is lying down on his back; bending of leg in the coxal joint cause's pain in the lumbar area and on course of sciatic nerve (this is result of nervous root and sciatic nerve stretch).

- *Wassermann's sign (femoral nerve stretch test).* Patient is lying down on his stomach; unbending of leg in the coxal joint cause's pain in the lumar area and on the front surface of thigh (this is result of nerve root and femoral nerve stretch).

- *Neri's sign.* Bending of head causes pain in the lumbar area and knee flexing (this is a result of nerve root stretch).

Neurology syndromes of vertebregic disorders of peripheral nervous system

	Reflex, syndrome						
	Cervical level			*Thoracic level*	*Lumbosacral level*		
	Cervicalgia	Cervicocranialgia	Cerebrobrahialgia	Thracalgia, dorsalgia	Lumbago	Lunbalgia	Lumbaischalgia
CLINICAL SIGNS	Acute, sybacute pain of neck. The pain is dullaching, bussting character pain increase with the movement of the head typical musculas tonic syndrome lesion of level C4-C5, C5-C6.	Syndrome vertebral arterior. Headache (temporal, parietal region), vestibulo coxlearis, visual and ear disorders. Dizziness may be drop-attack lesion level C5-C6, C6-C7, C7-Th1.	Pain in muscles of neck, sholder and arm, elboww and sholder joints, tension and limited mobility in these joints with possible arising of scopulo numeral periartrosis Steinbrocker (shoulder hand syndrome).	Pain in the chest back contractions of the thoracic muscles of the back, limited movement due to pain, absence of sensory motor or reflex abnormalities.	Acute pain at the lumbar level after phisical activities or awkward movements, accompanied by a significast constriction of the back muscles and limited mobility. It lasts from several hours to several days.	Sybacute and chronic pain at the lumbar level. Sensory disorders absent. Reflexes do not change.	Sybacute and chrionic pain at the lumbar level, that extends to the buttocks, lower limb sometimes both limbs but does not fall on the boot and fingers. A combination of muscular tonic and autonomic – vasonotu, neurodystrophic biolations.

Compressive syndrome

	Cervical level	*Thoracic level*	*Lumbar levrl*	*Spinal stenosis*
CLINICAL SIGNS	The features: - signs of reflex syndromes; - seasory deficits – hypalgesia in the area of innervation; - motor and reflex deficitis – muscular weakness and atrophy, reflexes are usually markedly diminished or absent; - autonomic disorders – skin atrophy, hyperhidrosis; -electromyography reveals of conduction velocity of those nerves, which are formed by certain roots. Nerve root C6 (intervertebral C5-C6 foramen): pain is projected from neck into the thumb; hypoesthesia of radial foream and thumb; biceps weakness, decreased or absent of biceps reflex. Nerve rooy C7 (intervertebral C6-C7 foramen): pain is projected intothe back surface of shoulder and foream to the	A disk herniation can compress a thoracic nerve root with sensory and motor deficit. These syndromes are rare.	A feature: see the compressive syndromes at the cervical level. Compressive syndromes at the lumbar level are most common. Sciatica is the clinical description of pain in the leg that occurs due to lumbrosacral nerve root compression usually secondary to lumbar disk prolapse or extrusion. L5-S1 disk level is the most common site of disc herniation. The following are the characteristic “lower back syndromes” associated with nerve root compresion. Nerve root L4 (intervertebral L4-L5 foramen): pain and hypoesthesia on the front of the thigh and the inner tibia surfacee, weakness and atrophy of the quadriceps muscle, decrease or loss of	Spinal stenosis is disorder that is caused by a narrowing of the spinal canal. This narrowing happens as a result of the degeneration of both the facet joints and the intervertebral discs. In this condition, bone spurs (also called osteophytes) grow into the spinal canal. The facet joint also enlarge as they become arthritic, which contributes to a decrease in the space available for the nerve roots. There are complaints on pain in the buttocks, thigh or leg that develops with standing or walking, and improves with rest. In some cases, a person will complain of leg pain and weakness without having any back pain. More severe symptoms include numbness, tingling, and weakness in the lower extremities. Certain

	middle finger; weakness of triceps and extensor fingers muscle; decreased or absent triceps reflex. Nerve root C5 (intervertebral C4-C5 foramen): pain in the shoulder; weakness of the deltoid, supra- and infraspinatus muscles.		knee reflex. Nerve root L5 (intervertebral L5-S1 foramen): pain and hypoesthesia in the buttocks, outer thigh surface, anterior outer surface of the calf, thumb; the weakness of the extensor muscles of foot and big toe, hypotonia and muscle hypotrophy on the front side of shin. A patient has diffculty to stand on the heels. Nerve root S1 (intervertebral S1S2 foramen): pain and hypoesthesia in buttock, on the outer surface of thigh, calf, foot, little toe; weakness of flexors of the foot and big toe; reduced or absence of Achilles reflex. A patient cannot stand on toes.	positions can alleviate the symptoms of spinal stenosis by increasing the amount of space available for the nerves.

The diagnosis of radicular syndromes and disk herniation

X-ray of the spine at different levels to diagnose injuries, osteoporosis, anomalies of the spine, bone changes, indirect signs of intervertebral dic herniation.

MRI of spine and spinal cord (degenerative changes of the spine, joints, intervertebral disc herniation, spinal cord patholology).

Conservative treatment of vertebrogenic disorders

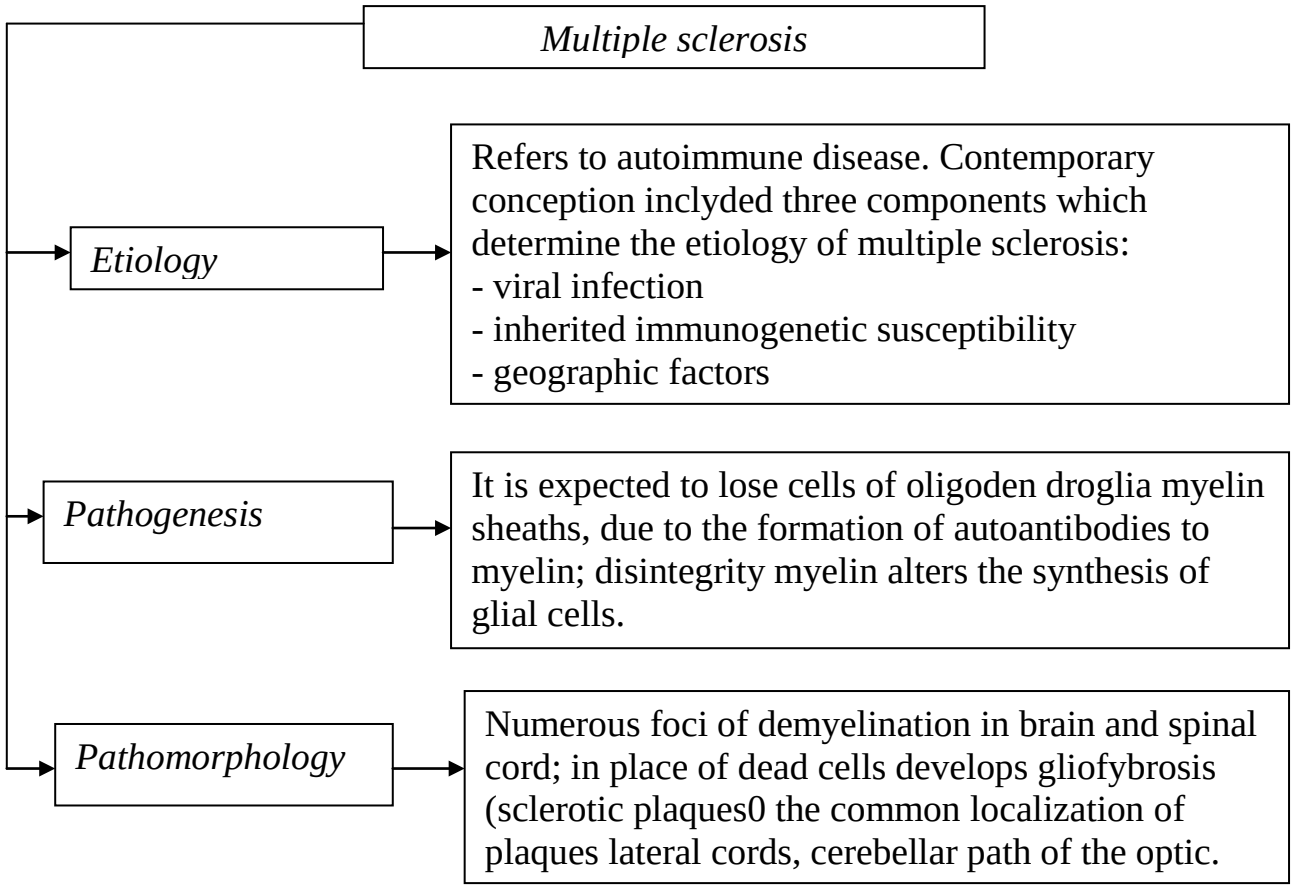
Acute period (its duration in case of reflex syndromy is up to 3-5 days, and in radicular syndrome it is 2 weeks).

1. Immobilisation, bed rest on hard surface, such as a film mattress or the floor.
2. Spine extension (on sloping surface).
3. Dehydration, using diuretics during 2-3 days.
4. Anaecthetic blokades (lidocaine, corticosteroids).
5. Nonsteroid antiinflammatory preperations: diclofenac, ketophrofen, desketophrofen, ketorolac, meloxicam, nimesulide, ibuprofen.
6. Myorelaxants: baclofen, sirdalud.
7. Vitamin B complex.
8. Physiotherapy (electrophoresis, phonophoresis, laserotheraphy), local anesthetic procedures.
9. Massage, gymnastics.

After acute period, a maximal effect has physiotherapy, massage and gymnastics.

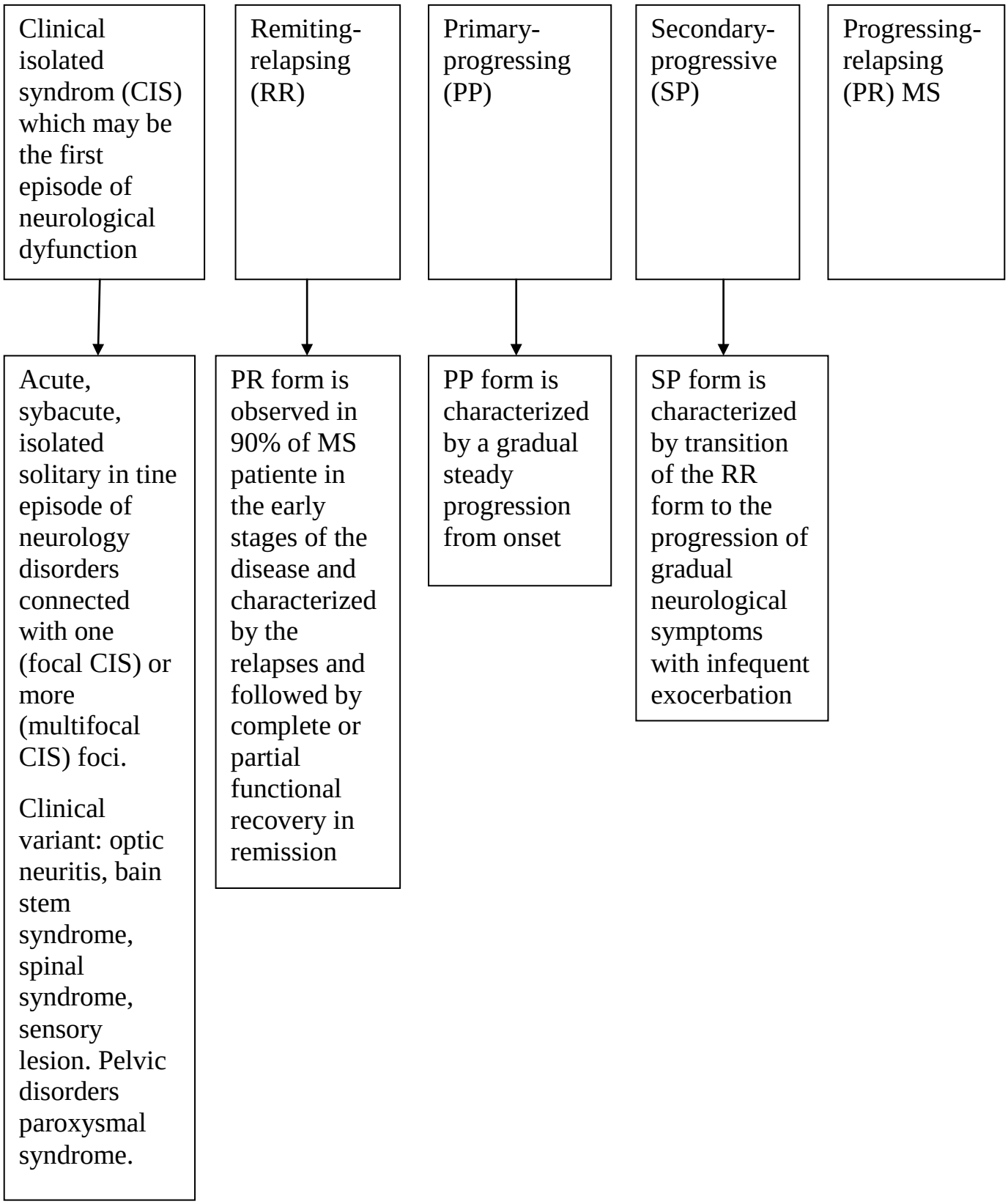
Chiropractic manipulation in vertebrogenic disorders is contraindicated in patients with disk herniation, as soon as it may lead to damage of the spinal cord.

THEMA: DEMYELINATING DISEASE: MULTIPLE SCLEROSIS (MS), ACUTE DISSEMINATED ENCEPHALOMYELITIS (ADEM)



Classification MS

1. By the type of disease:



2. By the period of disease:
I – relapse
II – remission (first remission longer than the next)

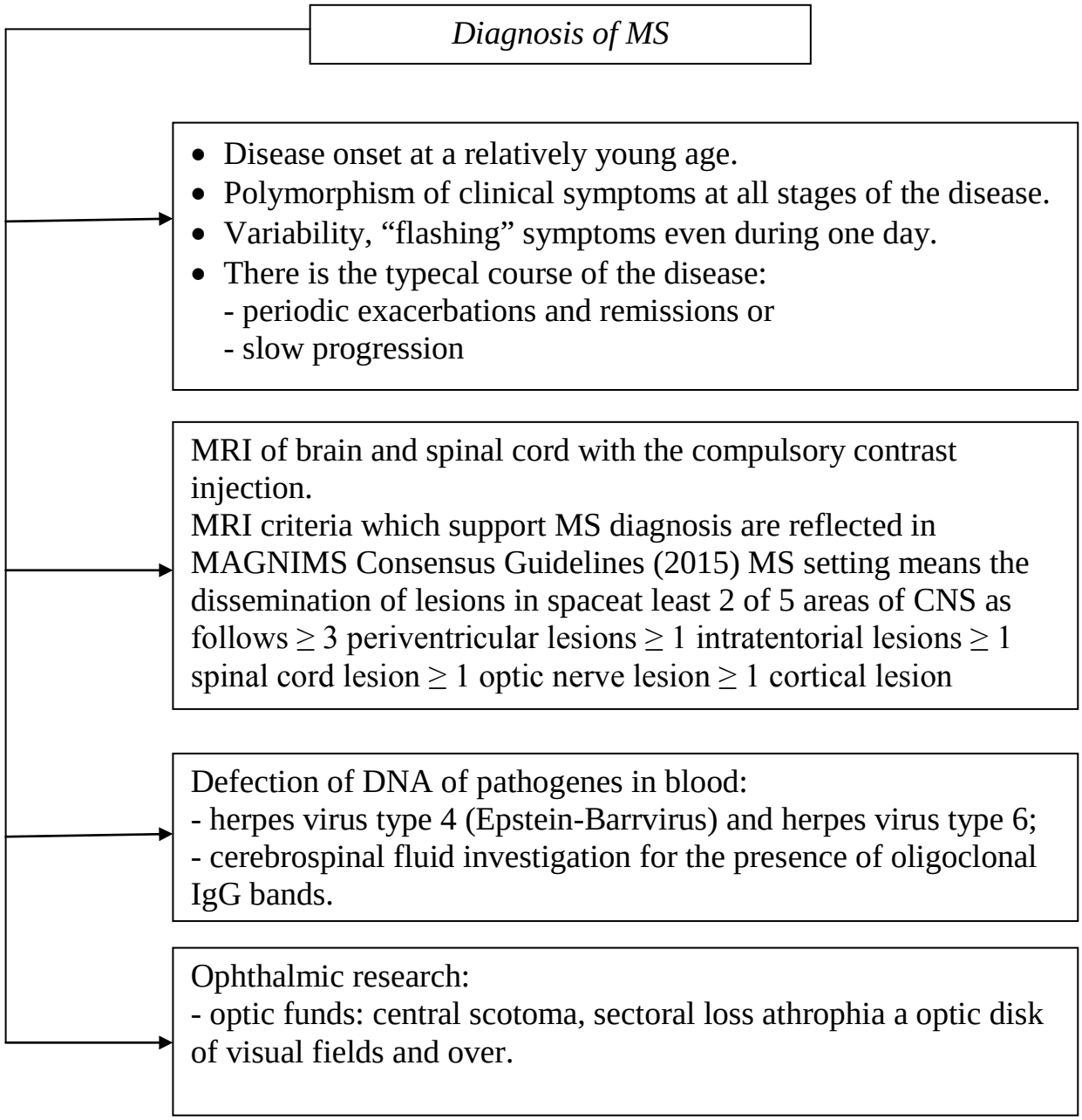
Clinical symptoms and syndromes of multiple sclerosiis

Clinical manifestations are associated with focal lesions of brain and spinal cord. Functional system score is used most commonly to assess the merological manifestation. This score evaluates the severity of symptoms of various major CNS systems.

Pyramidal tract lesion	Hemi-, para-, three- or tetraparesis. Monoparesis are observed rarely. Lower limbs suffer more likely than upper. Spasticity may prevail over the severity of paresis and is characterized by the restriction of active movement's involuntare reflex muscle spasm.
Cerebellr lesion symptoms.	Static and dinamic ataxia, the main manifestation of whih are body balance and gait disorders, dysmetria, asynergia, muscular hypotonia.
Brain stem and cranial nerve lesion	Eyes movement disorders, peripheral paresis of the mimic muscles 9face), nystagmus trigeminal neuralgia, bulbar disorders (dysartria, dysphagia, dysphonia).
Visual disorders	Single or bilatersl decrease in visual acuty colar perception violation; pallor of the temporal halves of optic disks, atrophy of optic disks; occurrence of scotomas.
Sensory disorders	Parasthesia and dysesthesia, deep sensetiviti disorders (vibrative), conductive anesthesia and segmental disorders – later stage.
Dysfunction of pelvic organs	Disorders of urination: incontinence, imperative urgensyy incontinenece, bladders emptying disorder, urine retention, disorders of defecation and sexual dysfunction.
Mental activity changes	The disorders of attention deterioration, memory, mood, hight level of anxiety is social contracts, emotional tension.
Chronic fatigue syndrome means	General fatigue decrease of working copacity without connection with depression and muscle weakness.
Paroxysmal states	Epileptic and nonepileotic origin are manifested by seizures, autonomic visceral paroxysms, syncopes, migraine.

The specific features in multiple sclerosis

Clinical dissociation is the disrepancy of dysfunction degree to objective neurological status date	1. In case of externally satisfactory state of the patient and absence of movement disorders, the hyperreflexia, refuxes are observed. 2. The optic disks may be changed without the clinical signs of visual analyzer disturbances.

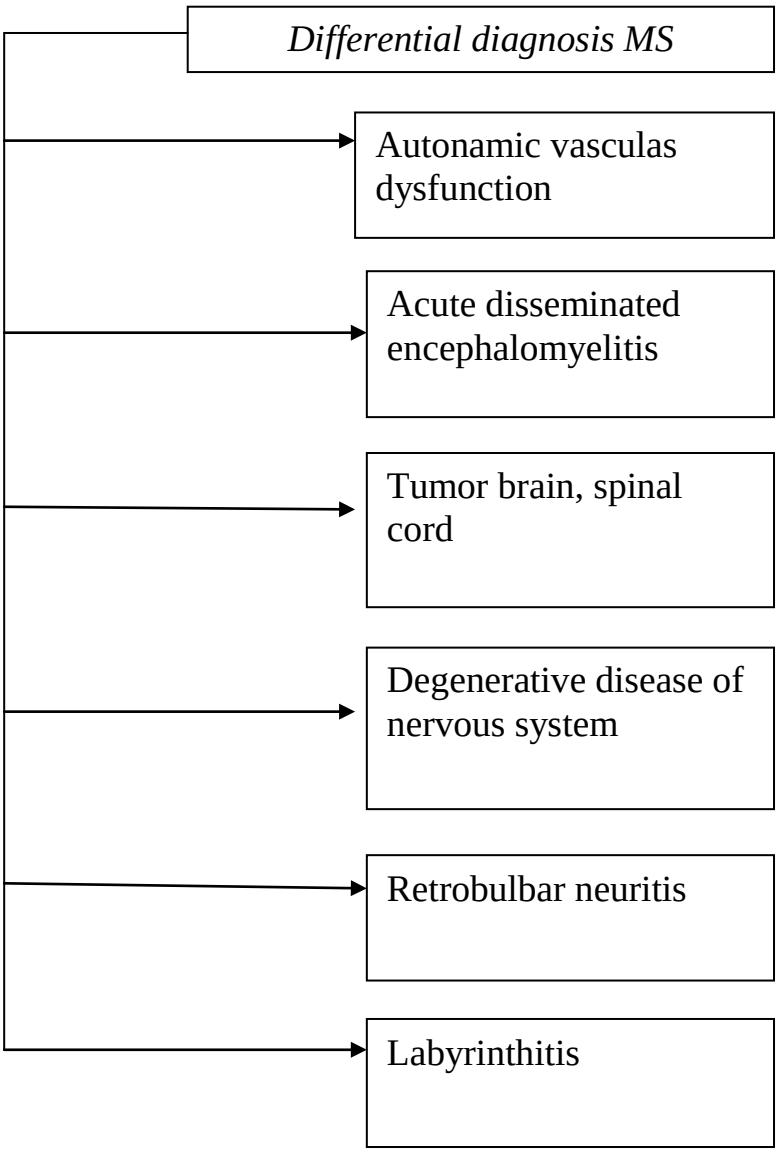


McDonalds criteria

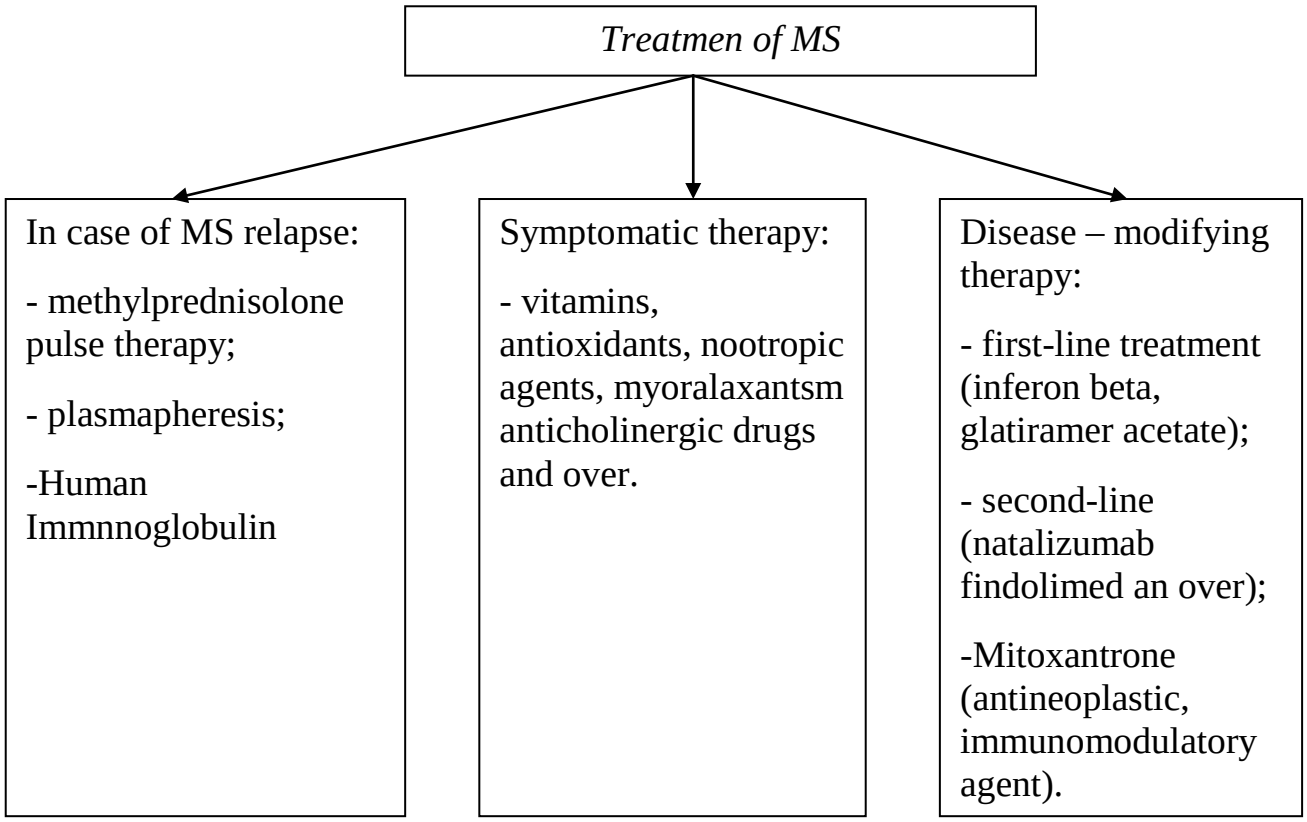
They are the most widely used criteria for evidence of “disseminztion of lesions on plase and in time”. These criteria take into accound both the clinical

manifestations and MRI of brain and spinal cord, and presence of oligoclonal immunoglobulin in cerebrospinal fluid.

Differential diagnosis MS



Treatmen of MS

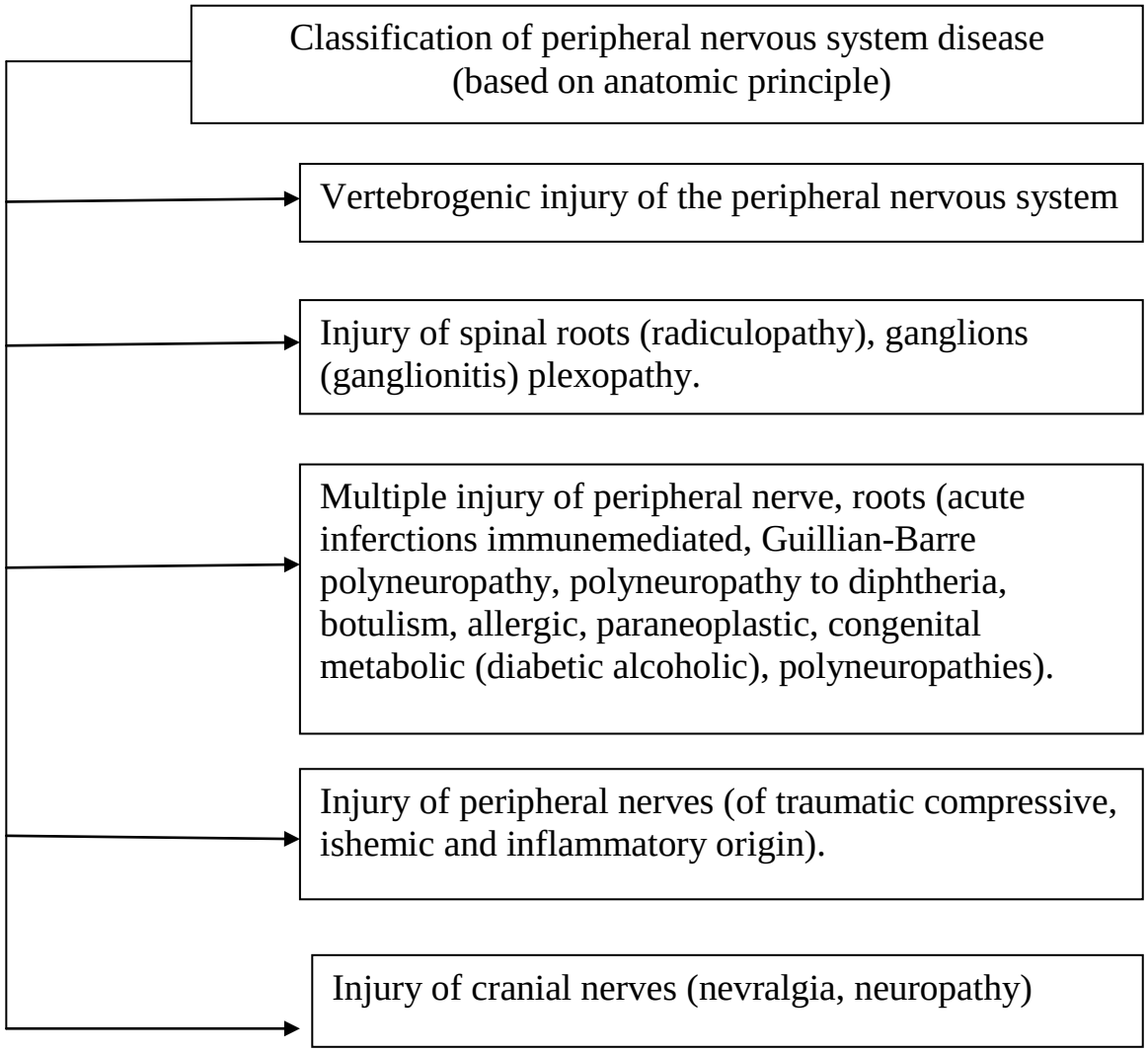


Acute disseminated encephalomyelitis (ADEM)

Etiology, pathogenesis	ADEM is an acute imfectious and allergic disease, in which the inflammatory foci of demyelization in braim and spinal cord are observed, both white and gray matter are affected. Sheth, roots and peripheral nerves can also be damaged. The formation of scleroyic plagues in ADEM is possible. Adem is divided into primary, which develops because of primary impact of fitering virus to the nervous system, and secondary, which occurs on a background of influenza, malaria and other acute infections. The main feature of ADEM is the development of disse,inated inflammation, perivascular inflitration by lymphocytes or macrophages, or monocytes, especially around small and medium-sized veins; perivascular demyelination is characteristic.
Clinical features	There are: - presense of recept acute viral infection or vaccination; - acute onset with fever and symptoms of imtoxication, characteristic changer in peripheral blood; - meningeal syndrome; - neurological disorders indicated the disseminated disorders of nervous system.
Diagnosis	- Clinical picture; - MRI data can reveal the multifocal changers in the white matters of the cerebral hemispheres, cerebellum and brain stem; - CSF data: a slight increase of protein content and lymphocytic pleocytosis (up to 100 cells in 1 ml). The courseof disease is acute and often with severe state of the patient. After 3-4 weeks symptoms regress. Exacerbations unlike multiple sclerosis are not observed.
Differential diagnosis	- Multiple sclerosis - Tumor brain and spinal cord - Meningoencephalomyelitis
Treatment	Acute stage: corticosteroid, antibiotics, antivirals, antihistomines. Recovery srage: anticholinergitic, vitamins, neurotrophic agents, physiotherapy, massage and over.

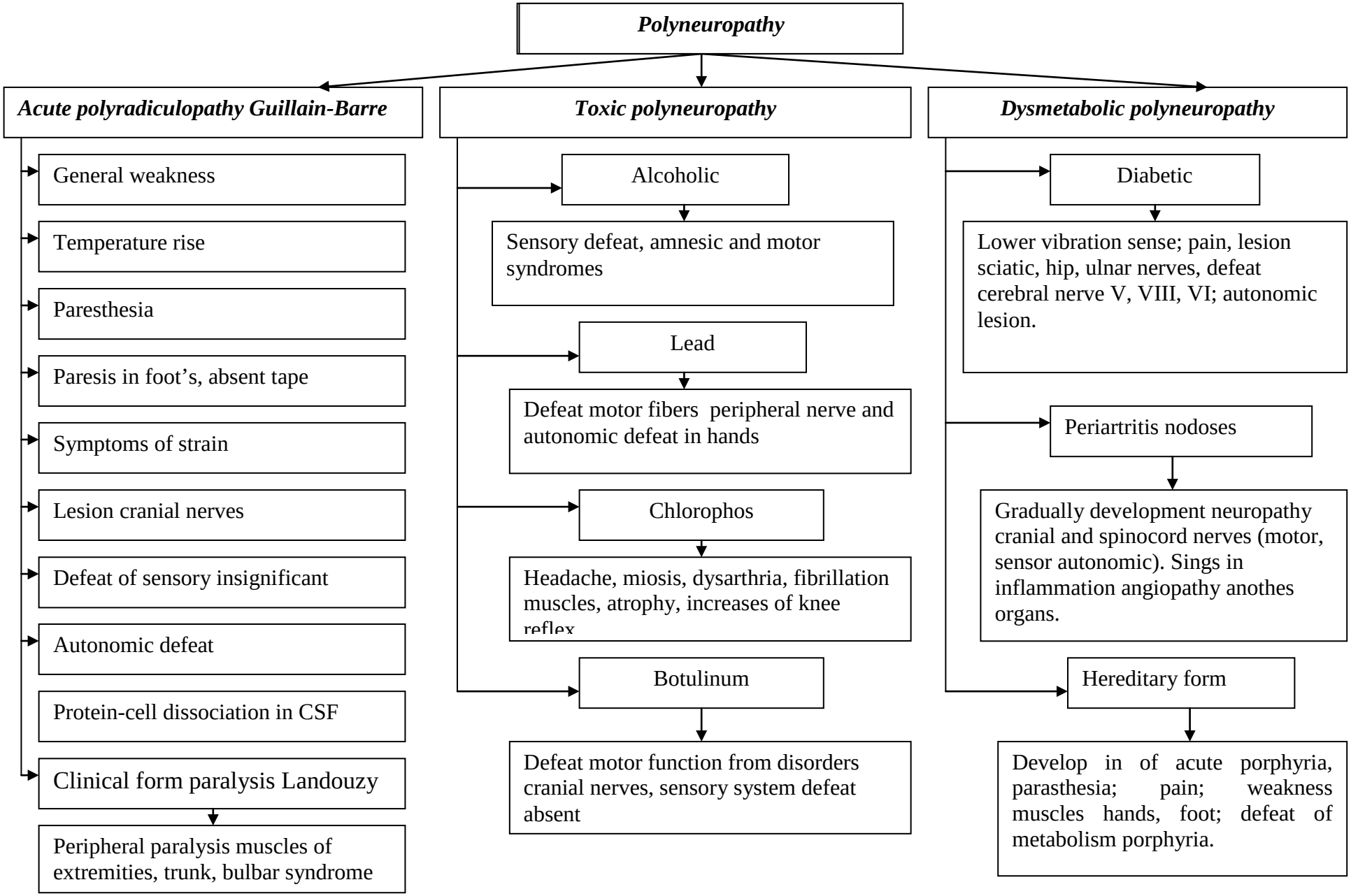
THEMA: PERIPHERAL NERVOUS SYSTEM DISEASE (PNS). CLASSIFICATION. POLYNEUROPATHY (GULLIAN-BARRE SYNDROME)

Classification PNS



Polyneuropathy

Etiology	– Viral and bacterial infections. – Tocxication (alcohol, arsenic compouds, lead, mercury and ets). – Iatrogenic facrors prising from the treatment with bismuth, salts of gold, isoniazid, chemotherapy and other. – Connective tissue disease; vasulitis and other. – After the introduction of serums and vaccines. – Vitamin deficiency. – Paraneoplastic processes. – In case of the disease of imternal organs endocrine glands, the genetic defects.
Pathogenesis	– Demyelinating polyneuropathy. – Axonal polyneuropathy
Pathomorphology and topic	– Distal-symmetric segmental demyelination of nerve fibers. – Degenerative-dystrophic process of axial cylindrs of peripheral nerves.
Main clinical syndromes	– Polyneuropathic syndrome: a) Peripheral distaltetraparesis b) Disorders of sensetivity in distal parts of handsand feet c) Pain and autonotic-throfic – Isolated form with a primary lesion motor, sensory or autonomic (vegetative) libers.
Diagnostic and differencial diagnostica	Anamnesis, symptoms: – Eletromyograhy and nerve conduction studies (signs of demyelination), determinant in serum antibodies to myelin peripheral nerve. – With all forms of neutopathies, Raynaud's disease, disease with liver connective tissue, blood disease.
Principle of treatment	Plasmapgeresis, hemosorption, antiviral drugs, corticosteroids, antiholinergec, anticonul sants, vitamins B, ascorbic asid, L-lipoic acid, antihistamines drugs, diuretics, physiotherapy, massage.

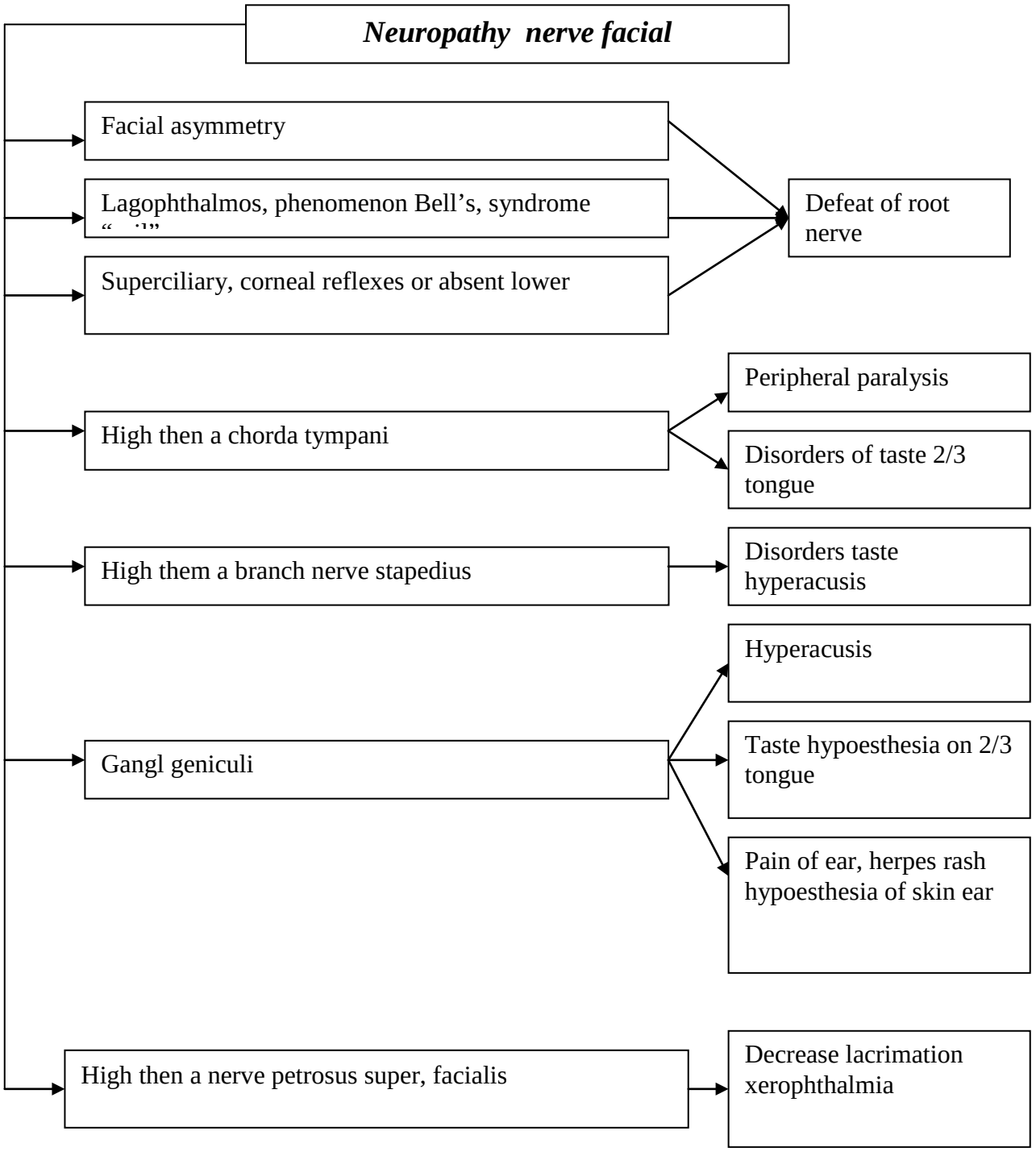

Polyneuropathy
Acute polyradiculopathy Guillain-Barre
Toxic polyneuropathy
Dysmetabolic polyneuropathy
General weakness
Temperature rise
Paresthesia
Paresis in foot's, absent tape
Symptoms of strain
Lesion cranial nerves
Defeat of sensory insignificant
Autonomic defeat
Protein-cell dissociation in CSF
Clinical form paralysis Landouzy
Peripheral paralysis muscles of extremities, trunk, bulbar syndrome
Alcoholic
Sensory defeat, amnesic and motor syndromes
Lead
Defeat motor fibers peripheral nerve and autonomic defeat in hands
Chlorophos
Headache, miosis, dysarthria, fibrillation muscles, atrophy, increases of knee reflex
Botulinum
Defeat motor function from disorders cranial nerves, sensory system defeat absent
Diabetic
Lower vibration sense; pain, lesion sciatic, hip, ulnar nerves, defeat cerebral nerve V, VIII, VI; autonomic lesion.
Periartritis nodoses
Gradually development neuropathy cranial and spinocord nerves (motor, sensor autonomic). Sings in inflammation angiopathy anothes organs.
Hereditary form
Develop in of acute porphyria, parasthesia; pain; weakness muscles hands, foot; defeat of metabolism porphyria.

Primary polyneuropathy

Guillan-Barre syndrome (Landry's ascendimg paralysis) – acute inflammatory demyelinating polyradiculoneurophthy with flassid paresis sensory and autonomic disorders.

Etiology	Guillan-Barre syndrome is unknown. Cause of disease: surgery, infection and viral disease, inflammation of the salivary gland, malignancies, lymphoma, vaccinations, HIV.
Immunopathogenesis	This syndrome causes the dstruction, removal, or loss of the myelin sheath of a nerve, Cuillain-Barre syndrome is considered as acquired immune neuropathy that develops because of pathological immune response to vaccination, viral infectioon, ets. Autoimmune reaction against myelin antigens of peripheral nerves leads to edema, infiltration and lymphocytic segmental demyelination of spinal and cranial nerves. Autoimmune reasction against axons of peripheral nerves leads t axonal variant of syndrome (less often).
Main clinical signs: Landry's syndrome, Miller-Fisher syndrome.	Typical: it begins with muscle weakness and (or) sensory disorders (membness) in the lower limbs, which in a few horsers or day spread to the hands – tetraplegia. Objective sensory changes are minimal. Cranial nerve involvement (III-VII and IX-XII) can be observed: fasial drop, diplopia, dysarthria, dysphagia, oropharyngeal weakness. Pain is most severe in shoulder girdle, back, butoks. Autonomic nervous system involvement with dyslunction in the sympathetic and parasympathetic system: paroxysmal hypertension, orthostatic hypotension, tachycardia and bradycardia, dysfunction of pelvic organs are not typical. The Miller-Fisher type – lesion of oculomotor nerve, ataxia, areflexia, cerebellar ar.
Current of disease	The duration of the augmentation of symptons is 2-4 weeks. A plateau phase of persistent, unchanging symptoms lasts up to 2-4 weeks folowed by gradual symptom improvement (3-12 month).
Diagnosis	Clinical symptoms, anamnesis present, electromyography and nerve conduction studies and albuminocytologic dissociation in CSF, MRI (differential diagnostic).
Diffential diagnosis	- Polyomuelitis - With all forms of neuropathies - Myelitis - Stroke of brain steam

	- Myastenia - Botulism
Treatment	Maintenance of vital function. Medical treatment: pulse-therapy with immunoglobulens, plasmapheresis. Symptomatic therapy: anticonvulsants, vitamina, anticholiergic drugs, massage, electrical stimulation of muscles (recoveri period).



THEMA: PERIPHERAL NERVOUS SYSTEM DISEASE (PNS). PlEXOPATHIES, NEUROPATHY OF UPPER AND LOWER LIMBS. CRANIAL NEUROPATHY

Facial neuropathy

The most often primary facial nerve neuropathy is its idiopathic form Bell's palsy.

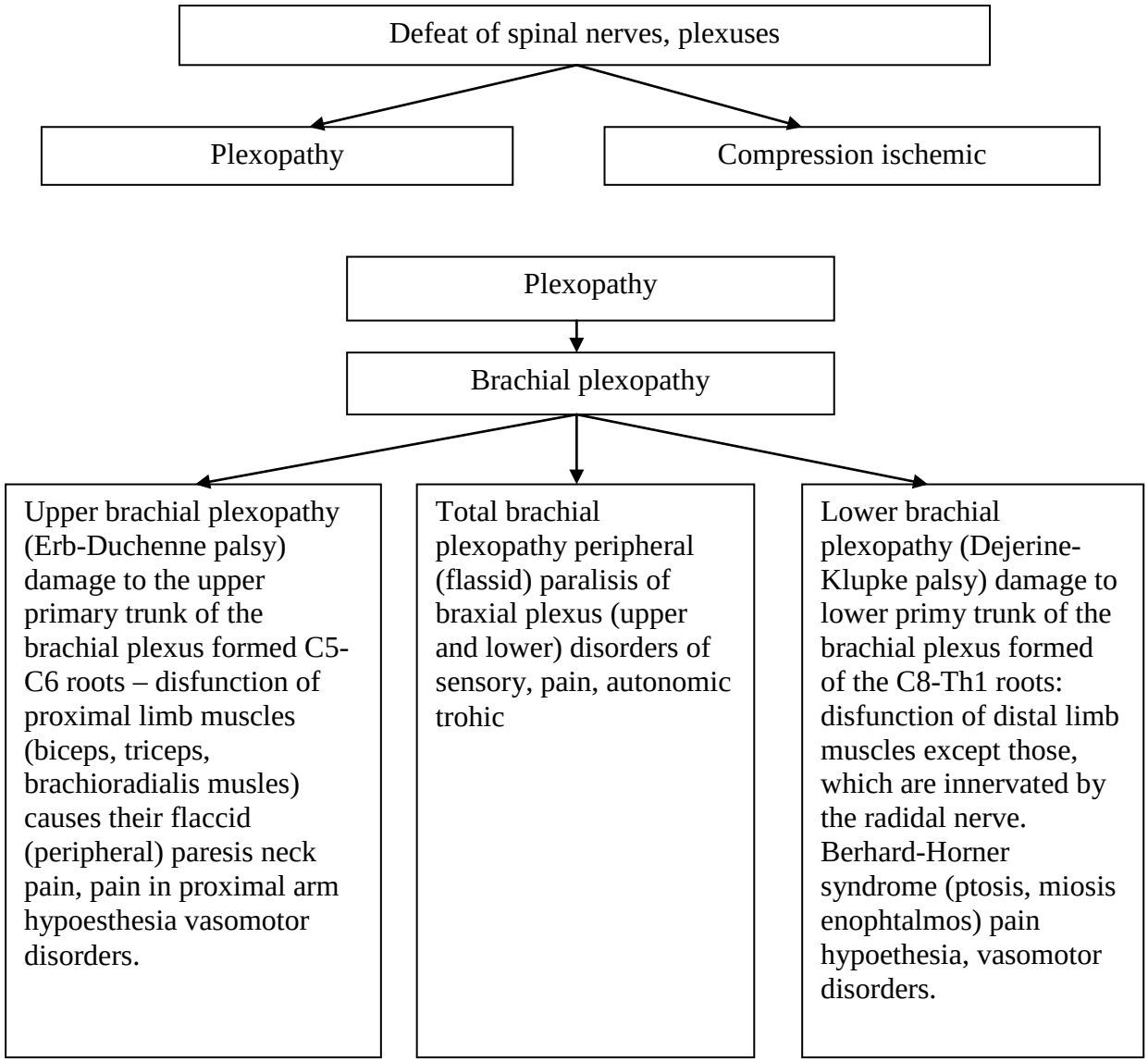
Main factors of development	The narrow bone canal in the pyramid of the temporal bone through which facial nerve passes. Endogenous or exogenous factors that together provoke facial nerve compression (tunnel syndrome).
Etiology	– Local hypothermia (cord air, air conditioner) – Of decreased immunity – activation of viruses, which persist facial nerve ganglion HSV1, mumps virus. – Infection (tick-bonne encephalitis lyme's disease, polyomyelitis). – Inflammation of the ear. – Face and skull traumatic injuries. Additional causes: stroke, diabeties, arterial hypertension, multiple sclerosis, HIV and over.
Pathogenesis	In case of primary neuropathy (Bell's palsy) as a result of above-mentioned factors there may be edema with nerve conpression and its ischemia, aseptic inflammation, that lead to the development of compression-ischemic lesion with facial nerve dysfunction.
Clinic symptomes (main)	Prosoparesis – peripheral mimic muscles paresis of the one half of the face with facial asymmetry at rest that inclease with mimic movements.
Diagnosis	- Clinical symptoms - Electroneuromyography (EMG) - CT-scan or MRI to defect focal lesions of the brain, which could cause lesion of the facial nerve.
Differential diagnosis	Limes disease, tumor brain Syndrome of Ramsei Hunt's Syndrome Melkersson-Rosenthal
Treatment	- Corticosteroids (5 day) - Diuretic (3-5 days) - Preparations for the improvement of microcirculation – pentoxifylline, nicotinic acid - Vitamin B grop. - Acetylcholinesterase (after 10-14 days) - Antiviral drugs (couser viruses) - Massage, facial muscles exercises muscle toningg

Trigeminal neuralgia

Etiology	The most common cause is it compression for additional influence of extra- and intracranial factors. Extracranial: tunnel syndrome (trigeminal nerve root compression in bone canal due to its congenital or acquired (dental, caries, sinusitis). Intracranial: aneurysm of basilar artery, tumor of ponto cerebelar angle and over. Primary (idiopatic) secondary (occurs in the backgrounde of the main disease.
Pathogenesis	It is considered that trigeminal neuralgia is caused by the appearance of paroxysmal discharges that resemble the mechanisms of epilepsy. Paroxysmal pain is generally thought to be due to aberrant transmission of nerve impulse from somatosensory to nociceptive fibers within the trigeminal nerve in a site of local damage to myelin sheaths. The myelin lesion is attributed to above mentioned factors or due to aging.
Clinic symptomes	– Recurrent paroxysms of sharp, lancinating or stabbing pain (electric shok type pain) that may last a few seconds or minutes. – Pain distribution: maxillar (II) or mandibular (III) branches of the trigeminal nerve are the most commonly affected. – Each attack is unilateral (may aiternate sides in up to 3-5% of cases). – Attacks may occur as often as multiple times daily or as infrequently as monthly, attcks become more frequent and severe over time, attcks are very rare during sleeo. – Some patients are sensetive in certain areas of the face, called trigger zones, light touch or other minimal stimulation in these zones triggers an attack. These zone are usually near the noce, lips, eyes, ear, or inside the mouth. – Everyday ativities can trigger an episode. Triggers of pai: talking eating, kissing, drinking, shaving, teeth brushing, face washing, cold explosure. – Appeatance of fasial muscles twitching at the height of the paroxysm – pain teak. – Trismus – spasm of the masticatiry muscles and redused opening of the jaws caused by trigeminal motor fibras irritation. In period between attacks, complaints and neurological symptoms are absent. At examination, these is pain at the exit points of the affected branch, but no violations of sensitivsty in the area of innervation.
Diagnosis	Anamnesis and neurology status; instrumental method MRI or CT to determine the cause of neuralgia.
Differential	– Postherpetic neuralgia

diagnosis	– Syndrome Tolosa-Hanta – Claster headache – Facialis migraine – Dental pain
Treatment	– Antiepileptic drugs: carbamezepine (Finlepsin) 600-1600 mg, gabapentin 300-2400 mg, pregabalin (Lyrica) 75-600 mg/ – Antidepressant: amitriptiline 25 mmg 3 time dayli. – Syrgery treatment: micvascular decopression, steriotactic syrgery.

Defeat of separate spinal nerves, plexuses



Lumbo-sacral plexopathy plexus is formed of the L5, S1-S2 roots

Etiologia → Infections, tumors of the pelvis abdomen

Clinic signs → Flaccid paralysis of the foot, shin, thigh adductors, loss of Achilles reflex, sensotr and autonomic disorders in the foot and shin.

Compression ischemic mononeuropathty

Upper extremities

Radial nerve neuropathy

Etiology: humerus fractures, compression od the nerve during the operation, in case of crutch using, while sleeping on arm (Saturday Night Palsy), especially after drinking alcohol.

Clinical signs:
- writs drop that is the inability to extend the wrist upward when the hend is palm down;
- inability to voluntary straighten the fingers or extend the thumb;
- loss of wrist extension due to paralysis of the posterior compartment of foream muscles;
- hypoesthesia of interval between I and II metacarpal bone of hand dorsum.

Ulnar nerve neuropathy

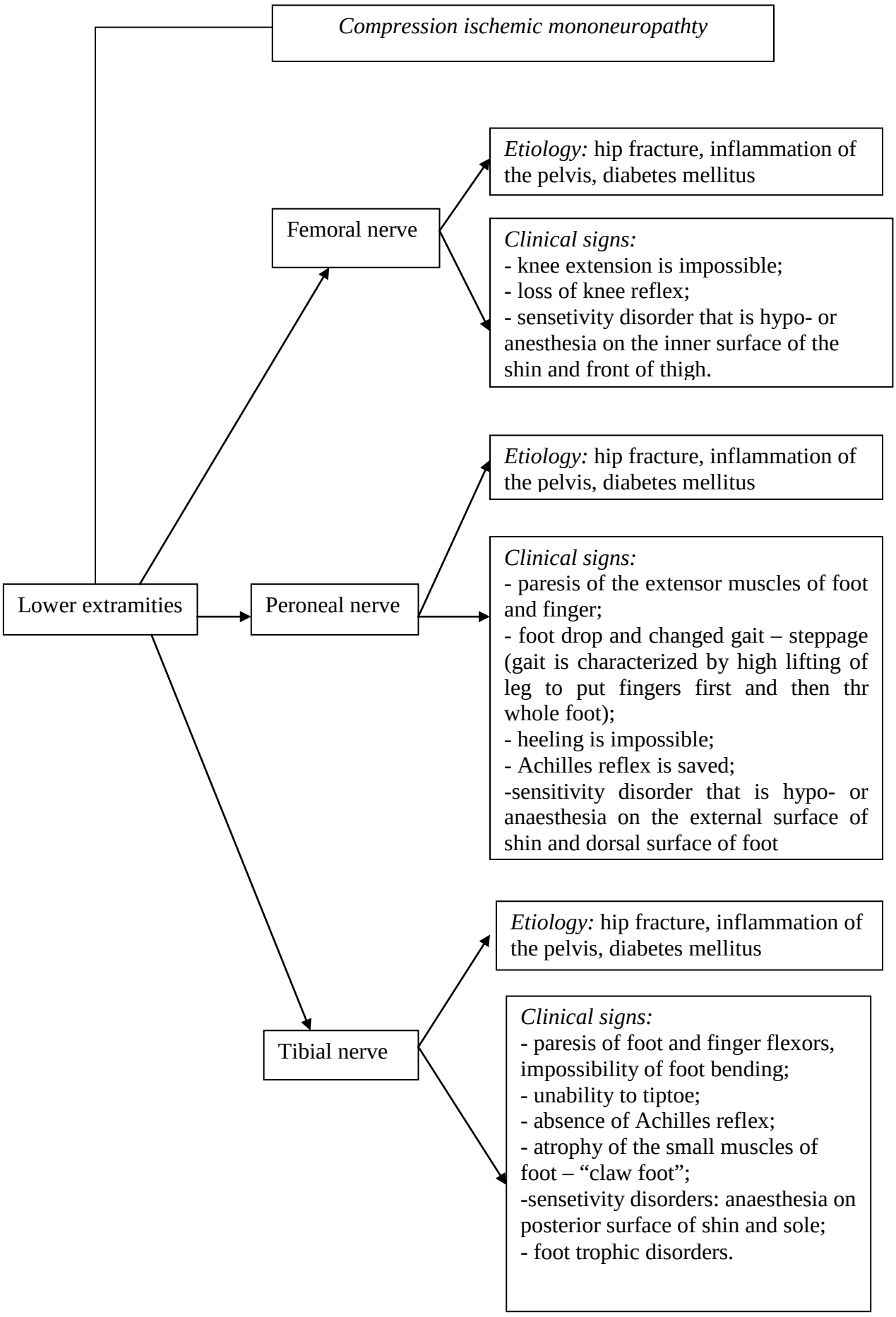
Etiology: trauma

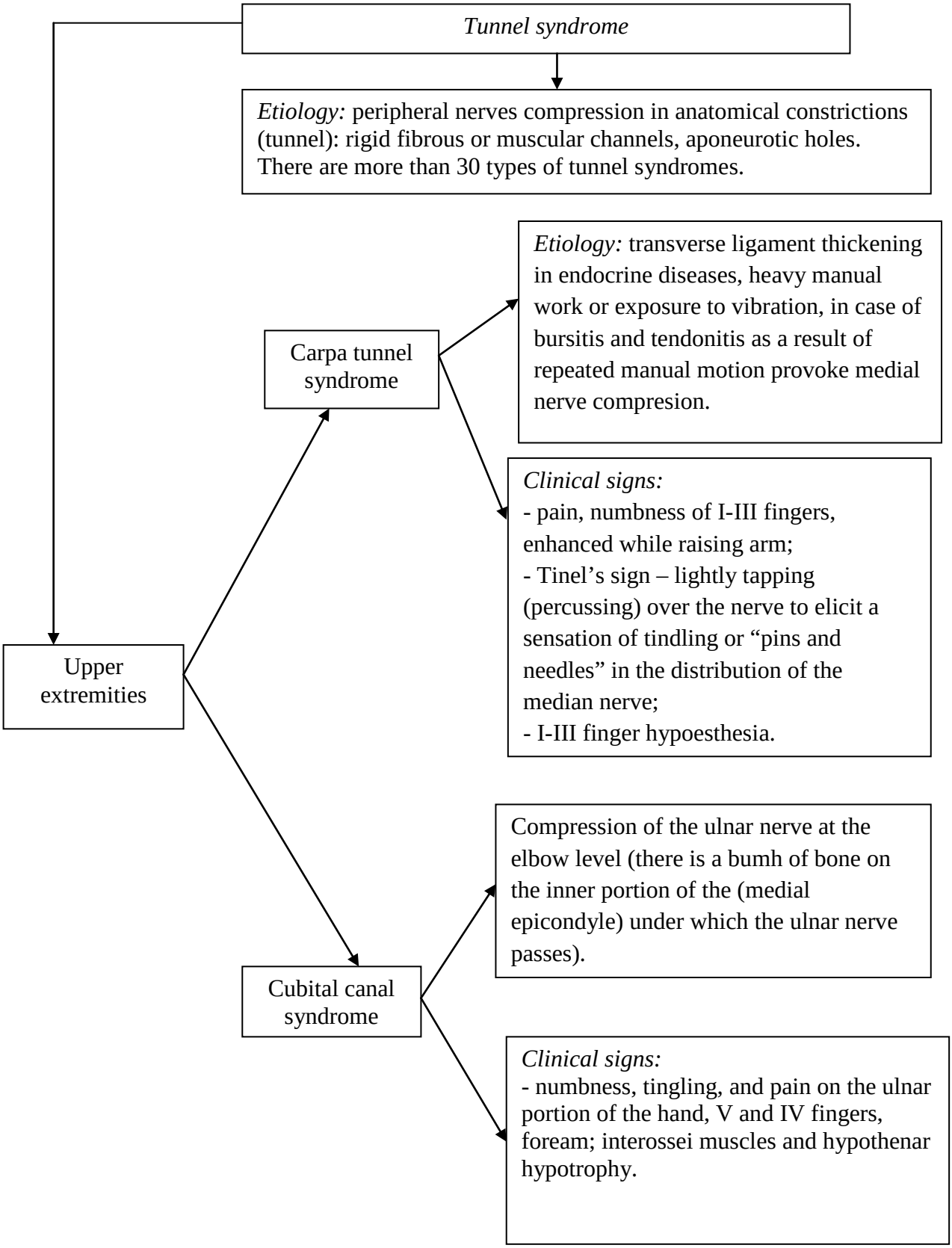
Clinical signs:
- "claw hand" – hyperextension of the IV and V finger bend up) at the interphalangeal joints (middle joints);
- atrophy of hand interossei muxcles, especially in I-II fingers;
- weakness of foream and hand elbow flexion, IV and V finger flexion;
- sensetivity disorders in dorsal and palmar surfaces of the V and medial aspect of IV finger, ulnar part of hand palmar and dorsal surfaces.

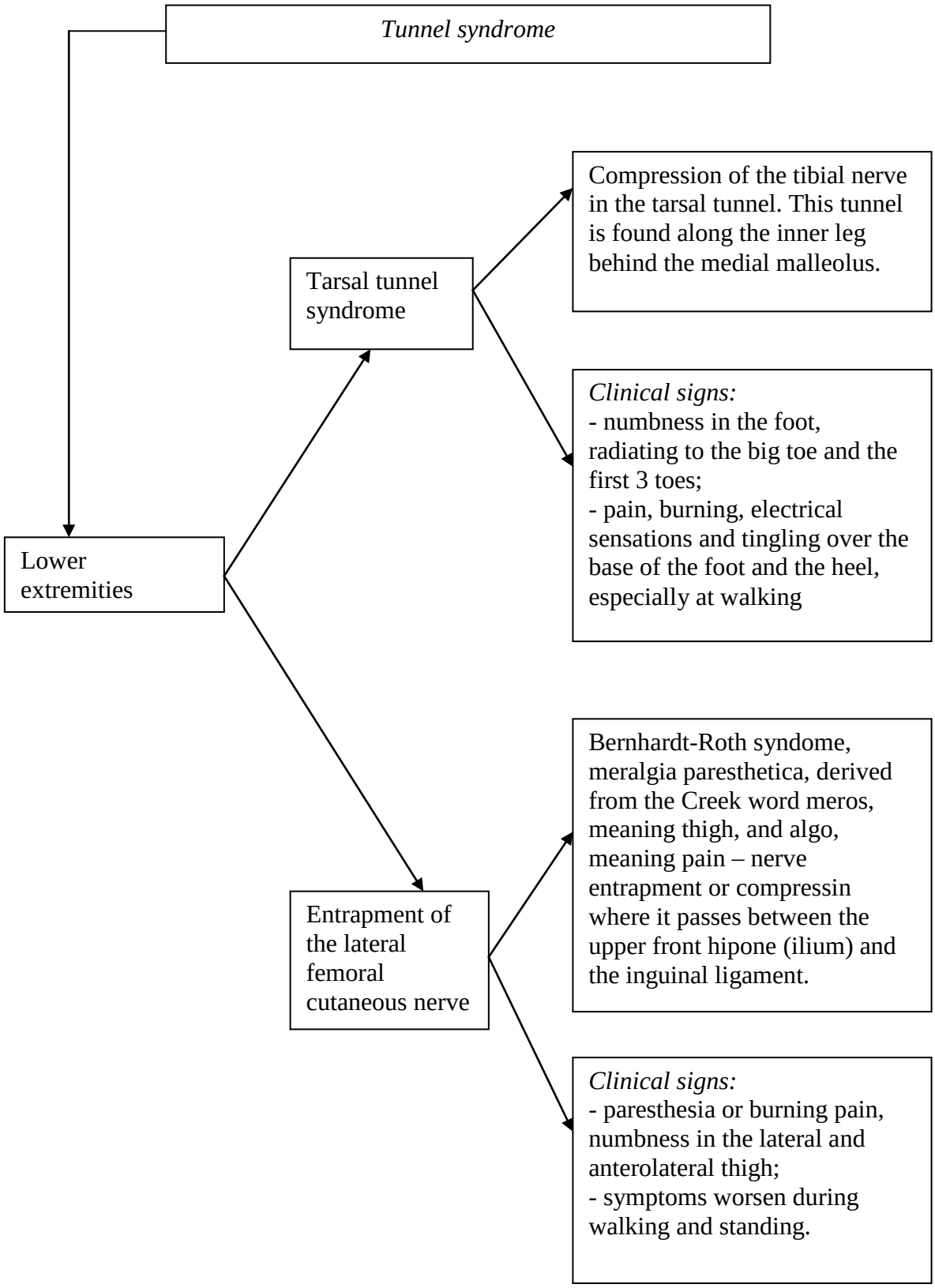
Median nerve neuropathy

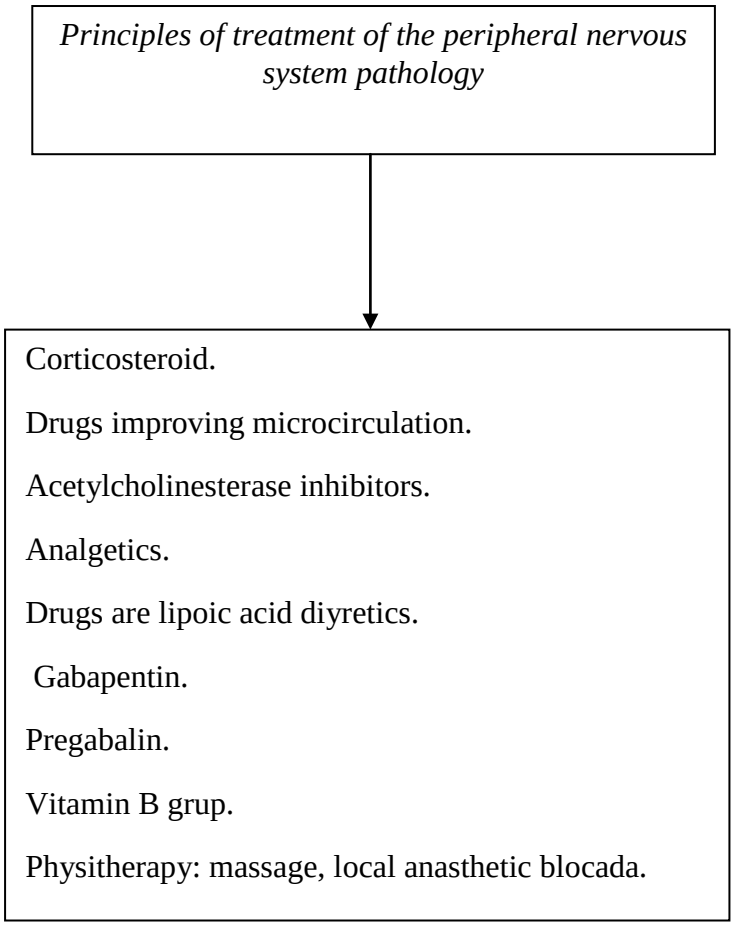
Etiology: trauma

Clinical signs:
- lack oa ability to abduct and oppose the thumb due to paralysis of the thenar muscles with their atrophy, "ape-hand deformity" forming;
- weakness of foream pronation and wrist, and in finger flexion;
- weakness of radial hand flexion and I, II, III finger flexion – impossibility of bending of the I, II and II (partially) fingers, "prophet hand" forming;
-froment's sign in case of median nerve neuropathy – a patienr is asedto hold an object, ussualy a flat object such as a piece of paper, between their thumb and inde[finger – in left hand patient can hold a piece of paper only by m. adductor pollicis;
- sensory loss in the thumb, index finger, long finger, and the radial aspect of the ring finger;
- vasimotor and thropic disorders, causalgia.


Compression ischemic mononeuropathty
Lower extramities
Femoral nerve
Etiology: hip fracture, inflammation of the pelvis, diabetes mellitus
Clinical signs:
- knee extension is impossible;
- loss of knee reflex;
- sensetivity disorder that is hypo- or anesthesia on the inner surface of the shin and front of thigh.
Peroneal nerve
Etiology: hip fracture, inflammation of the pelvis, diabetes mellitus
Clinical signs:
- paresis of the extensor muscles of foot and finger;
- foot drop and changed gait – steppage (gait is characterized by high lifting of leg to put fingers first and then thr whole foot);
- heeling is impossible;
- Achilles reflex is saved;
-sensitivity disorder that is hypo- or anaesthesia on the external surface of shin and dorsal surface of foot
Tibial nerve
Etiology: hip fracture, inflammation of the pelvis, diabetes mellitus
Clinical signs:
- paresis of foot and finger flexors, impossibility of foot bending;
- unability to tiptoe;
- absence of Achilles reflex;
- atrophy of the small muscles of foot – “claw foot”;
-sensetivity disorders: anaesthesia on posterior surface of shin and sole;
- foot trophic disorders.


Tunnel syndrome
Etiology: peripheral nerves compression in anatomical constrictions (tunnel): rigid fibrous or muscular channels, aponeurotic holes. There are more than 30 types of tunnel syndromes.
Upper extremities
Carpa tunnel syndrome
Etiology: transverse ligament thickening in endocrine diseases, heavy manual work or exposure to vibration, in case of bursitis and tendonitis as a result of repeated manual motion provoke medial nerve compresion.
Clinical signs:
- pain, numbness of I-III fingers, enhanced while raising arm;
- Tinel's sign – lightly tapping (percussing) over the nerve to elicit a sensation of tindling or "pins and needles" in the distribution of the median nerve;
- I-III finger hypoesthesia.
Cubital canal syndrome
Compression of the ulnar nerve at the elbow level (there is a bumh of bone on the inner portion of the (medial epicondyle) under which the ulnar nerve passes).
Clinical signs:
- numbness, tingling, and pain on the ulnar portion of the hand, V and IV fingers, foream; interossei muscles and hypothenar hypotrophy.

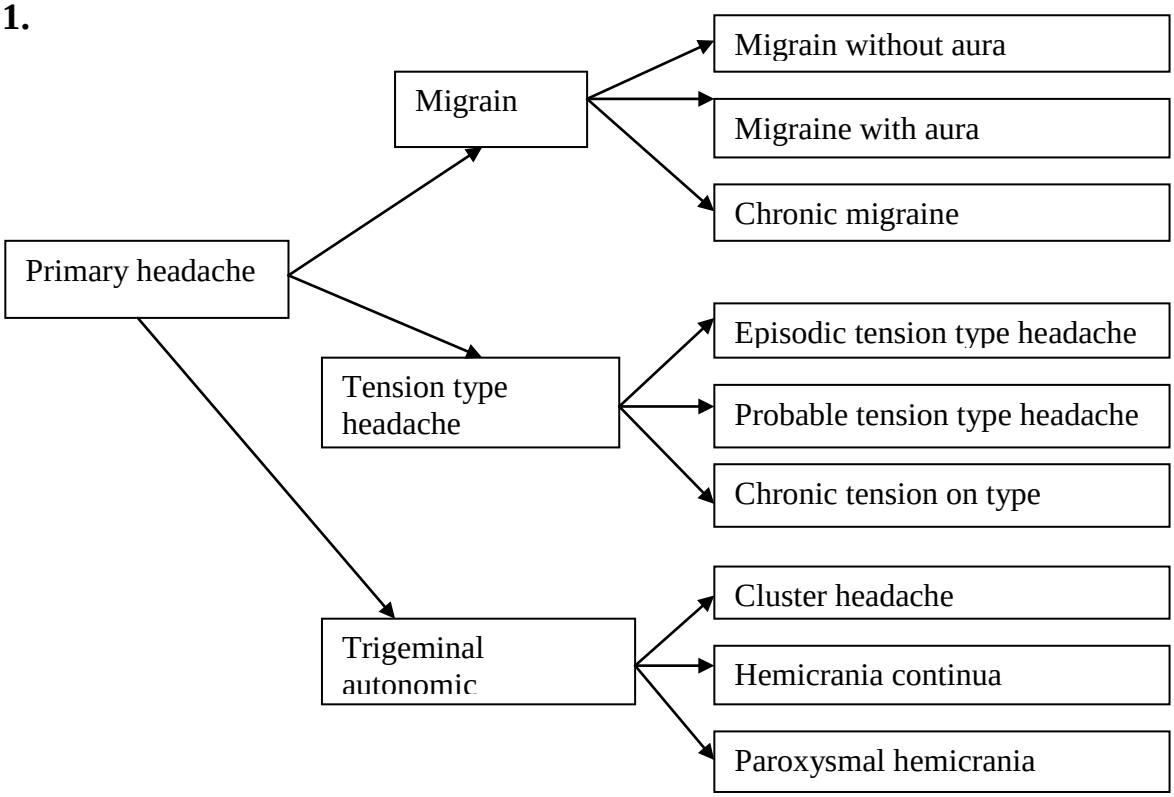

Tunnel syndrome
Lower extremities
Tarsal tunnel syndrome
Compression of the tibial nerve in the tarsal tunnel. This tunnel is found along the inner leg behind the medial malleolus.
Clinical signs:
- numbness in the foot, radiating to the big toe and the first 3 toes;
- pain, burning, electrical sensations and tingling over the base of the foot and the heel, especially at walking
Entrapment of the lateral femoral cutaneous nerve
Bernhardt-Roth syndome, meralgia paresthetica, derived from the Creek word meros, meaning thigh, and algo, meaning pain – nerve entrapment or compressin where it passes between the upper front hipone (ilium) and the inguinal ligament.
Clinical signs:
- paresthesia or burning pain, numbness in the lateral and anterolateral thigh;
- symptoms worsen during walking and standing.


Principles of treatment of the peripheral nervous system pathology
Corticosteroid.
Drugs improving microcirculation.
Acetylcholinesterase inhibitors.
Analgetics.
Drugs are lipoic acid diyretics.
Gabapentin.
Pregabalin.
Vitamin B grup.
Physitherapy: massage, local anasthetic blocada.

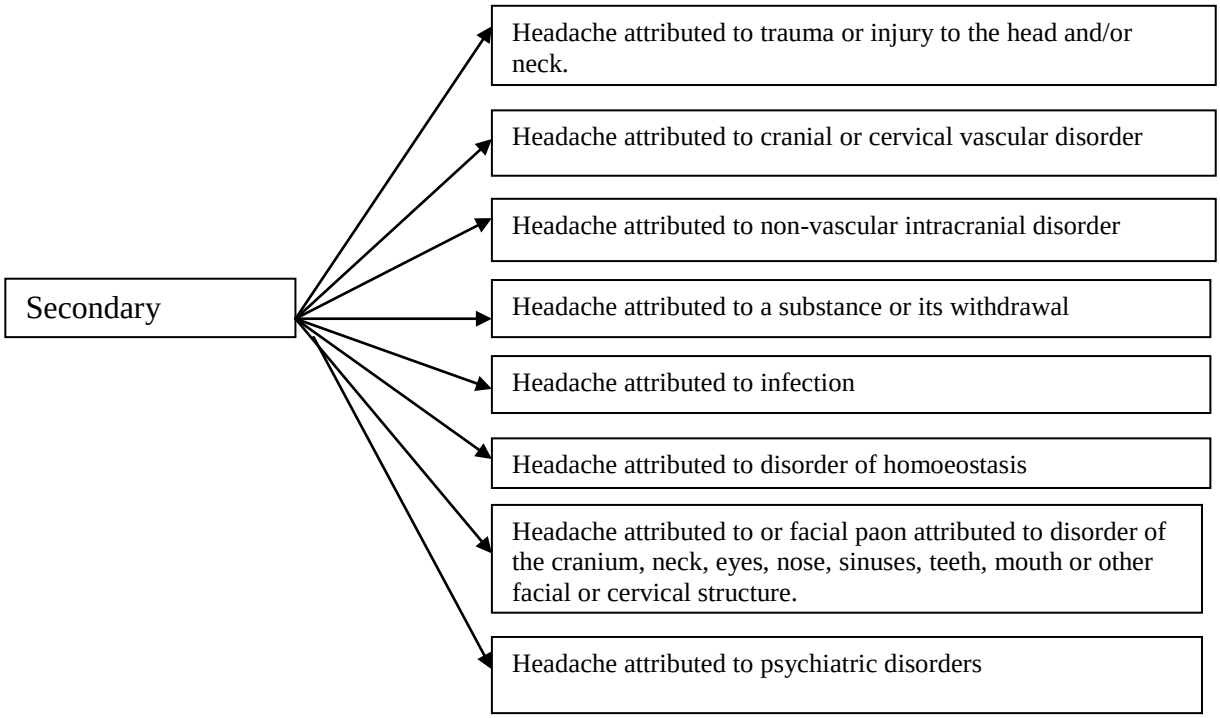
THEMA: HEADACHE. CLASSIFICATION HEADACHE. MIGRAINE.

Classification of headache

1.



2.



3. Painful cranial neuropathies and other facial pains.

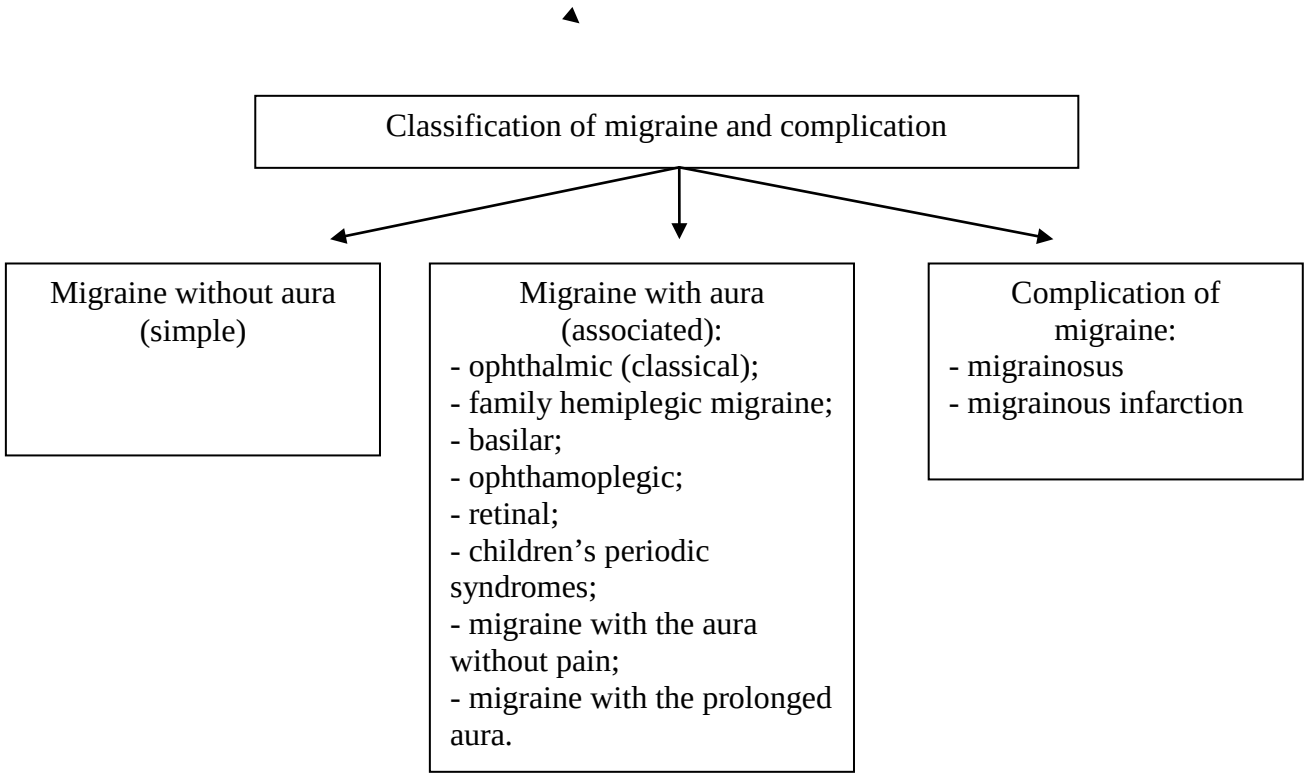
4. Other headache disorders.

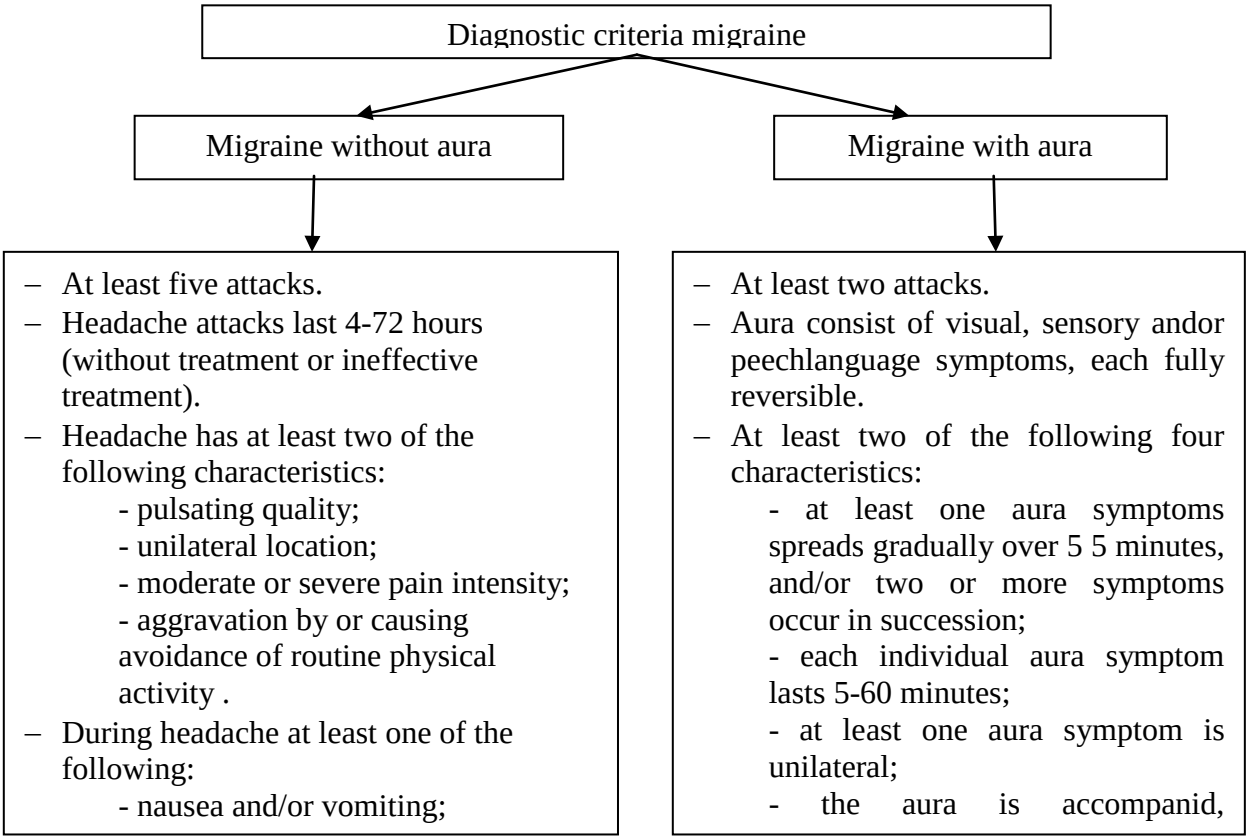
Migraine

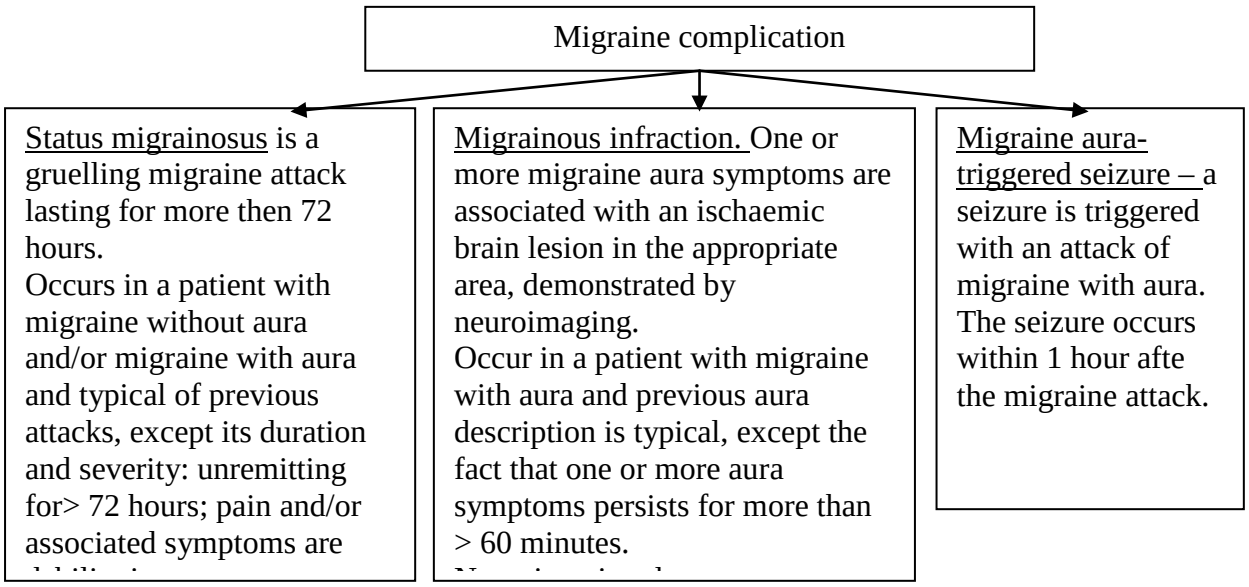
Migraine is common often familial disorder characterized by unilateral throbbing headache.

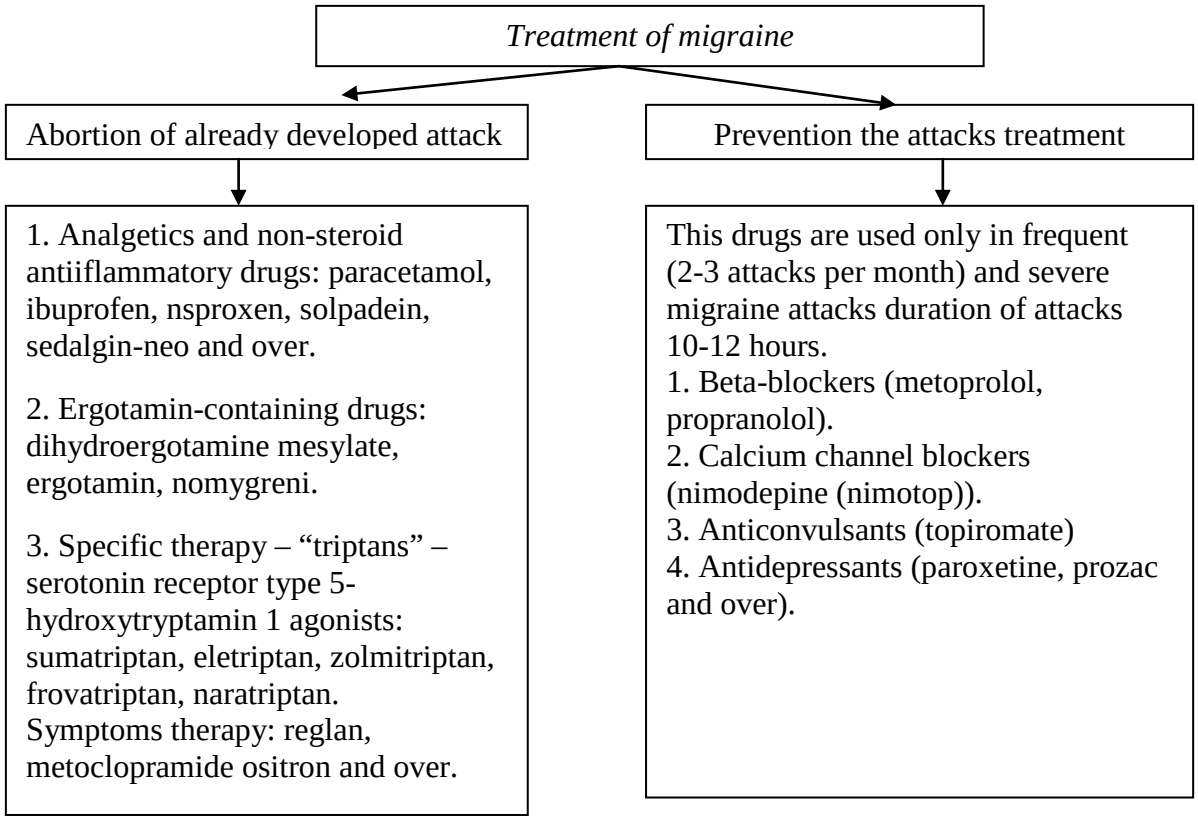
Mechanism

Mutation in mitochondrial DNA and Ca^{2+} channel genes may explain familial cases. Vascular and neuronal process probably co-exist with changes in serotonin activity attacks.




Diagnostic criteria migraine
Migraine without aura
Migraine with aura
– At least five attacks.
– Headache attacks last 4-72 hours (without treatment or ineffective treatment).
– Headache has at least two of the following characteristics:
- pulsating quality;
- unilateral location;
- moderate or severe pain intensity;
- aggravation by or causing avoidance of routine physical activity .
– During headache at least one of the following:
- nausea and/or vomiting;
– At least two attacks.
– Aura consist of visual, sensory andor peechlanguage symptoms, each fully reversible.
– At least two of the following four characteristics:
- at least one aura symptoms spreads gradually over 5 5 minutes, and/or two or more symptoms occur in succession;
- each individual aura symptom lasts 5-60 minutes;
- at least one aura symptom is unilateral;
- the aura is accompanid,


Migraine complication
Status migrainosus is a gruelling migraine attack lasting for more then 72 hours.
Occurs in a patient with migraine without aura and/or migraine with aura and typical of previous attacks, except its duration and severity: unremitting for> 72 hours; pain and/or associated symptoms are
Migrainous infraction. One or more migraine aura symptoms are associated with an ischaemic brain lesion in the appropriate area, demonstrated by neuroimaging.
Occur in a patient with migraine with aura and previous aura description is typical, except the fact that one or more aura symptoms persists for more than > 60 minutes.
Migraine aura-triggered seizure – a seizure is triggered with an attack of migraine with aura. The seizure occurs within 1 hour afte the migraine attack.



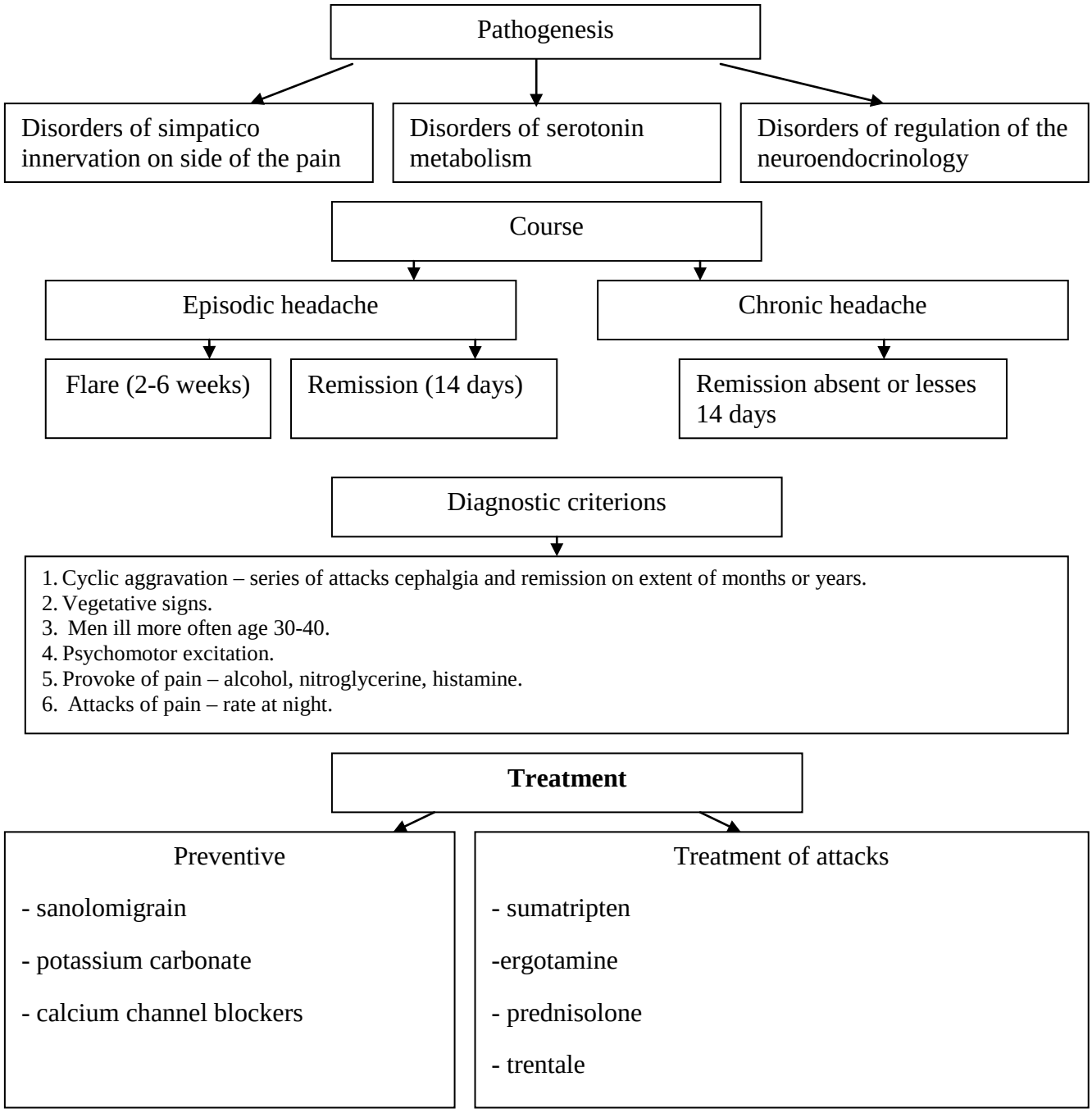
Status migrainous. Severe paroxysmal migraine or a continuous series of maigraine attacks lasting more then three days.

Clinical signs. In diffuse intense cephalgia, more than 3 days, does not regress after sleep. General weakness adynamia, pale skin, vomiting, photophobia, hyperacusia. Possible spasm and meningeal sundrome, general cerebral signs, change of consciousness transient visual diorders. Status migraine can threat the development of stroke (lacunar stroke).

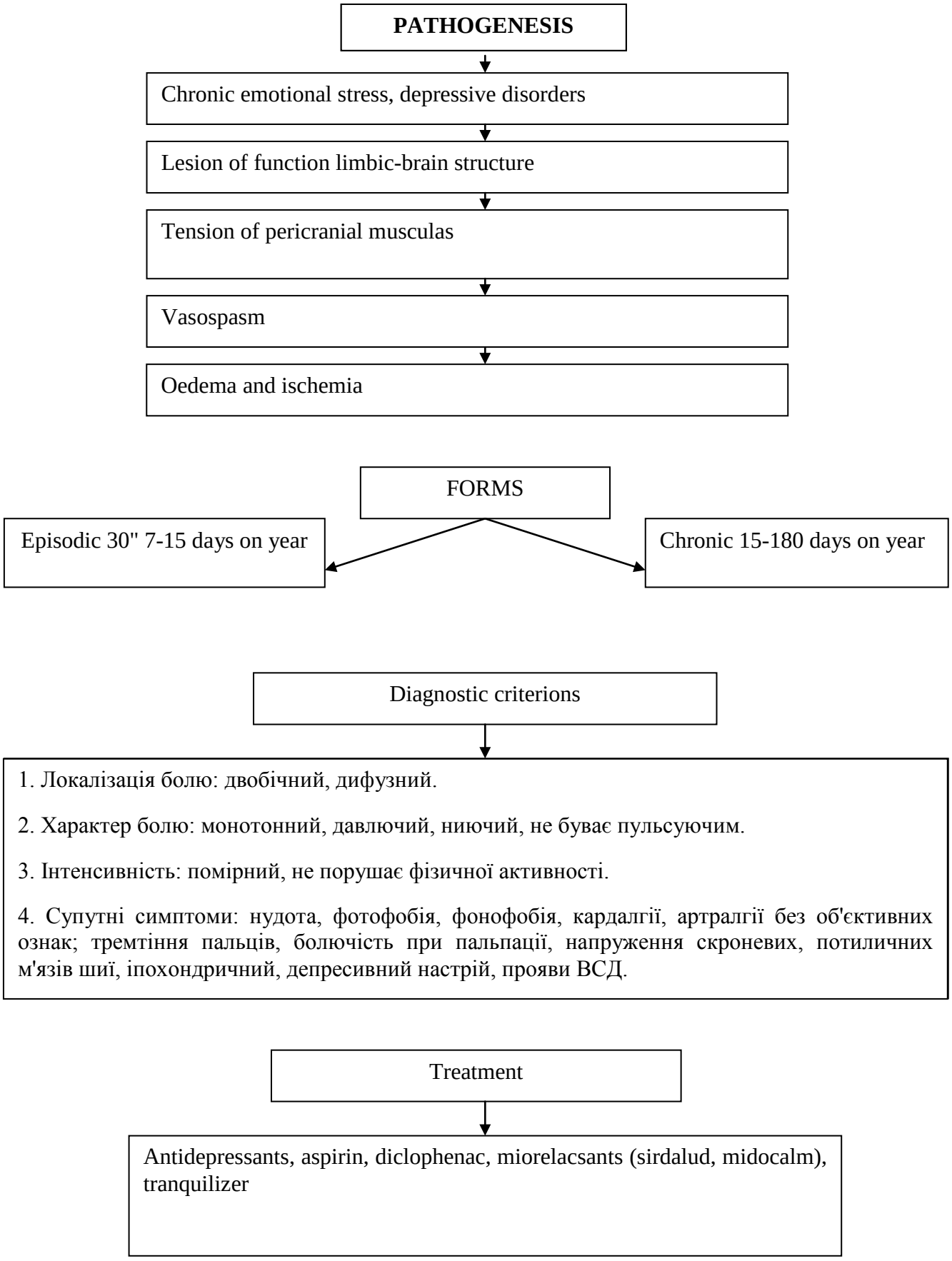
Diagnostic: neurology status, ophtalmoscopia, CT scan, MPI of brain, CSF.

Differential diagnosis: corticosteroids (prednisolone, dexametasone), ergotamine drugs, antidepressants, tranqullizers and over.

CLUSTER HEADACHE



HEADACHE OF TENSION



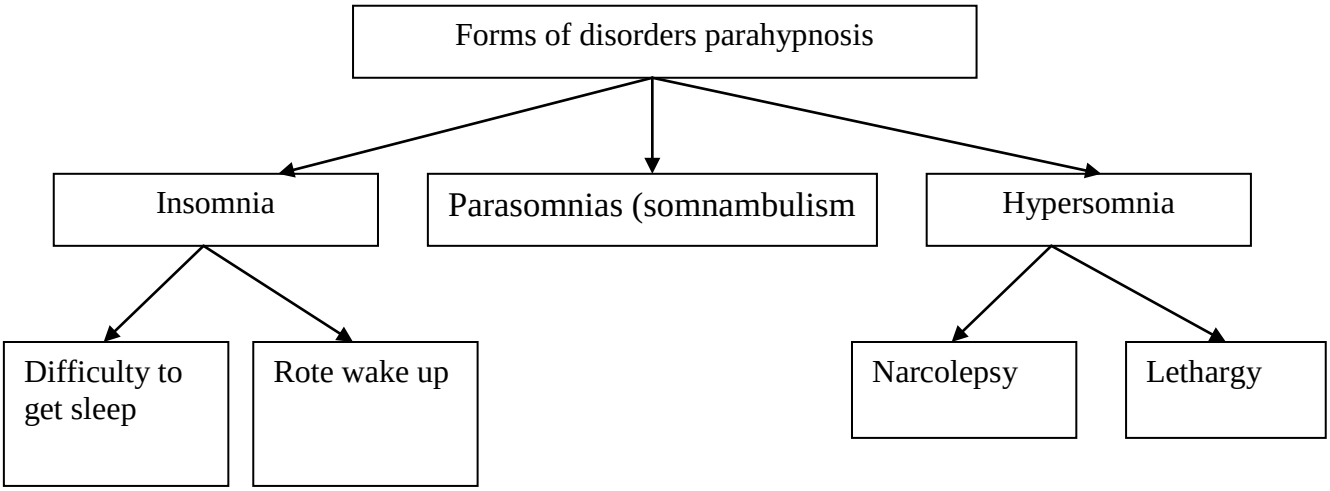
Ubusuna headache

Ubusuna headache – drug headache, one the secondaru forms of headache associated with migraine. This headache manifested by bilateral, pressing or constricting naturemof maderate intensity.

Pain when patients abuse pain medications (at least 15 days per month for 3 month or more) worries from 15 days or more up to daily.

The basis of the busal headache is the presence of migraine. Abusson pain often causes analgetics, NSAIDs, ergotamine drugs, tritan, and opioids.

FORMS OF PARAHYPNOSIS (DISOMNIYA)



The syndrome of intracranial hypotension

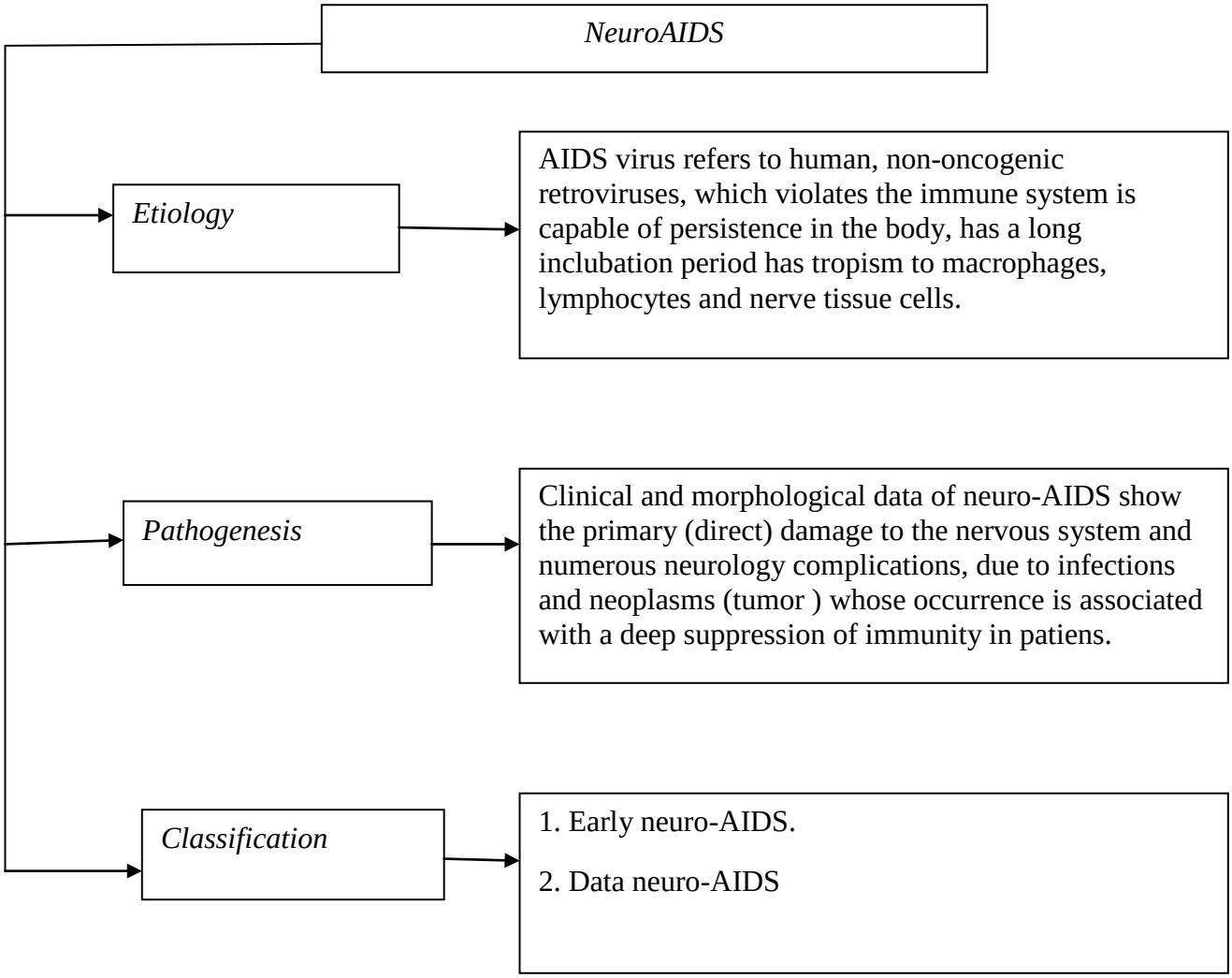
Etiology, pathogenetis factors	Therapeutic and diagnostic intervention on the liquor system,liquory fistula with likoria. Violation of water-salt metabolism (frequent vomiting, diarrhea, forced diuresis). The decreased production of cerebrospinal fluid (after a traumatic brain injury, due to autonomic dysregulation, vascular sclerosis of choroidal plexus blood. Arterial hypotesia.
Subjective data.	Headache, often compressive character. The desire to lower your head down. Nausea or vomiting. Gneral weakness.
Clinical and istrumental data research methods.	Meningeal symptoms (sometimes)/ Sparing the head position. The reduced pressure at lumbar puncture. Strengthening of all symptoms in a vertical position and a decrease in lying, while lowering the head.

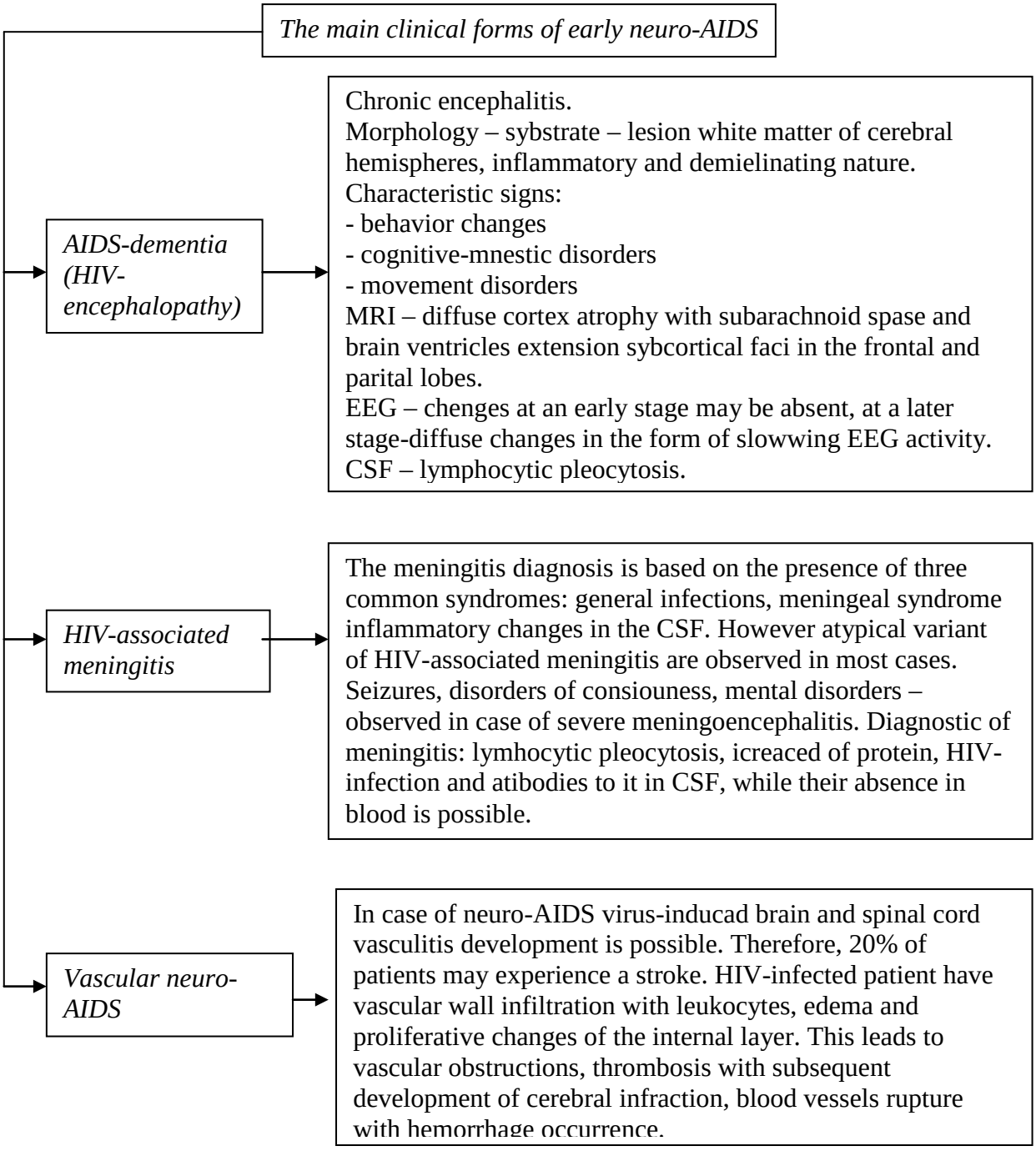
The syndrome of intracranial hypertension

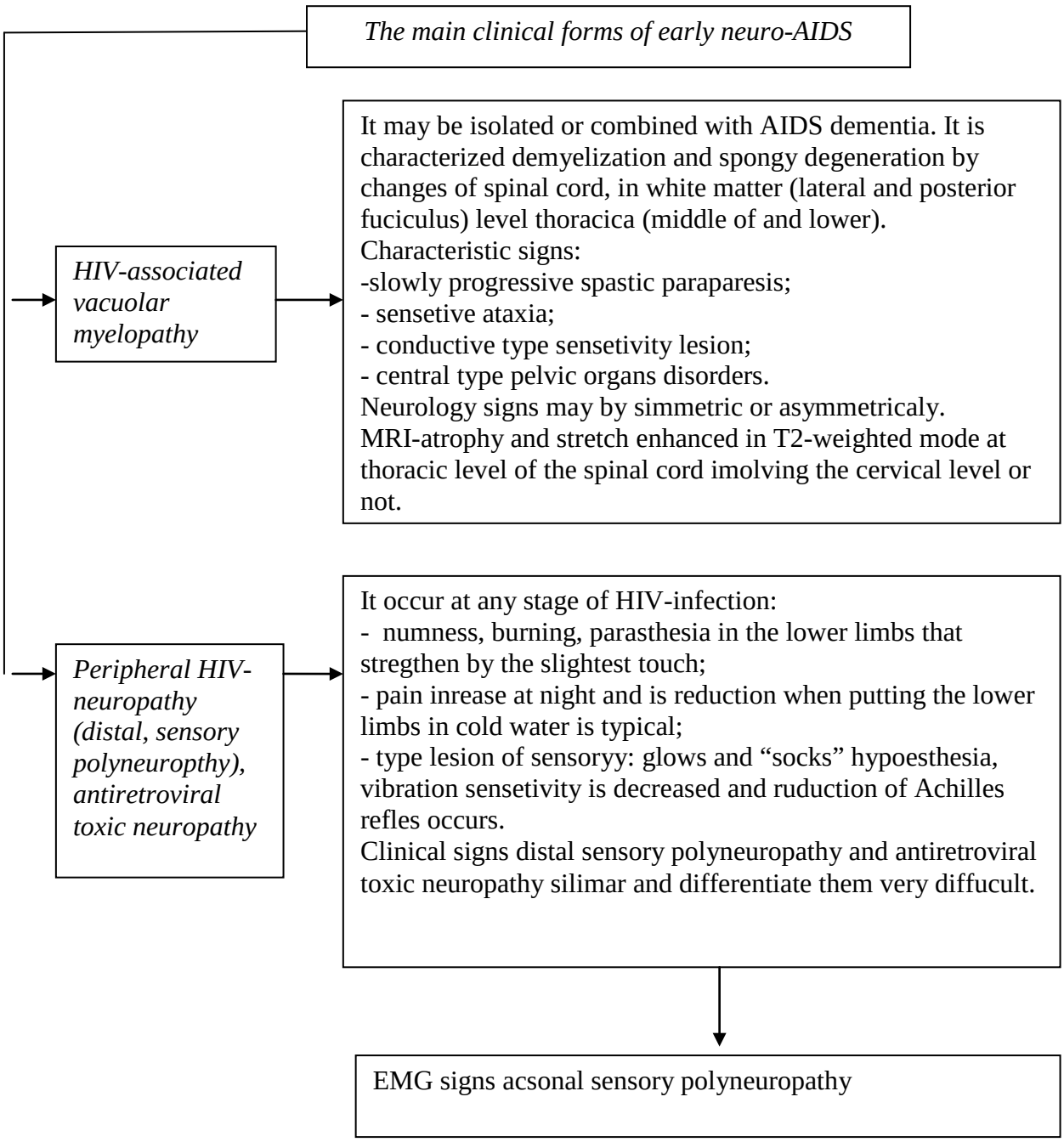
Etiology . Pathogenesis	The reduction in intracranial spase (hemotoma, abscesses and over tumor)	Reactive of brain edema	Complication of venous outflow	Increace in production of liquor	Difficulty in outflow of liquor from the ventricular system of tne brain (occlusive hydrocephalia)
Subjective data	Headache (expander nature) of the pain whwn moving eyeballs.	Vomiting, nausea		Dizziness (not a permanent syndrome)	
Clinical data	Lesion of granial nerves (more often than VI pair cranial nerve.	Change of pulse, breathing and other visceral vegetative disorders.		Disorders of consciousness with severe hypertension (inhibition sopor, come).	
Data of instrumental research methods	The expansion of the ventricular complex tj the EchoEg and CT-scan	The pressure increase in luncture. Protein-cellular dissociation in CSF.		Change of X--Ray of skill: - increased digital impressions; - Turkish saddle; - increased vascular pattern; - rashotte joints in children.	Stagnant discs of the optic nerves (ophthalmoscopy).

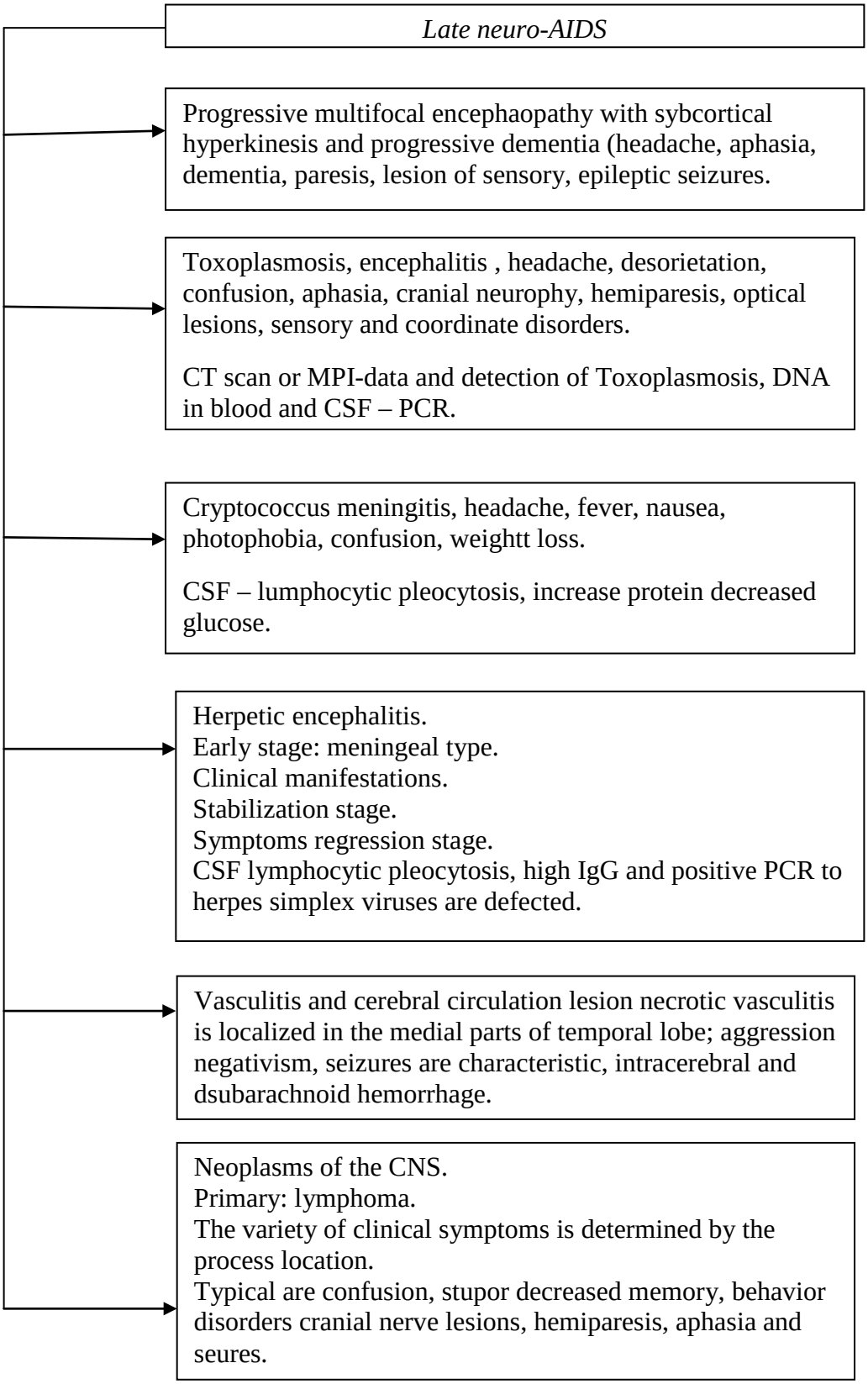
THEMA: NEUROLOGICAL ASPECTS OF ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS)

AIDS – the final stage of infection, which is human caused by the immunodeficiency virus (HIV) and occurs with the defeat of all organs and human systems; already in the early stage affected by the central nervous system and peripheral nervous system.




The main clinical forms of early neuro-AIDS
AIDS-dementia (HIV-encephalopathy)
Chronic encephalitis.
Morphology – sybstrate – lesion white matter of cerebral hemispheres, inflammatory and demielinating nature.
Characteristic signs:
- behavior changes
- cognitive-mnestic disorders
- movement disorders
MRI – diffuse cortex atrophy with subarachnoid spase and brain ventricles extension sybcortical faci in the frontal and parital lobes.
EEG – chenges at an early stage may be absent, at a later stage-diffuse changes in the form of slowwing EEG activity.
CSF – lymphocytic pleocytosis.
HIV-associated meningitis
The meningitis diagnosis is based on the presence of three common syndromes: general infections, meningeal syndrome inflammatory changes in the CSF. However atypical variant of HIV-associated meningitis are observed in most cases. Seizures, disorders of consiouness, mental disorders – observed in case of severe meningoencephalitis. Diagnostic of meningitis: lymhocytic pleocytosis, icreaced of protein, HIV-infection and atibodies to it in CSF, while their absence in blood is possible.
Vascular neuro-AIDS
In case of neuro-AIDS virus-inducad brain and spinal cord vasculitis development is possible. Therefore, 20% of patients may experience a stroke. HIV-infected patient have vascular wall infiltration with leukocytes, edema and proliferative changes of the internal layer. This leads to vascular obstructions, thrombosis with subsequent development of cerebral infraction, blood vessels rupture with hemorrhage occurrence.


The main clinical forms of early neuro-AIDS
HIV-associated vacuolar myelopathy
It may be isolated or combined with AIDS dementia. It is characterized demyelization and spongy degeneration by changes of spinal cord, in white matter (lateral and posterior fuciculus) level thoracica (middle of and lower).
Characteristic signs:
-slowly progressive spastic paraparesis;
- sensetive ataxia;
- conductive type sensetivity lesion;
- central type pelvic organs disorders.
Neurology signs may by simmetric or asymmetricaly.
MRI-atrophy and stretch enhanced in T2-weighted mode at thoracic level of the spinal cord imolving the cervical level or not.
Peripheral HIV-neuropathy (distal, sensory polyneuropthy), antiretroviral toxic neuropathy
It occur at any stage of HIV-infection:
- numness, burning, parasthesia in the lower limbs that stregthen by the slightest touch;
- pain inrease at night and is reduction when putting the lower limbs in cold water is typical;
- type lesion of sensoryy: glows and “socks” hypoesthesia, vibration sensetivity is decreased and ruduction of Achilles refles occurs.
Clinical signs distal sensory polyneuropathy and antiretroviral toxic neuropathy silimar and differentiate them very diffucult.
EMG signs acsonal sensory polyneuropathy

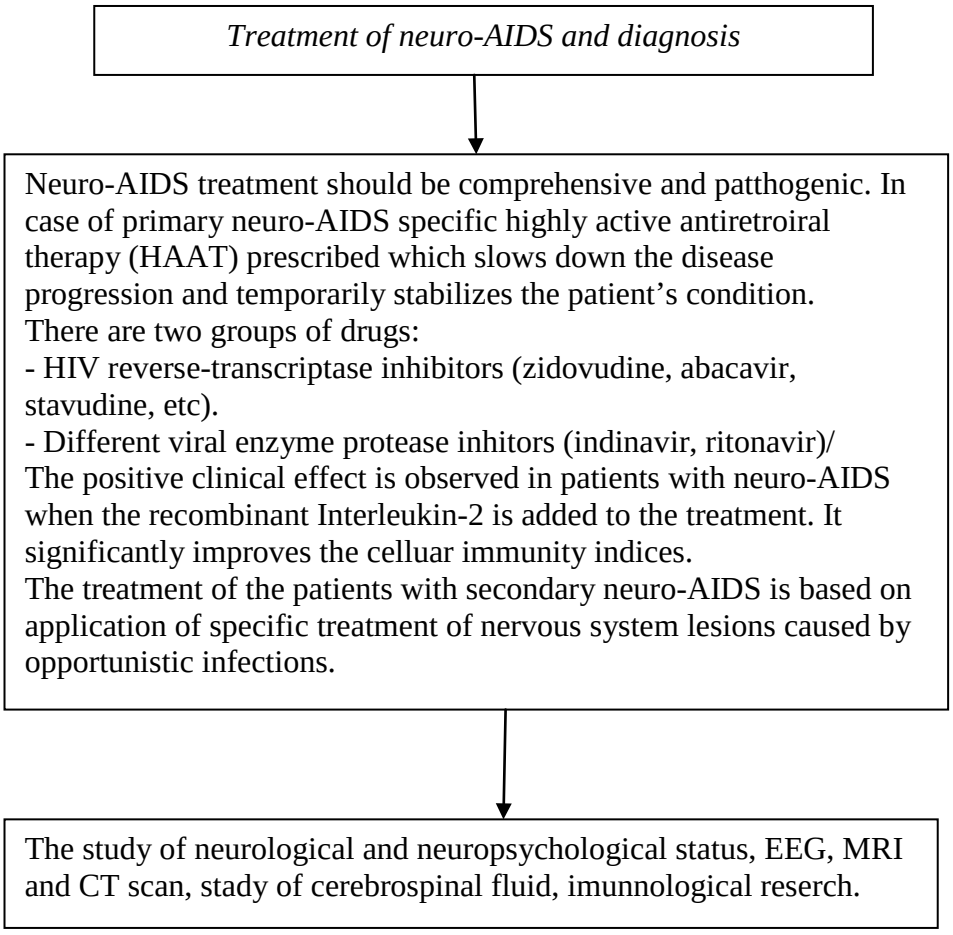

Late neuro-AIDS
Progressive multifocal encephaopathy with sybcortical hyperkinesis and progressive dementia (headache, aphasia, dementia, paresis, lesion of sensory, epileptic seizures.
Toxoplasmosis, encephalitis , headache, desorietation, confusion, aphasia, cranial neurophy, hemiparesis, optical lesions, sensory and coordinate disorders.
CT scan or MPI-data and detection of Toxoplasmosis, DNA in blood and CSF – PCR.
Cryptococcus meningitis, headache, fever, nausea, photophobia, confusion, weightt loss.
CSF – lumphocytic pleocytosis, increase protein decreased glucose.
Herpetic encephalitis.
Early stage: meningeal type.
Clinical manifestations.
Stabilization stage.
Symptoms regression stage.
CSF lymphocytic pleocytosis, high IgG and positive PCR to herpes simplex viruses are defected.
Vasculitis and cerebral circulation lesion necrotic vasculitis is localized in the medial parts of temporal lobe; aggression negativism, seizures are characteristic, intracerebral and dsubarachnoid hemorrhage.
Neoplasms of the CNS.
Primary: lymphoma.
The variety of clinical symptoms is determined by the process location.
Typical are confusion, stupor decreased memory, behavior disorders cranial nerve lesions, hemiparesis, aphasia and seures.

Clinical HIV infection in children

HIV in early childhood contributes to the physical and psychomotor development. Recurrent bacterial infections are marked more often in children than in adults; lymphoid pneumonitis, pulmonary lymph nodes increase, encephalopathy and anemia are also common.

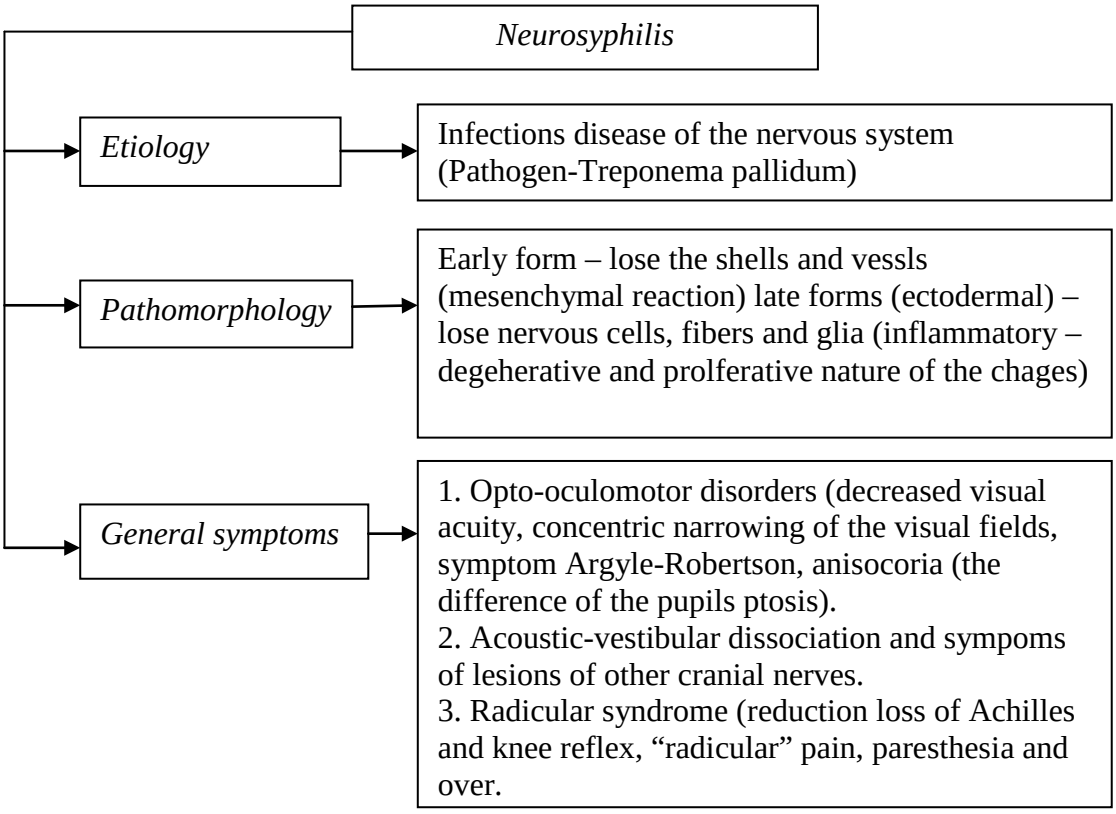
A common cause of infant mortality in case of HIV infection is hemorrhagic syndrome as a result of severe thrombocytopenia.

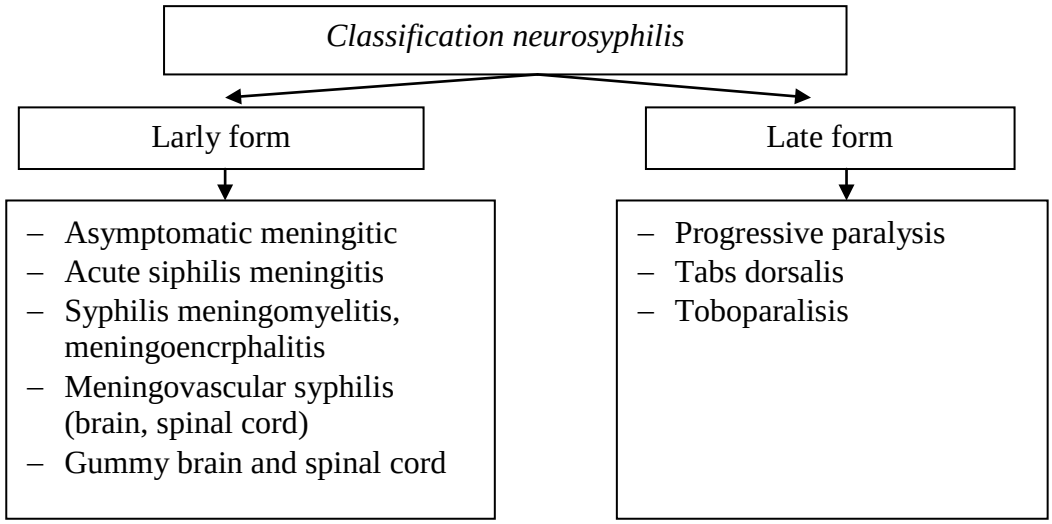
The disease in children who have get HIV from their mothers during pregnancy or in the perinatal perid proceeds considerably more difficult and rapidly progressive than in children infected after year of life.

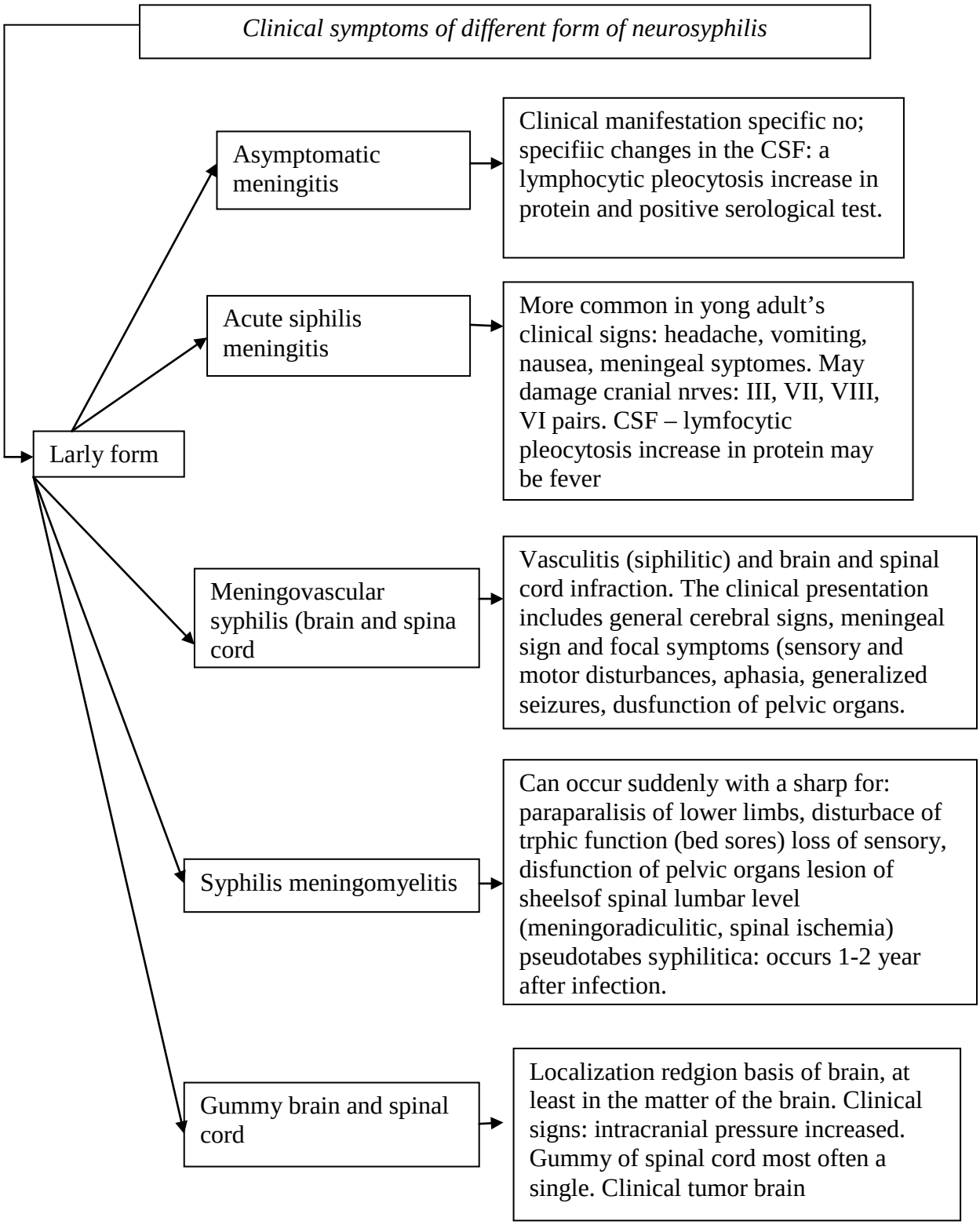


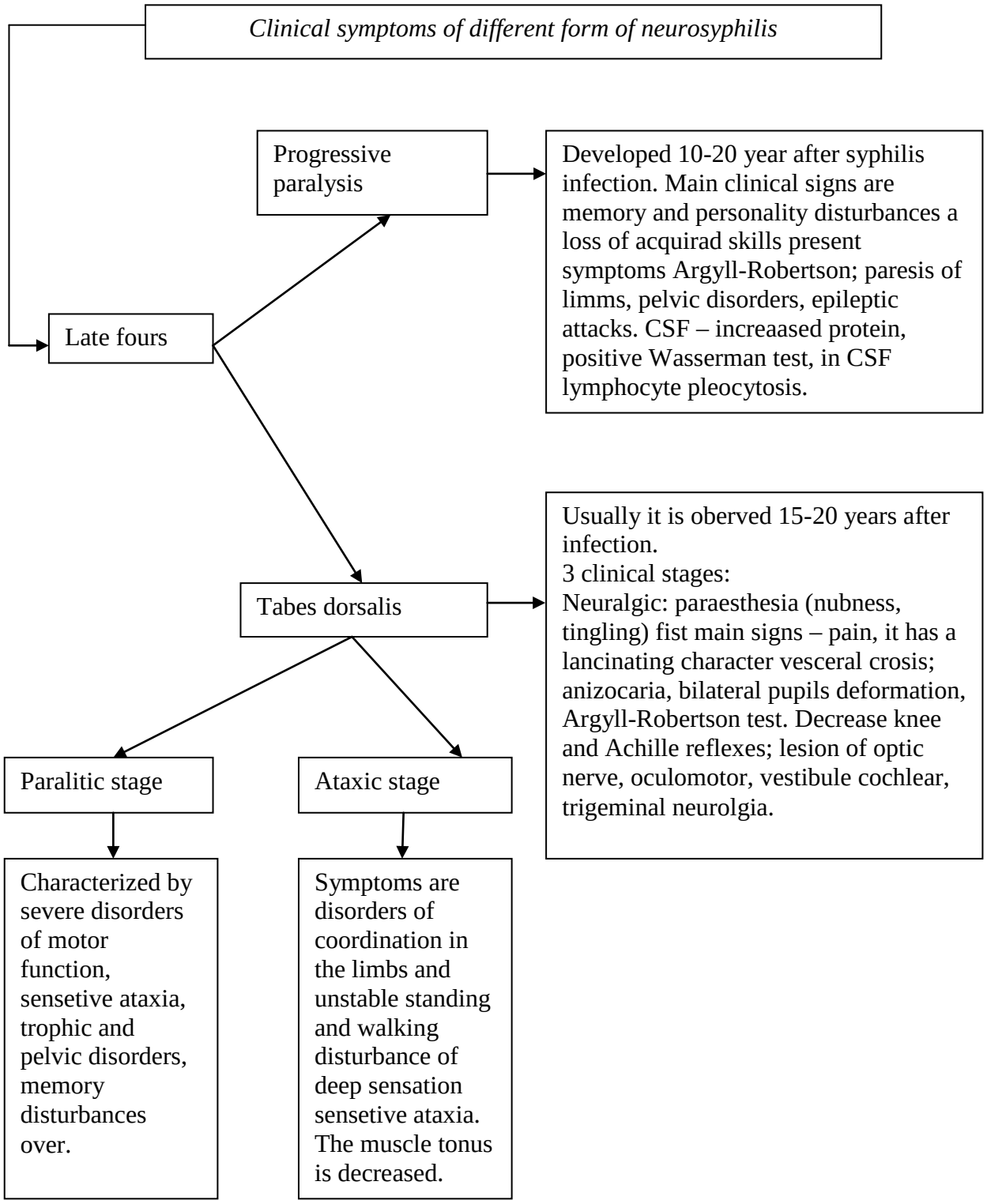
THEMA: NEUROSYPHILIS

Syphilis is caused by the motile spirochaete Treponema pallidum. The natural history of untreated infection is divided into three stages. Neurological involvement occurs in the third stage, which is typecally many years after the initial infection. Neurosyphilis occurs in less than 10% of all untreated cases. Penicillinsare widely used for the treatment of other infections and thus many unsuspected cases of syphilis are treated without progressing to stages two and three.






Clinical symptoms of different form of neurosyphilis
Larly form
Asymptomatic meningitis
Clinical manifestation specific no; specifiic changes in the CSF: a lymphocytic pleocytosis increase in protein and positive serological test.
Acute siphilis meningitis
More common in yong adult's clinical signs: headache, vomiting, nausea, meningeal syptomes. May damage cranial nrves: III, VII, VIII, VI pairs. CSF – lymfocytic pleocytosis increase in protein may be fever
Meningovascular syphilis (brain and spina cord
Vasculitis (siphilitic) and brain and spinal cord infraction. The clinical presentation includes general cerebral signs, meningeal sign and focal symptoms (sensory and motor disturbances, aphasia, generalized seizures, dusfunction of pelvic organs.
Syphilis meningomyelitis
Can occur suddenly with a sharp for: paraparalisis of lower limbs, disturbace of trphic function (bed sores) loss of sensory, disfunction of pelvic organs lesion of sheelsof spinal lumbar level (meningoradiculitic, spinal ischemia) pseudotabes syphilitica: occurs 1-2 year after infection.
Gummy brain and spinal cord
Localization redgion basis of brain, at least in the matter of the brain. Clinical signs: intracranial pressure increased. Gummy of spinal cord most often a single. Clinical tumor brain


Clinical symptoms of different form of neurosyphilis
Progressive paralysis
Developed 10-20 year after syphilis infection. Main clinical signs are memory and personality disturbances a loss of acquirad skills present symptoms Argyll-Robertson; paresis of limms, pelvic disorders, epileptic attacks. CSF – increaased protein, positive Wasserman test, in CSF lymphocyte pleocytosis.
Late fours
Tabes dorsalis
Usually it is oberved 15-20 years after infection.
3 clinical stages:
Neuralgic: paraesthesia (nubness, tingling) fist main signs – pain, it has a lancinating character vesceral crosis; anizocaria, bilateral pupils deformation, Argyll-Robertson test. Decrease knee and Achille reflexes; lesion of optic nerve, oculomotor, vestibule cochlear, trigeminal neurolgia.
Paralitic stage
Ataxic stage
Characterized by severe disorders of motor function, sensetive ataxia, trophic and pelvic disorders, memory disturbances over.
Symptoms are disorders of coordination in the limbs and unstable standing and walking disturbance of deep sensation sensetive ataxia. The muscle tonus is decreased.

Taboparalisis

Taboparalisis – combinztion of neurology symptoms of progressive psrslysis and neurology symptoms of tabes dorsalis.

Diagnostic of neurosyphylis

- Wasseman's positive reaction in blood and CSF.
- Positive sorological reaction immobilization paltreponemes (RIPt).
- Positive reaction Lange of CSF.
- Lumphocytic pleocytosis and protein (meningeal form).
- CT, MRI, ophtalmology.

Differential diagnosis

- Meningitis not syphilis etioligy
- Progressing disturbances of cerebral blood (vascular syphilis)
- Tumor brain (gumma brain)
- Myelitis and spinal form amyotrophic lateral sclerosis

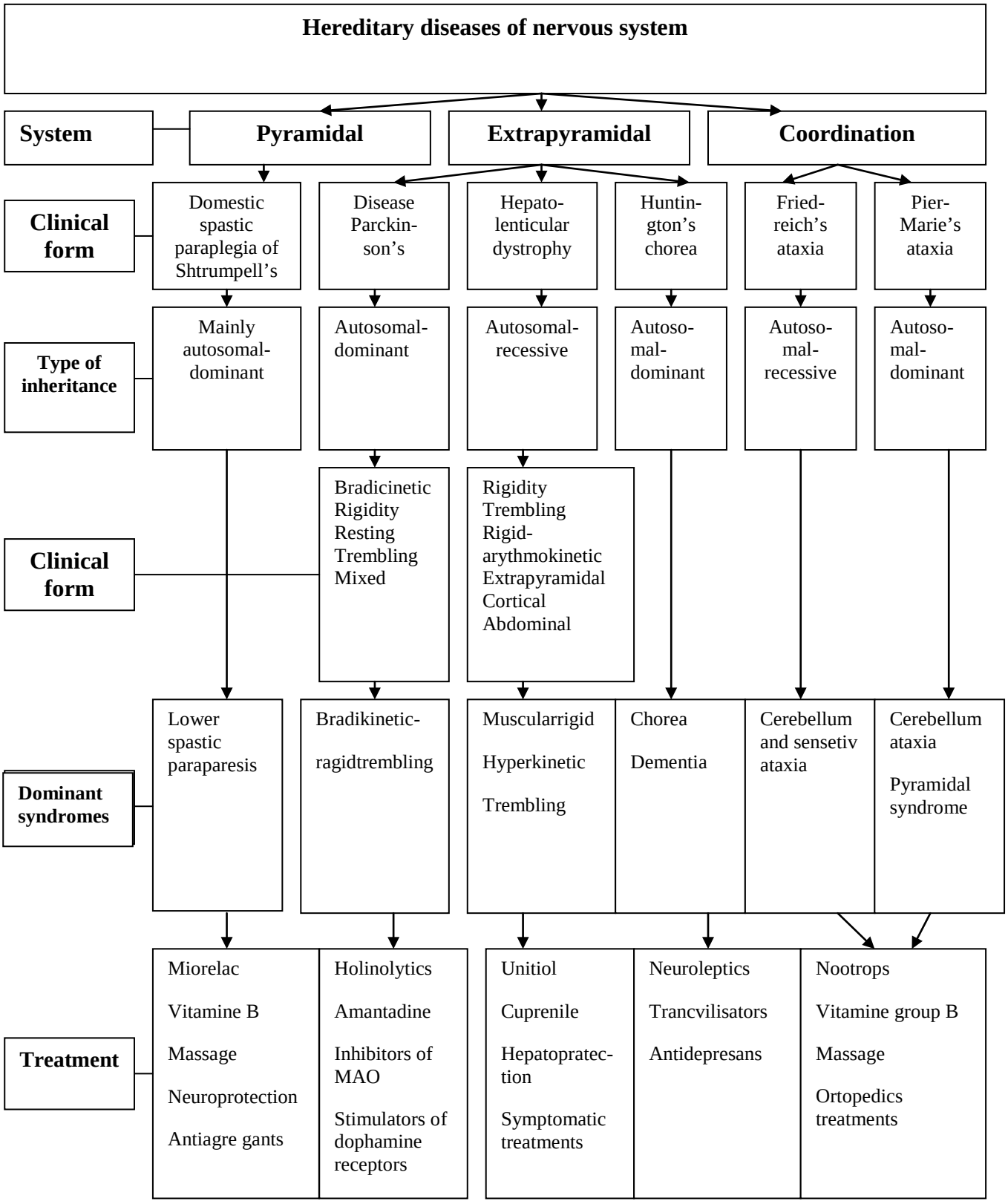
Treatment

- Drugs that improve hemodynamics: trental acidi nicotinici
- Vitamin (group B, C)
- Neuroprotection (piracetam, gliatilin, actovegin and over)
- Symptomatic therapy

Treatment of neurosyphilis

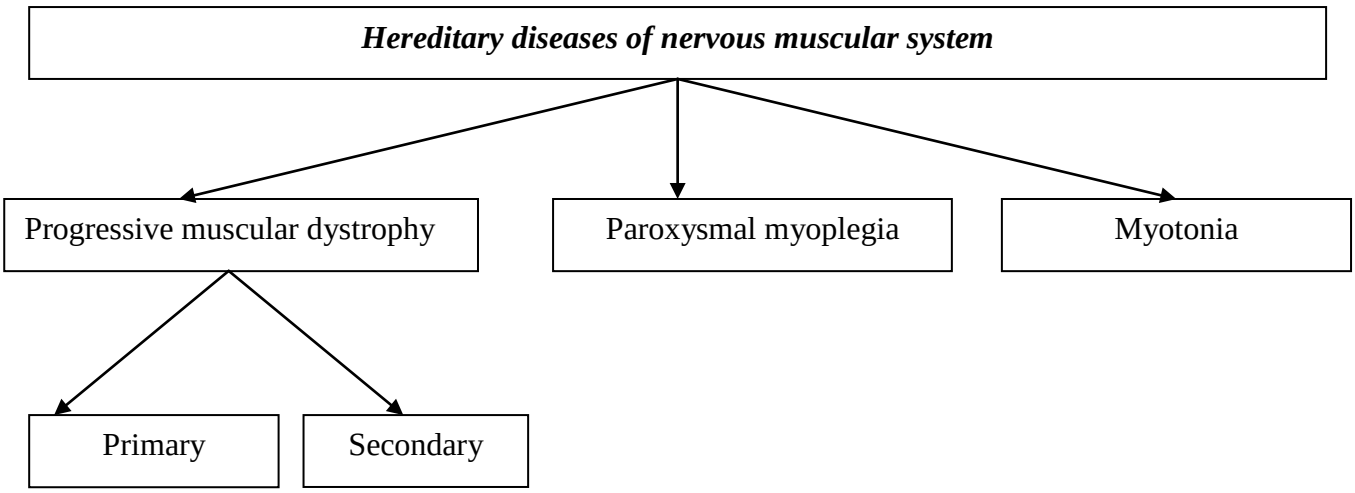
- Basic treatment of all forms
- Penicillin of 2000000-4000000 ED – 3 weeks.
- The effective of treatment is determined according to the blood tests and CSF examination. That's why lumbar puncture is made just after penicillin treatment and then every 3 months.
- Symptomatic treatment: trental, nicatinici, ascorbic acid, complex b vitemis, piracetami, physiotherapy.

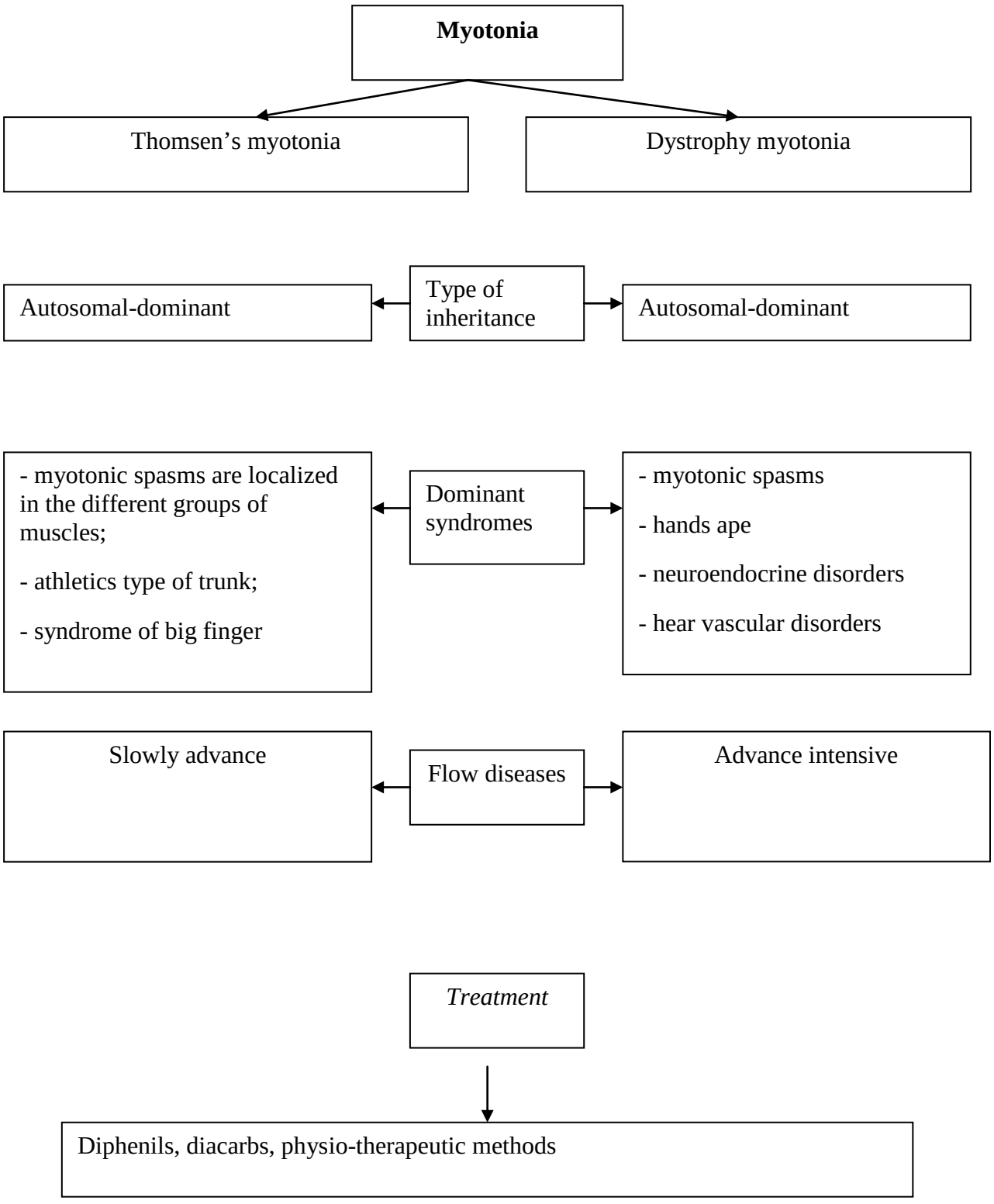
THEMA: HEREDITARY DISEASE OF NERVOUS SYSTEM

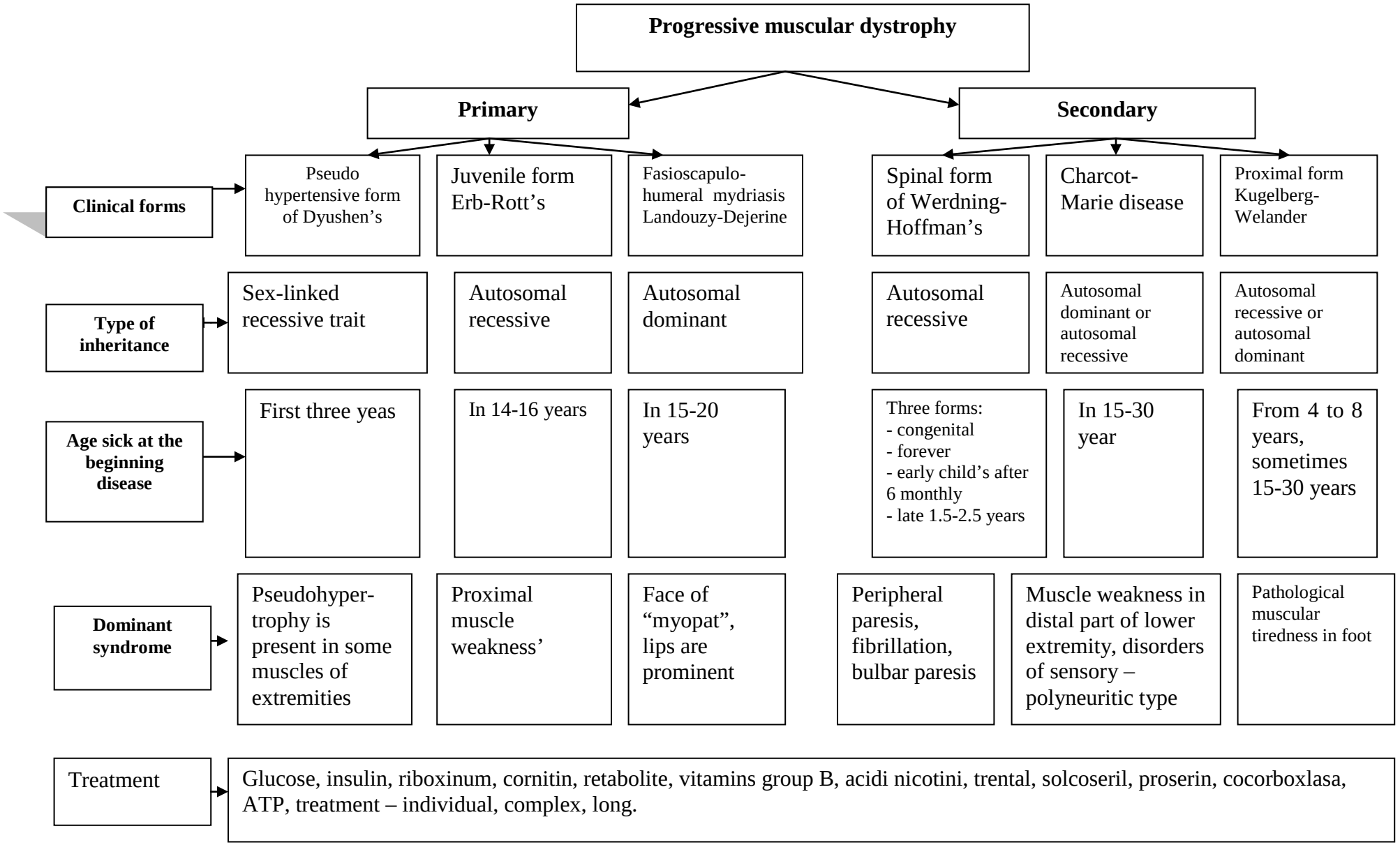


Differentially diagnostic criteria of ataxia of Friedraich's and Pier- Marie

Signs	**Ataxia of Friedraich's**	***Ataxia of Pier -Marie***
Type of heredity	Autosom-recession, very rarely - dominant	Autosomno-dominantniy
Age sick at the beginning of disease	6-15 years	20-40 years, middle – 34 years
Character of changes of reflexes	Lower	High
Presence of pyramid signs	Observed on the late stages of disease	Observed already on the early stages
Defeat of cranial nerves	Absent	Oculomotor disorders, declines lower of visual
Presence of sensitive ataxia	Observed already on the early stages	Absent
Deformations feet, spine	It is practical in all of cases	Not characteristic

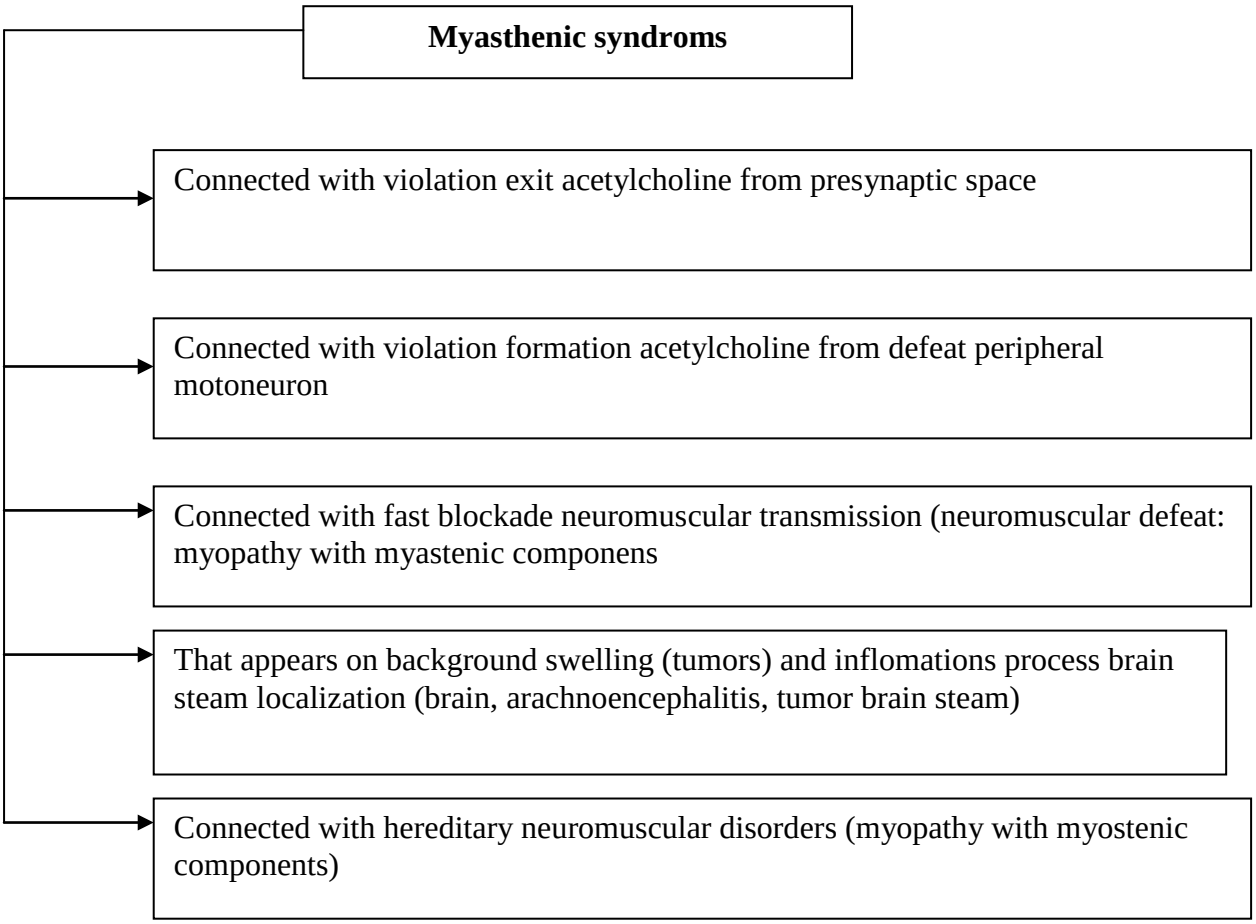



Myotonia
Thomsen's myotonia
Dystrophy myotonia
Autosomal-dominant
Type of inheritance
Autosomal-dominant
- myotonic spasms are localized in the different groups of muscles;
- athletics type of trunk;
- syndrome of big finger
Dominant syndromes
- myotonic spasms
- hands ape
- neuroendocrine disorders
- hear vascular disorders
Slowly advance
Flow diseases
Advance intensive
Treatment
Diphenils, diacarbs, physio-therapeutic methods


Progressive muscular dystrophy
Primary
Secondary
Clinical forms
Pseudo hypertensive form of Dyushen's
Juvenile form Erb-Rott's
Fasioscapulo-humeral mydriasis Landouzy-Dejerine
Spinal form of Werdning-Hoffman's
Charcot-Marie disease
Proximal form Kugelberg-Welander
Type of inheritance
Sex-linked recessive trait
Autosomal recessive
Autosomal dominant
Autosomal recessive
Autosomal dominant or autosomal recessive
Autosomal recessive or autosomal dominant
Age sick at the beginning disease
First three yeas
In 14-16 years
In 15-20 years
Three forms:
- congenital
- forever
- early child's after 6 monthly
- late 1.5-2.5 years
In 15-30 year
From 4 to 8 years, sometimes 15-30 years
Dominant syndrome
Pseudohyper-trophy is present in some muscles of extremities
Proximal muscle weakness'
Face of "myopat", lips are prominent
Peripheral paresis, fibrillation, bulbar paresis
Muscle weakness in distal part of lower extremity, disorders of sensory – polyneuritic type
Pathological muscular tiredness in foot
Treatment
Glucose, insulin, riboxinum, cornitin, retabolite, vitamins group B, acidi nicotini, trental, solcoseril, proserin, cocorboxlasa, ATP, treatment – individual, complex, long.

Myasthenia

Etiology	Autoimmune diseases, hyperplasia of the thymus
Clinical features	Muscles weakness, specific feature of this weakness is its increasing with oculomotorius disorders, weakness of mimic muscles. Generalized forms - heavy form, weakness of respiratory muscles.
Flow	Progressive with remission Myasthenic crisis
Diagnosis	Proserine test, phenomenon Uolker's, CT, EMG
Differential diagnosis	Guillain's-Barre syndrome, botulism, muscles dystrophy, stroke on tumor, encephalitis
Treatment	Compensation of neuro-muscular transference, thymus influence, correction of immune disorders, treatment conservative and surgical


Myasthenic syndroms
Connected with violation exit acetylcholine from presynaptic space
Connected with violation formation acetylcholine from defeat peripheral motoneuron
Connected with fast blockade neuromuscular transmission (neuromuscular defeat: myopathy with myastenic componens
That appears on background swelling (tumors) and inflomations process brain steam localization (brain, arachnoencephalitis, tumor brain steam)
Connected with hereditary neuromuscular disorders (myopathy with myostenic components)

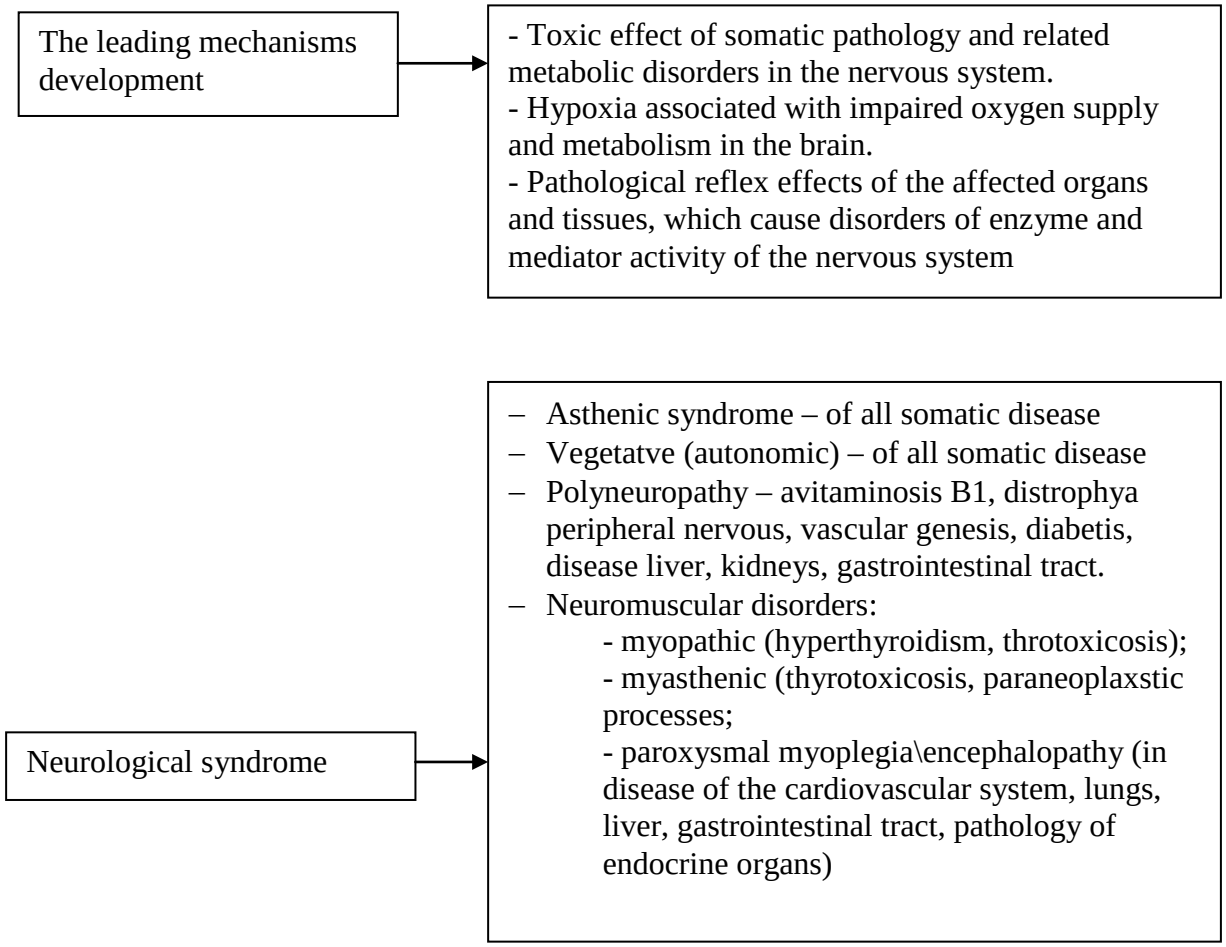
Paroxysmal myoplegia

Type of inheritance	Autosomal-dominant
Clinical forms	Classic paroxysmal myoplegia (hypokalemic, Vestphal's diseases); hyperkalemic (adynamia episodic a hereditary, Gamstrop's diseases)
Dominant syndromes	Weakness extremity and body, arise a surprise (in tame of sleep), reach general plegia deep and skin reflexes is automic defeat absent
Diagnosis	Exsame of electrolytes, wheys of blood are in the period of attack, electromyography.
Treatment	Gypokalemic form: to potassium chlorid, veroshpirons, diacarb's, diet (to limit the reception of carbonhydratess). Gyperkalemic form: calcium chloride, hydrochlorothiazide

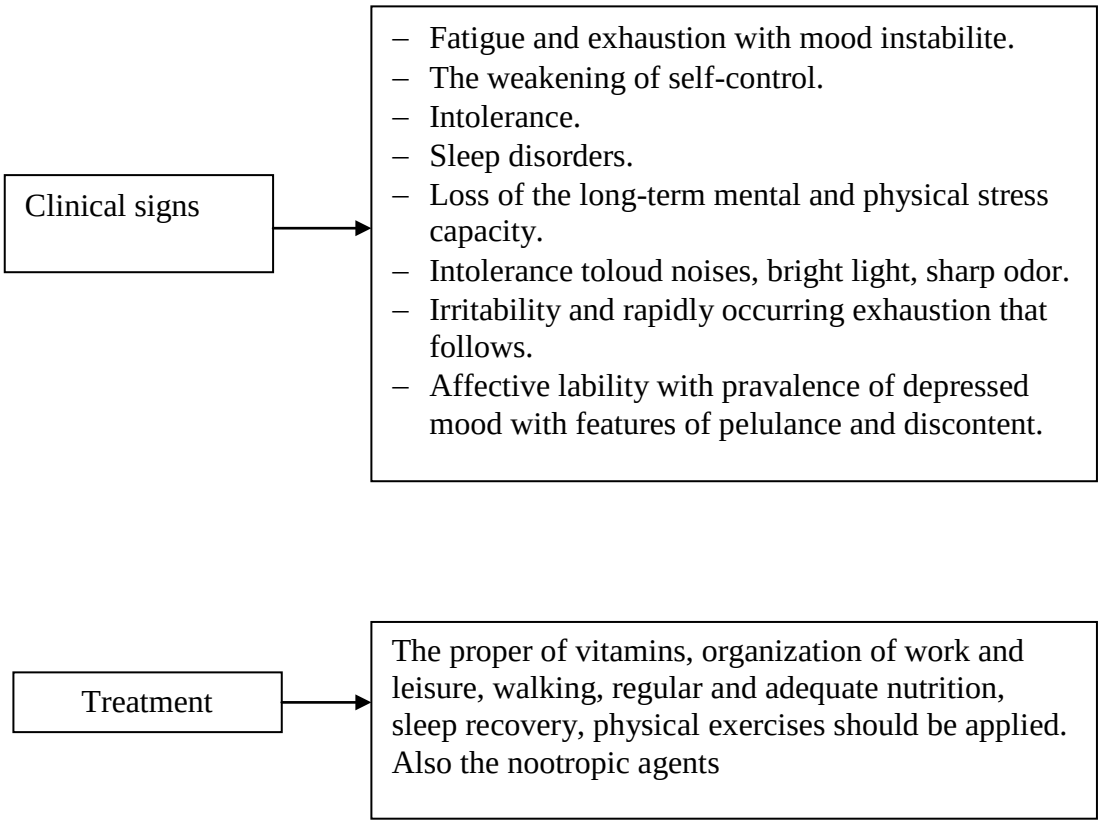
Muscular dystonia

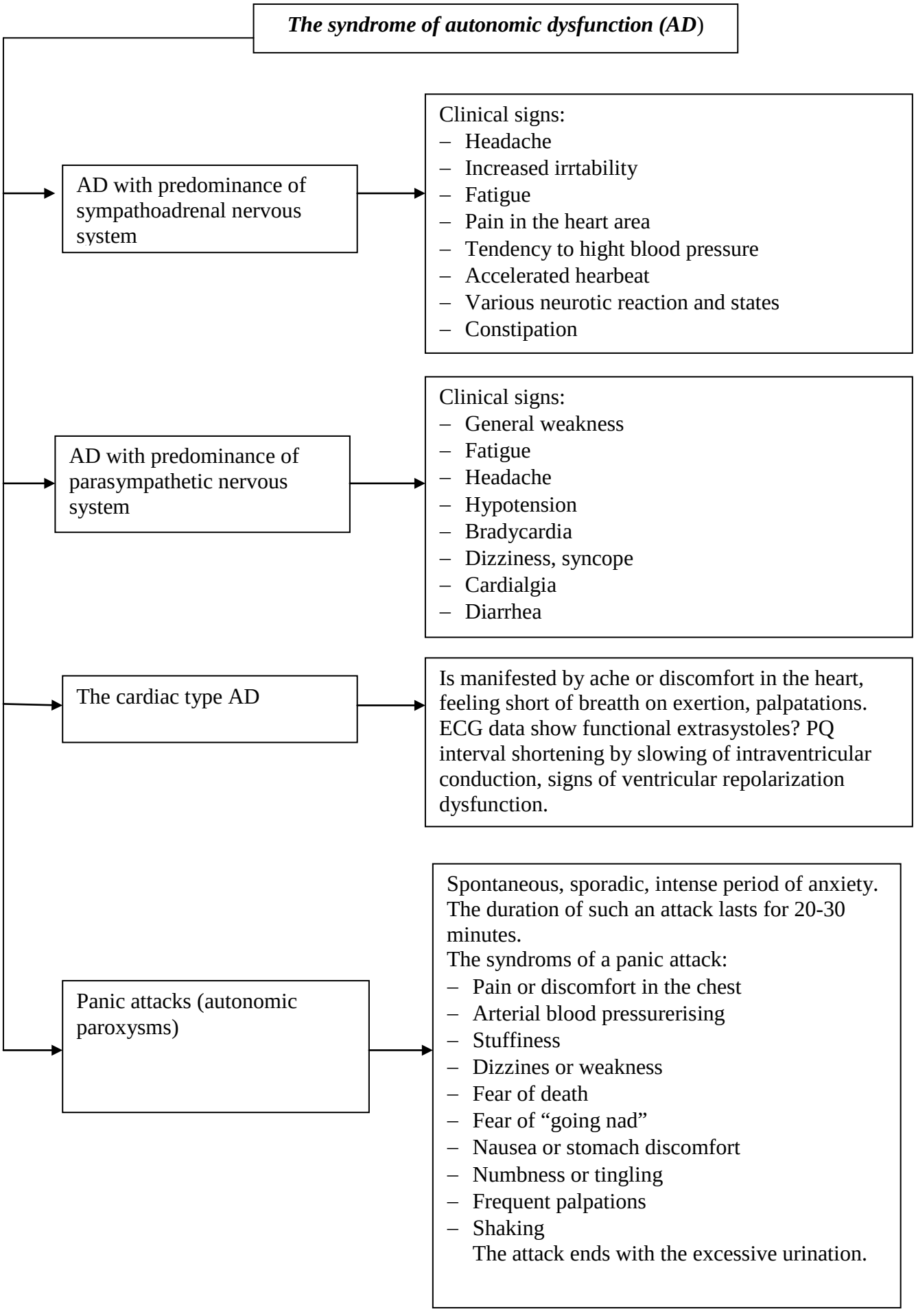
Focal dystonia Blepharospasm Mimic muscles spasm Spastic dystonia	Multifocal in two and more not adjacent (blepharospasm, dystonia foot's	Hemidystonia (for example, dystonia of foot and hand on one side)	Segmental dystonia in two segments adjacent (spasm winking, writers spasm rotatory)

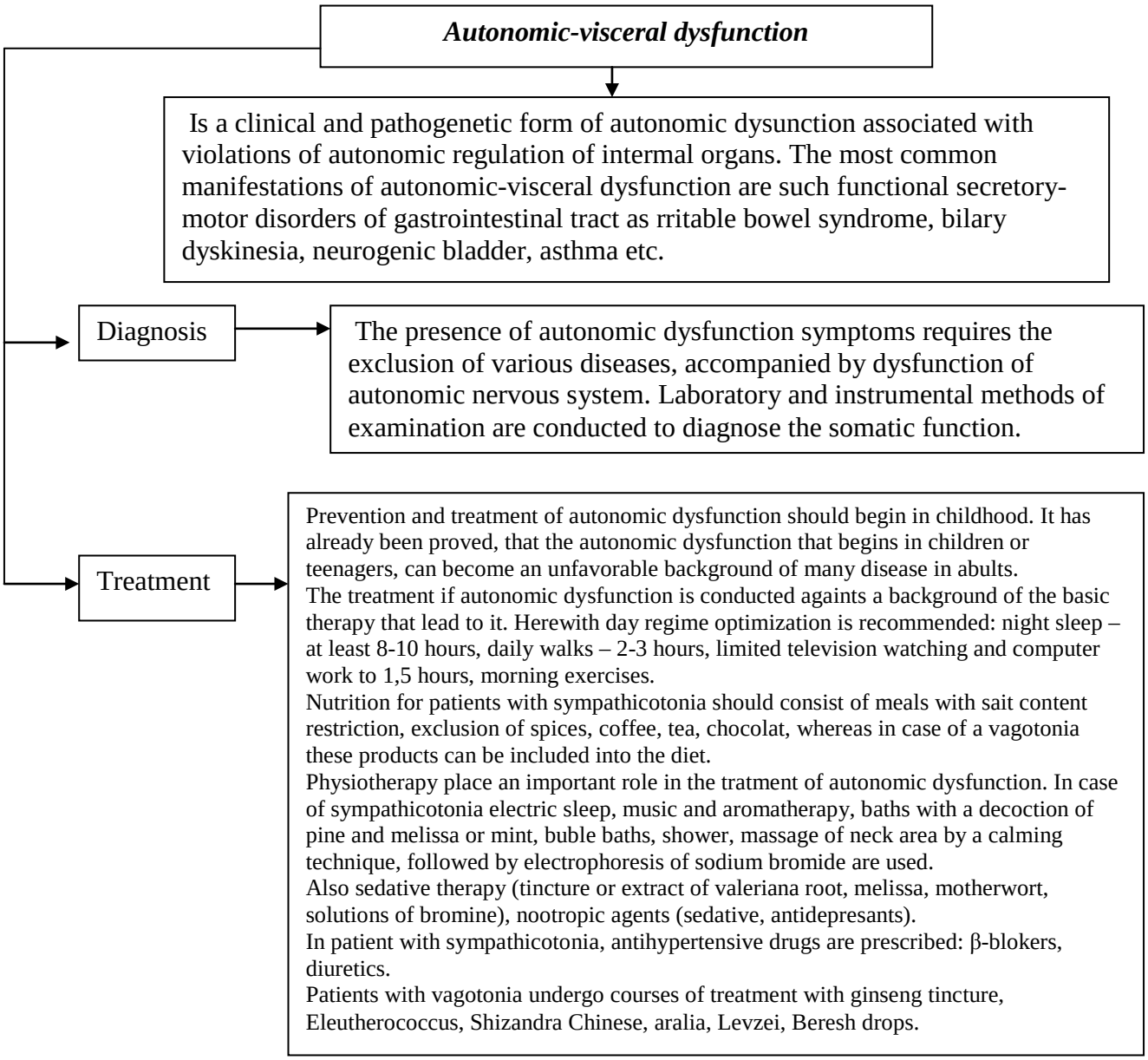
THEMA: SOMATONEUROLOGIC SYNDROME



Acthenic syndrome (neuro-psychlogical weakness)




The syndrome of autonomic dysfunction (AD)
AD with predominance of sympathoadrenal nervous system
Clinical signs:
– Headache
– Increased irrtability
– Fatigue
– Pain in the heart area
– Tendency to hight blood pressure
– Accelerated hearbeat
– Various neurotic reaction and states
– Constipation
AD with predominance of parasympathetic nervous system
Clinical signs:
– General weakness
– Fatigue
– Headache
– Hypotension
– Bradycardia
– Dizziness, syncope
– Cardialgia
– Diarrhea
The cardiac type AD
Is manifested by ache or discomfort in the heart, feeling short of breatth on exertion, palpatations. ECG data show functional extrasystoles? PQ interval shortening by slowing of intraventricular conduction, signs of ventricular repolarization dysfunction.
Panic attacks (autonomic paroxysms)
Spontaneous, sporadic, intense period of anxiety. The duration of such an attack lasts for 20-30 minutes.
The syndroms of a panic attack:
– Pain or discomfort in the chest
– Arterial blood pressurerising
– Stuffiness
– Dizzines or weakness
– Fear of death
– Fear of “going nad”
– Nausea or stomach discomfort
– Numbness or tingling
– Frequent palpations
– Shaking
The attack ends with the excessive urination.

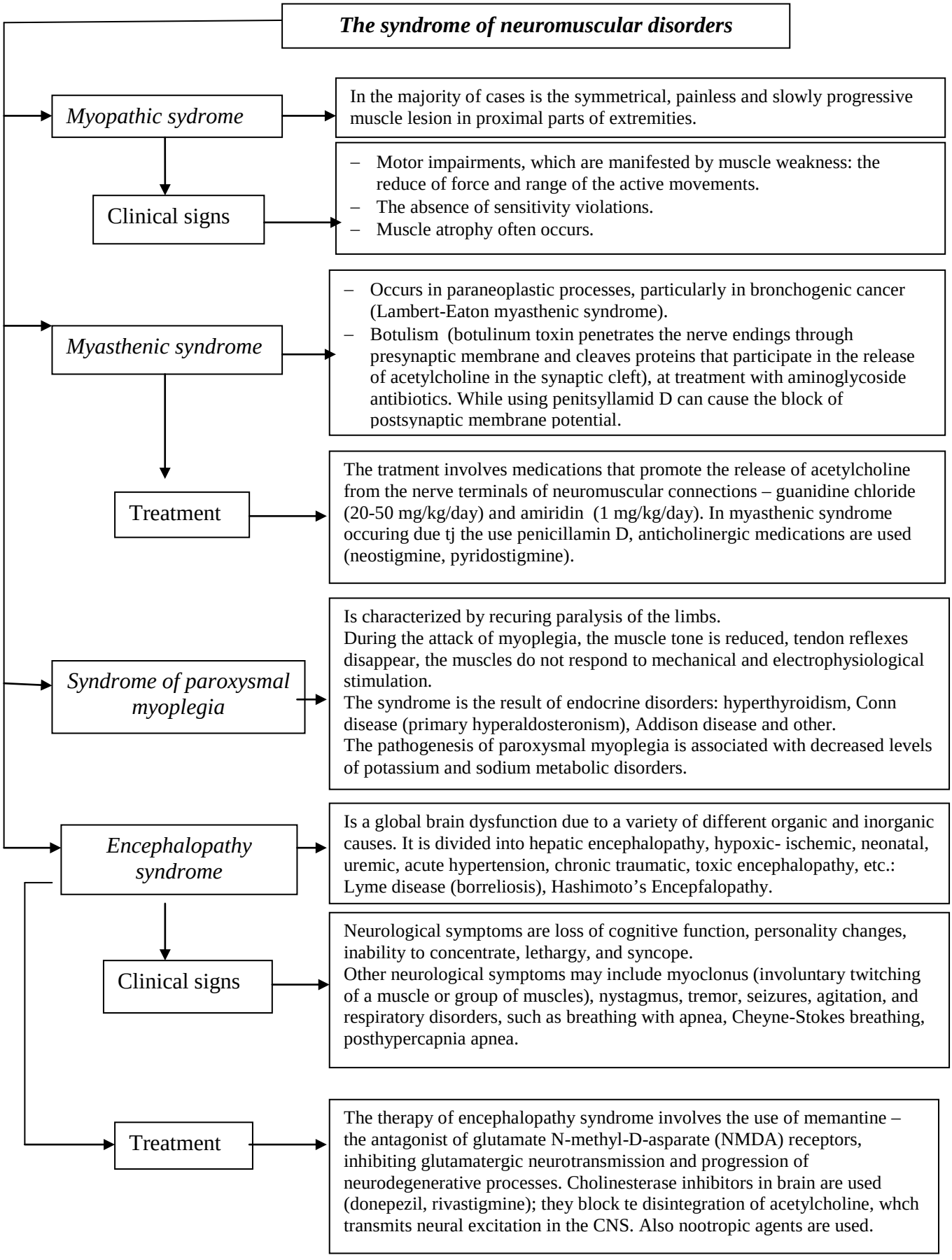


Syndrome of polyneuropathy

Syndrome of polyneuropathy is a multiple lesion of the distal regions of peripheral nerves (see topic: peripheral nerve)/ it is observe in infection (influenza, diphtheria), intoxications (alcohol, lead, etc.), metabolic disorders (diabetes mellitus, etc), avitaminosis.

Depending on the affected tunks of peripheral nerves (autonomic, sensory, motor), polyneuropathy is characterized by pain, numbness in the limbs, sensetivity disturbances by the type of “glover” and “socks”, weakness and hypotonia of muscles in distal parts of arms and legs accordingly.

The therapy is based on treating the main disease, also vitamin B complex, antichlinergic agents, massage, physical therapy, exercise therapy are used.


The syndrome of neuromuscular disorders
Myopathic sydrome
In the majority of cases is the symmetrical, painless and slowly progressive muscle lesion in proximal parts of extremities.
Clinical signs
– Motor impairments, which are manifested by muscle weakness: the reduce of force and range of the active movements.
– The absence of sensitivity violations.
– Muscle atrophy often occurs.
Myasthenic syndrome
– Occurs in paraneoplastic processes, particularly in bronchogenic cancer (Lambert-Eaton myasthenic syndrome).
– Botulism (botulinum toxin penetrates the nerve endings through presynaptic membrane and cleaves proteins that participate in the release of acetylcholine in the synaptic cleft), at treatment with aminoglycoside antibiotics. While using penitsyllamid D can cause the block of postsynaptic membrane potential.
Treatment
The tratment involves medications that promote the release of acetylcholine from the nerve terminals of neuromuscular connections – guanidine chloride (20-50 mg/kg/day) and amiridin (1 mg/kg/day). In myasthenic syndrome occuring due tj the use penicillamin D, anticholinergic medications are used (neostigmine, pyridostigmine).
Syndrome of paroxysmal myoplegia
Is characterized by recuring paralysis of the limbs.
During the attack of myoplegia, the muscle tone is reduced, tendon reflexes disappear, the muscles do not respond to mechanical and electrophysiological stimulation.
The syndrome is the result of endocrine disorders: hyperthyroidism, Conn disease (primary hyperaldosteronism), Addison disease and other.
The pathogenesis of paroxysmal myoplegia is associated with decreased levels of potassium and sodium metabolic disorders.
Encephalopathy syndrome
Is a global brain dysfunction due to a variety of different organic and inorganic causes. It is divided into hepatic encephalopathy, hypoxic- ischemic, neonatal, uremic, acute hypertension, chronic traumatic, toxic encephalopathy, etc.: Lyme disease (borreliosis), Hashimoto's Encepfalopathy.
Clinical signs
Neurological symptoms are loss of cognitive function, personality changes, inability to concentrate, lethargy, and syncope.
Other neurological symptoms may include myoclonus (involuntary twitching of a muscle or group of muscles), nystagmus, tremor, seizures, agitation, and respiratory disorders, such as breathing with apnea, Cheyne-Stokes breathing, posthypercapnia apnea.
Treatment
The therapy of encephalopathy syndrome involves the use of memantine – the antagonist of glutamate N-methyl-D-asparate (NMDA) receptors, inhibiting glutamatergic neurotransmission and progression of neurodegenerative processes. Cholinesterase inhibitors in brain are used (donepezil, rivastigmine); they block te disintegration of acetylcholine, whch transmits neural excitation in the CNS. Also nootropic agents are used.

THEMA: CEREBRAL PALSY

(INFANT CEREBRAL PALSY)

Etiology	Pathology of intrauterine development (during pregnancy pathology, diseases of the mother, intoxication, immunological incompatibility between mother and fetus) and mechanical factors.
Pathogenesis and pathomorphology	Fetal brain hypoxia; presence of embryonic cells in the cortex, areas of softening, caveties with glial cells, necrosis in the subcortical nodes, various anomalies odf development.
Mzin clinical syndromes	Pyramidal (paresis, paralysis). Extrapyramidal (various variants of hyperkinesis). Myscular dystonic. Cerebellum. Intellectual disability.
Clinical forms	Hemiplegic, tetraplegic. Little. Cerebellum. Hyperkinetic.
Diagnostic	Clinic signs, anamnesis, MRI, CT-scan of brain.
Treatment	Medical gumnastics. Balneotherapy and mud therapy. Nootropics. Holinoloki. Agonists and antagonists. Muscle relaxants. Ascular therapyy.

RECOMMENDED LITERATURE

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