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"NORTH OSSETIAN STATE MEDICAL ACADEMY"
Ministries of Health of the Russian Federation**

DEPARTMENT OF CHILDREN'S DISEASES №2



**EDUCATIONAL AND METHODOICAL RECOMMENDATIONS
OF DISCIPLINE PEDIATRICS**

Part II

Vladikavkaz, 2020

The methodical recommendations for out-of-class independent work for students of discipline pediatrics was approved at the meeting of the Department of Children's Diseases No.2 dated November 25, 2020, protocol No.4.

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Class in a subject:
"RICKETS"

I. Scientific and methodical justification of a subject.

Rickets is one of the most widespread diseases of children of early age. Without being a cause of death, rickets, nevertheless, indirectly increases the lethality of children since promotes heavier and adverse course of all diseases of children of the first year of life. The expressed forms of rickets lead to permanent deformations of skeletal system which remain for the rest of life. Doctors of different specialties (therapists, oculists, stomatologists) will deal with consequences of this pathology. In this regard recognition and timely treatment and prevention of this disease is important.

II. Purpose of activity of students on occupation:

The student has to know:

- the contributing factors to development of rickets;
- main pathogenetic links of rickets;
- morphological changes in a bone tissue in rickets;
- main clinical symptoms of rickets;
- functional changes from internals and systems in rickets;
- classification of rickets;
- laboratory and radiological diagnostic methods of rickets;
- basic principles of treatment and prevention of rickets;
- possible complications at vitamin D use.

The student has to be able:

- to purposefully collect the anamnesis;
- to perform objective examination of the child;
- to interpret laboratory and radiological data;
- to make the developed diagnosis taking into account classification;
- to make the treatment plan of the patient;
- to write prescriptions on the main medicines;
- to define preventive actions.

III. Content of training:

1. The major factors contributing to development of rickets.
2. Main pathogenetic links of rickets. A role of epithelial bodies in pathogenesis of rickets

3. Morphological changes in a bone tissue in rickets.
4. Main clinical symptoms of rickets.
5. Classification of rickets (the principles of division on the periods, a course, weight).
6. Laboratory and radiological changes in rickets.
7. Treatment and prevention of rickets at children.

IV. Educational material security.

1. Visual aids: tables, schemes, multimedia presentations, videos, audiogramma.
2. Educational medical documentation (case histories, laboratory researches, roentgenograms).
3. Technical means of training.
4. Literature.

V. The list of the recommended literature.

1. Children's diseases: the textbook / under the editorship of A.A. Baranov. – M.: GEOTAR-media, 2009. – 1008 pages.
2. Pediatrics: The textbook for medical schools. Under the editorship of N.P. Shabalov. – SPb: SpetsLit, 2006. – 895 pages.
3. Propaedeutics of children's diseases / to N.A. Geppa. – M.: GEOTAR-media, 2009. – 464 pages.
4. F.P. Romaniuk Rickets. A grant for doctors. – SPb.: 2002.
5. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Background diseases of children of early age. Manual for students. – Vladikavkaz, 2011. – 64 pages.
6. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Technique of a research of the child. The study guide for students. – Vladikavkaz, 2011. – 51 pages.
7. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Scheme of a case history. The study guide for students. – Vladikavkaz, 2011. – 38 pages.
8. Lectures on pediatrics.
9. Methodical instructions for out-of-class work of students 4 courses of medical faculty on discipline "Pediatrics".

VI. List of questions for check of initial level of knowledge:

1. What features of the structure of long tubular bones at children?
2. Call morphological features of a bone tissue at children.
3. What need for phosphorus and calcium at children of chest age?

4. Call features of a mineral metabolism at children.
5. What role of vitamin D in a human body?
6. Call the main metabolic transformations of vitamin D in an organism.

VII. List of questions for check of final level of knowledge:

1. List the major factors contributing to development of rickets.
2. Characterize the main pathogenetic links of rickets.
3. Classification of rickets (the principles of division on the periods, a course, weight).
4. Call the main clinical symptoms of rickets depending on the period and a course.
5. What laboratory and radiological changes can be revealed at the patient with rickets.
6. Call the main methods treatment and prevention of rickets at children. What is specific and nonspecific methods of treatment and prevention of rickets?
7. Give basic reasons of development of a hypervitaminosis of D in children.
8. List clinical manifestations of a hypervitaminosis of D.
9. What does prevention and treatment of this state consist in?

Information block.

RICKETS

– the disease of children of early age caused by disorder of calcic and phosphoric exchange in connection with deficiency of vitamin D is shown by disturbance of processes of education and a mineralization of bones and also functions of nervous system and internals.

Epidemiology

Classical rickets remains one of the most widespread diseases of children's age. It strikes children during rapid growth, aged up to 2-Z years the frequency of rickets reaches 35%.

Etiology and pathogenesis

At deficiency of vitamin D the synthesis of the calcium connecting protein providing calcium transport in intestines decreases owing to what concentration of calcium in blood decreases. The hypocalcemia stimulates activity of epithelial bodies – the products of parathyroid hormone raise. Owing to its surplus there is a strengthened removal of calcium from a bone tissue and also the reabsorption of phosphates in renal tubules decreases. Quickly the hypophosphatemia develops, the alkaline reserve of blood decreases, there is acidosis. In the conditions of acidosis the process of a mineralization of ossiform fabric is broken. Reduction of content of salts of calcium and phosphorus in bones leads to osteoporosis and osteomalacy. Bones become soft and are easily deformed. At the same time in regions of growth there is a growth of

defective ossiform fabric. The developed acidosis leads to disturbance of the central nervous system functions and internals.

The disease usually develops at the children having any given factors of predisposition which range at each child is individual. The combination of exogenous and internal causes determines terms of a demonstration and weight of a course of rickets.

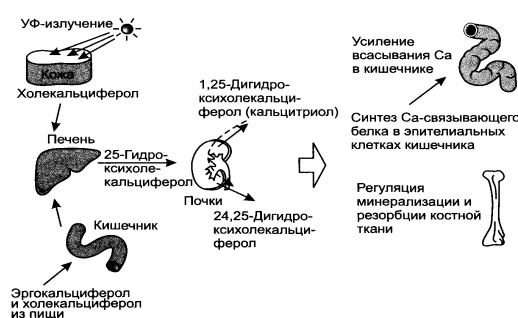
The factors contributing to rickets from the pregnant woman:

- age less than 18 and more than 36 years;
- gestosis;
- extragenital pathology (exchange diseases, pathology of a GIT, kidneys);
- defects of food during pregnancy and a lactation (deficiency of vitamin E), protein, calcium, phosphorus, vitamins of group C);
- non-compliance with a day regimen (insufficient insolation, hypodynamia);
- the complicated childbirth;
- unsuccessful social and economic conditions.

The factors contributing to rickets from the child:

- birth time (the children born from June to December are ill more often);
- prematurity, ZVUR;
- big body weight at the birth (more than 4 kg);
- big increase of body weight within the first 3 months of life.

Exchange of vitamin D and its physiological effects



Classification of rickets

Disease period	Weight of a course	Nature of a course
Initial	The I degree (easy)	Sharp
Disease heat	The II degree (cf. weights)	Subacute
Convalescence	The III degree (heavy)	Wavy
Residual phenomena		

Clinical picture

The leading clinical signs of rickets – bone changes.

➤ *Skull bones.*

- Craniotabes – softening and thinning of flat bones of a skull, Usually reveal in occipital or parietal area where the skull is softened so that gives in to squeezing. Not, which authors consider this symptom as the physiological phenomenon up to 4 months.

- Delay of closing of fontanelles and seams between skull bones. It should be noted, however, that terms of closing of fontanelles and seams of a skull are subject to considerable individual variability

- Delay of emergence of teeth.

➤ *Thorax.*

- Rachitic "beads" (thickenings on both sides of a breast owing to a hypertrophy of ossiform fabric in the field of costochondral joints).

- Deformation of a thorax (impression in the lower third of a breast – "the shoemaker's breast", its protrusion – a "chicken" or "keeled" breast).

➤ *Backbone.*

- Lack of physiological bends or appearance of a pathological kyphosis, lordoses and scolioses.

➤ *Extremities.*

- Thickening of epiphyses because of proliferation of badly calcific bone matrix that is especially noticeable on anklebones and wrists (rachitic "brasetka").

- The deformations of pelvic bones and lower extremities becoming noticeable at the end of the first and the beginning of the 2nd year of life (About - To - or a H-shaped curvature of legs, a flat rachitic basin).

In rickets, besides skeletal system, also other bodies and systems are surprised. Changes from central nervous system are most typical and the phenomenon of muscle weakness. The behavior of the child changes, he becomes irritable, the appetite decreases. Development of conditioned reflexes slows down, and acquired reflexes weaken or absolutely disappear. Hypotonia of muscles leads to decrease in physical activity, a stomach otvisaniye. Frequent SARS, latent anemia are characteristic.

Depending on expressiveness of clinical manifestations distinguish three severity of rickets. Now easy forms prevail that creates certain difficulties in diagnostics, especially at assessment of activity and sharpness of pathological process.

The course of rickets depends on age of the child, the nature of feeding, a season of year,

features of the mode and other factors. The beginning and exacerbation of a disease observe, as a rule, late fall, in the winter and in the early spring. In summertime the process calms down and there occurs recovery. Rickets usually on the 2-3rd month of life, to 5-6-month age begins, especially in the absence of preventive and treatment, in process of development of the profound acidosis the course of the disease becomes sharp and is followed by fast development of all symptoms. If medical measures are not undertaken or they are inadequate, the subacute course of rickets with moderate changes from neuromuscular and bone systems develops. Even easy forms of rickets with hardly noticeable external manifestations reduce body resistance that creates prerequisites for developing of other diseases. Especially adversely rickets at newborn and premature children proceeds.

At some children with rickets, mainly at the age of 5-15 months, note tendency to tonic and tonico-clonic spasms – a so-called spasmophilia the reason of increase in neuromuscular excitability and spasms consider decrease in concentration of the ionized calcium in blood serum and interstitial liquid. Allocate the obvious and hidden forms of a spasmophilia.

The obvious spasmophilia is shown by a laryngospasm, a carpopedal spasm and eklampsichesky attacks.

The laryngospasm proceeds with partial or full closing of a glottis. During a partial laryngospasm at the child observe the scared look, protruding eyes, cyanosis, characteristic "cock" shout on a breath. At full closing of a glottis and the termination of breath there is a loss of consciousness. Attacks proceed from several seconds to 1-2 min. and can repeat several times a day. Each attack poses a threat for life. The external irritating factors provoke attacks: loud sounds, bright light and so forth.

The carpopedal spasm arises at children more often 1 years are more senior and it is shown by tonic myotonia of brushes and feet.

The Eklampsichesky form of a disease proceeds with heavy attacks during which note the tonic and clonic spasms of extremities and trunks which are followed by a loss of consciousness. Attacks repeat, each of them proceeds 2-3 min. and threatens life of the sick child.

At the latent form of a spasmophilia at children reveal the symptoms demonstrating presence of the increased neuromuscular excitability. So, at percussion the reduction of mimic muscles on the relevant party (Hvostek's symptom) happens a percussion hammer or a halfbent finger between a zygoma and a corner of a mouth. At compression of a neurovascular bunch in a shoulder there is a convulsive reduction of the brush adopting the provision "obstetrical hands" (Trusso's symptom). At a prick of skin of a leg there is short-term an apnoea (normal breath

amplifies) – Maslov's symptom.

Diagnostics

In the presence of typical clinical manifestations the diagnosis usually does not cause difficulties. In case of the subacute course of the disease the leading value is gained by laboratory researches. Due to the complexity and inaccessibility for practical health care of methods of early diagnosis of hypovitaminosis by direct definition of concentration in blood of metabolites of vitamin D use indirect diagnostic methods, such as definition in blood serum of concentration of calcium, inorganic phosphorus, activity of alkaline phosphatase.

- Concentration of calcium in blood serum is usually reduced to 2.0-2.2 mmol/l (at norm of 2.4-2.7 mmol/l).
- Concentration of phosphorus in blood serum can be normal or is reduced to 0.65 mmol/l and below (at norm at children of the first year of life of 1.3-2.3 mmol/l).
- The activity of alkaline phosphatase (more than 220 Pieces/l) increases.
- With urine the increased amount of amino acids – an aminoaciduria more than 10 mg/kg/days is distinguished.
- On roentgenograms of bones reveal diffusion osteoporosis with the significant structural changes of a bone tissue (melkoyacheistost).

Differential diagnostics

Differential diagnosis of rickets is carried out with so-called rakhitopodobny diseases (tubulopatiya).

Treatment

Medical influences at children with rickets are directed to vitamin D shortage control, normalization of calcic and phosphoric exchange, elimination of acidosis, strengthening of processes of an osteogenesis. Children with rickets need good nutrition, it is desirable to keep breastfeeding

Medicinal therapy of rickets consists in prescribing of cholecalciferol (vitamin D). Intake of cholecalciferol should be begun as soon as possible: up to 3 months. Drug is appointed in drops in a daily dose 1500-4000 ME (depending on severity). For these purposes use water (for example, Aquadetrim) or oil (for example, "Videcholum" of 0.125% or 0.25%) solution in drops. Drug is released for intake, 1 ml (30 drops) contains ME cholecalciferol 15,000 for aqueous solution and 20,000 ME for Solutio oleosa, 1 drop – 500-600 ME (or 1000 ME for 0.25% of solution of "Videcholum"). Drug is recommended to dissolve in a milk spoon, addition of drops in a small bottle is not recommended as at the same time necessary concentration of active

ingredient usually is not reached.

At intake of cholecalciferol it is necessary to provide receipt of enough calcium (the diet enriched with calcium or calcium drugs). At oral prescribing of calcium preference is given to bioavailable forms, for example calcium carbonate. Use of glycerophosphate or a gluconate of calcium is also possible. Dosages make from 250-500 mg. in the first half of the year of life up to 400-750 mg in the 2nd half-year.

For improvement of digestion of salts of calcium and phosphorus in intestines, increase in a reabsorption of phosphates in kidneys and strengthenings of processes of bone formation appoint citrate mix on 1 teaspoon 3 times a day within 10-12 days.

For the purpose of normalization of functions of epithelial bodies and elimination of a hypomagnesiemia include one of magniysoderzhashchy drugs in complex treatment of rickets (potassium and magnesium asparaginate) or magnesium sulfate (1% solution) at the rate of 10 mg. magnesium on 1 kg. in day during 3-4 weeks.

Hospitalization is shown to children with an obvious spasmophilia. For stopping of spasms use diazepam (0.1 ml of 0.5% of solution for 1 kg. body weights), magnesium sulfate (0.5 ml of 25% of solution for 1 kg. body weights), piperidic acid (0.5 ml of 20% of solution for 1 kg. body weights). Administration of Calcii chloridum is obligatory (0.3-0.5 ml of 10% of solution for 1 kg. body weights, enter intravenously slowly).

For removal of a laryngospasm create the prepotent center of excitement in a brain by irritation of a mucous membrane of a nose (blow in a nose, tickle, bring liquid ammonia), skin (a prick, pat and douche by cold water), a vestibular mechanism (stirring of the child, change position of a body).

When assigning high doses of vitamin D it is necessary to control a calciuria (Sulkovich's test) and also, whenever possible, and a kaltsiyemiya.

In 2 weeks from the beginning of medicinal therapy include massage and LFK in a complex of treatment by all patient. To children 6 months reasonably carrying out a balneoterapiya in the form of medical bathtubs are more senior: coniferous, salt or from broth of herbs. Pine needle baths are shown to excitable children. For preparation of a bathtub in 10 l. waters with a temperature of 37 °C add 1 teaspoon of natural liquid coniferous extract or a standard strip of a briquette. The first bathtub is carried out within 5 min., then time is extended up to 6-10 min., all for a course is recommended by 12-15 bathtubs daily or every other day.

After a rate of cholecalciferol the radiation can be appointed a mercury-quartz lamp (Ural federal district). Ural federal district is carried out after determination of individual sensitivity to

ultraviolet rays (biodose) and appointed daily or every other day with $1/4$ — $1/2$ up to 4 biodoses to certain fields at focal length of 50-100 cm and duration of a course of treatment of 20-25 days. During intake of Ural federal district vitamin D do not carry out.

Prevention

Prevention of rickets should be begun till the child's birth. At patronage of pregnant women draw the attention of future mother to need of observance of the correct day regimen with alternation of work and rest, an exception of physical overworks, sufficient stay in the fresh air, a balanced diet. For receipt of adequate amount of calcium the diet of the pregnant woman has to contain enough milk or dairy products, in case of their intolerance appoint calcium drugs. The daily dose of vitamin D for pregnant women makes 400 ME. The feeding woman has to receive 1200 mg a day. calcium and 800 ME vitamins D.

Post-natal prevention of rickets at children is connected with the organization of healthy nutrition of the child from the first days of life. To the children who are on natural feeding, vitamin D is appointed from 3-4th week of life on 500 ME daily, excepting summer months. Prevention is carried out to 1-1.5 years. The children who are on artificial feeding by the adapted mixes receive all necessary vitamins B physiological doses therefore they usually do not need additional intake of vitamin D.

Tasks for independent preparation:

1. Solve situational problems and tasks of test control.
2. Examine the patient with rickets, describe the changes in the state of health revealed by you.
3. Write out in a workbook:
 - Videcholum
 - calcium gluconate

Scheme of inspection of the patient.

When collecting the anamnesis pay attention on:

- features of feeding of the child;
- gestation term at the birth;
- living conditions;
- features of child care, its stay in the fresh air;
- incidence of the child;
- quality of antenatal and post-natal prevention of rickets.

At an objective research to pay attention on:

- existence at the child of perspiration, hyperexcitability, a sleep disorder, nocturnal myoclonias;
- color of integuments and visible mucous membranes;
- size and form of a head of the child;
- size and condition of edges of a big fontanel;
- state at a palpation of an occipital bone;
- quantity and quality of teeth, terms and sequence of their eruption;
- existence of deformations of a thorax, backbone and extremities;
- decrease in a muscle tone;
- size and shape of a stomach, divergence of direct muscles.

At interpretation of datas of laboratory:

- in complete blood count test to pay attention to the maintenance of erythrocytes, hemoglobin;
- to estimate the content of phosphorus, calcium and alkaline phosphatase in blood serum;
- to give an assessment of test of Sulkovich.

When reading roentgenograms to pay attention on:

- condition of a cortical layer of a bone;
- condition of the region of growth;
- structure of a bone tissue.

Situational tasks***Task No. 1***

The girl of 10 months came to clinic with complaints to pallor, a loss of appetite, slackness. Was born full-term (body weight 3300 gr., length of 49 cm), from the I pregnancy proceeding with toxicosis and births in time. From 3.5 months was on artificial feeding, practically did not receive fruit and berry juice, from 5 months it is raised mainly by porridges. Prevention of rickets was not carried out. Began to hold the head from 4 months, to sit from 9 months, the first teeth were cut through in 8 months, only 4 teeth.

At survey: pallor, tearfulness, a hypomyotonia, a stomach it is increased in volume, the umbilical ring is expanded, frontal and occipital hillocks act, at a palpation of a thorax is defined costal beads. Pulse of 142 beats/min, symmetric, rhythmical, satisfactory filling. The upper bound of warm dullness at the level of II edges, right – the right sternal line, left – the left mamillar line.

Cardiac sounds are moderately muffled, on a top gentle systolic noise. The liver acts from under a costal arch on 3 cm, edge equal, smooth. The spleen is not palpated.

Complete blood count test: Ayr – $2,8 \cdot 10^{12}/l$, Hb – 76 g/l, Ley – $13,2 \cdot 10^9/l$, Tsv. the item – 0.6, reticulocytes – 2.8%, e/f – 1%, p.b. – 2%, with / I am 29%, l/c – 57%, m/c – 8%.

Questions:

1. Your diagnosis?
2. What possibilities of a cause of illness and risk factors?
3. Offer the plan of additional inspection, a balanced diet and medicamentous therapy.

Task No. 2

Mother with the boy of 6 months came to the next preventive reception to the pediatrician.

The child from the IV pregnancy proceeding against the background of toxicosis in the I first trimester. Childbirth in time, with stimulation. Was born with body weight 3500 gr., length – 53 cm, cried at once. Natural feeding up to 2 months, then cow's milk in half with boiled water, semolina porridge, kefir. Mother visits children's polyclinic irregularly, inoculations with disturbance of the schedule.

Within the last 2 months mother of the child pays attention that the child began to sweat, shudder strongly in a dream, from pampers a pungent smell of ammonia.

At survey: body weight 8000 gr., length is 66 cm. Flattening and baldness of a nape, pliability of bones of a skull on the course of arrow-shaped and lyambdovidny seams, a big fontanel 3 x 3 cm pays attention, edges are pliable. The lower aperture of a thorax is developed, noticeable Garrisonova a furrow, costal "beads" are palpated. The hypomyotonia, badly leans on legs. In natural folds of skin not plentiful elements of a heat rash, a resistant red dermographism. Mucous clean. Puerile breath, there are no rattles. Clear cardiac sounds, rhythmical, beats/min ChSS-120. The pot-belly spread. The liver on 2.5 see below edges of a costal arch, a spleen is not palpated. A chair with tendency to constipations.

Questions:

1. Make the preliminary diagnosis.
2. Draw up the plan of inspection.
3. What results do you expect to receive?
4. Appoint treatment.

Task No. 3

The girl of 8 months, in March came to chest department with the profound tonic spasms.

From the anamnesis it is known that the girl from the first pregnancy proceeding with toxicosis, arterial hypotonia, anemia of the I degree, spasms in gastrocnemius muscles. Childbirth in time. Weight at the birth 3800 gr., length – 53 cm. Since the birth on artificial feeding. The feeding up is entered since 4 months. Now receives: porridges, vegetable puree, kefir. From 5 months the rickets is diagnosed. Treatment is appointed vitamin D Solutio oleosa₂.

Objectively: the girl of supernutrition, the head of a hydrotsefalny form, frontal and occipital hillocks are expressed. Craniotabes. A big fontanel of 2x2 cm, edges are pliable. The thorax of a keeled form, the lower aperture is developed. The turgor of fabrics is sharply reduced. Tension of gastrocnemius muscles, symptom of "obstetrical hand". Cardiac sounds are slightly muffled, ChSS 159 ud. in min. In lungs non-constant small-bubbling damp rattles. The stomach is spread. The liver on 3 cmacts from under a costal arch, the spleen is not palpated. Sits with a support, is not necessary, periodically tonic spasms.

Task:

1. Formulate the preliminary diagnosis.
2. What researches need to be conducted for confirmation of the diagnosis?
3. What pathogenesis of development of a convulsive syndrome?
4. What therapy of this disease?

Test control:

1. Of rickets it is characteristic:
 - a) metabolic acidosis
 - b) alkalosis
 - c) respiratory acidosis
 - d) metabolic alkalosis
2. The ratio of level of calcium and phosphorus in blood is normal equally:
 - a) 2:1
 - b) 1:2
 - c) 3:1
 - d) 1:3
3. 25 hydrocholecalciferol are formed in:
 - a) kidneys
 - b) liver
 - c) in intestines
 - d) bones

4. 1, 25-dihydroxycholecalciferol is formed in:
- a) kidneys
 - b) liver
 - c) in intestines
 - d) bones
5. In the period of a heat of rickets it is observed:
- a) muscular hypertension
 - b) craniotabes
 - c) convulsive syndrome
6. In an organism **does not influence** phosphorus-calcium exchange:
- a) calcitonin
 - b) 1, 25-dihydroxycholecalciferol
 - c) somatotrophic hormone
 - d) parathormone
 - e) corticosteroids
7. Vitamin D contains in:
- a) yolk
 - b) bread of a rough grinding
 - c) vegetables
 - d) meat
8. The preventive dose of water vitamin D makes:
- a) 200 ME
 - b) 500 ME
 - c) 2-3 thousand ME
 - d) 1000 ME
9. Of an initial stage of rickets **it is not characteristic**:
- a) tearfulness
 - b) perspiration
 - c) loss of appetite
 - d) the increased convulsive readiness
 - e) bone deformations
10. Is the reason of spasms in a spasmophilia:
- a) hypocalcemia

- b) hypophosphatemia
 - c) hypercalcemia
 - d) hypopotassemia
11. The spasmophilia meets:
- a) at newborns
 - b) at babies
 - c) in the pubertal period
12. Gipokaltsiyemichesky spasms arise at decrease in the ionized blood plasma calcium below:
- a) 0.5 mmol/l
 - b) 0.85 mmol/l
 - c) 1.0 mmol/l
 - d) 1.5 mmol/l
13. Reduction of muscles of the person at percussion on a zygoma is called a symptom:
- a) Tail
 - b) Trusso
 - c) Maslova
14. Is season when the spasmophilia meets more often:
- a) winter
 - b) fall
 - c) summer
 - d) spring
15. The caused muscular contraction reminding the provision "obstetrical hand" is called a symptom:
- a) Tail
 - b) Trusso
 - c) Maslova
16. An antagonist of vitamin D is vitamin:
- a) And
 - b) In₆
 - c) In₁₂
 - d) E
17. The contributing factors to development of a hypervitaminosis of D are:

- a) total dose of ME D 1000 000 vitamin and more
- b) hypersensitivity to vitamin D
- c) chronic diseases of kidneys
- d) anemia

18. The porridge having antikaltsifitsiruyushchy property and recommended in the child's diet in a hypervitaminosis of D is:

- a) buckwheat
- b) rice
- c) oat

19. Of the II degree of a hypervitaminosis of D it is characteristic:

- a) absence of toxicosis
- b) moderate toxicosis
- c) loss of appetite
- d) vomiting
- e) poorly positive test of Sulkovich
- e) sharply positive test of Sulkovich
- g) falling of body weight

Class in a subject: "SCARCE ANEMIAS"

I. Scientific and methodical justification of a subject.

Anemias are widespread as among adults, among children of different age so to a large extent that it is quite often closely interconnected, being implemented through interrelation "mother child". Knowledge of the main medical and diagnostic actions in anemia of different genesis is necessary for the doctor any and specialties.

Considering a set of clinical forms of anemias when passing a course of pediatrics by students of medical faculty, analysis of the scarce anemias which are most often found and having significant effect on an organism of the child and his future, having directly causal relationship with mother's organism developing both at children of early age, and at teenagers is reasonable.

II. Purpose of activity of students.

The student has to know:

- types of scarce anemias;

- reasons and risk factors of deficiency of iron of mother and child;
- features of exchange of iron at children and an iron role in an organism;
- main mechanisms of pathogenesis of iron deficiency anemias;
- reasons and mechanism of development of vitaminodeficiency anemias;
- food role in development of scarce anemias;
- value of background and intercurrent diseases in development, a course and treatment of scarce anemias;
- clinical and hematologic signs of scarce anemias;
- principles of treatment and prevention of iron- and vitaminodeficiency anemias.

The student has to be able:

- to collect the purposeful anamnesis and to analyze it;
- from the general objective survey to mark out clinical signs of anemia;
- to make the plan of inspection and to appoint the additional researches necessary for disclosure of weight, the nature of anemia;
- to estimate blood test and other additional researches;
- to make the diagnosis according to modern classifications by carrying out the differential diagnosis of anemias of different genesis;
- to make the specific treatment plan of the patient landmark (a hospital, polyclinic);
- to appoint dietary food to the patient with anemia or to the child from risk group;
- drug treatment with the indication of doses, duration of courses, etc., depending on weight and pathogenesis of anemia;
- to recommend preventive actions for mother and the child.

III. Content of training:

1. Classification of anemias.
2. The major etiological factors leading to development of anemias in children.
3. Concept "scarce anemias". Reasons of their development.
4. The main pathogenetic mechanisms leading to a clinical syndrome of anemias.
5. Clinical laboratory characteristics of scarce anemias at children.
6. Modern methods of treatment and prevention of scarce anemias at children.

IV. Educational material security.

1. Visual aids: tables, schemes, multimedia presentations, videos, audiogramma.
2. Educational medical documentation (case histories, laboratory researches,

roentgenograms).

3. Technical means of training.
4. Literature.

V. The list of the recommended literature.

1. Children's diseases: the textbook / under the editorship of A.A. Baranov. – M.: GEOTAR-media, 2009. – 1008 pages.
2. Pediatrics: The textbook for medical schools. Under the editorship of N.P. Shabalov. – SPb: SpetsLit, 2006. – 895 pages.
3. Propaedeutics of children's diseases / to N.A. Geppa. – M.: GEOTAR-media, 2009. – 464 pages.
4. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. An iron deficiency anemia at children. Manual for students. – Vladikavkaz, 2011. – 41 pages.
5. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Background diseases of children of early age. Manual for students. – Vladikavkaz, 2011. – 64 pages.
6. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Technique of a research of the child. The study guide for students. – Vladikavkaz, 2011. – 51 pages.
7. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Scheme of a case history. The study guide for students. – Vladikavkaz, 2011. – 38 pages.
8. Lectures on pediatrics.
9. Methodical instructions for out-of-class work of students 4 courses of medical faculty on discipline "Pediatrics".

VI. List of questions for check of initial level of knowledge:

1. Physiology of an erythrocyte system (what is erythron, erythrocyte balance, a normal erythrokinetics).
2. Features of an anti- and post-natal erythrocytogenesis.
3. Functions of an erythrocyte.
4. Iron role (features of forming of depot) in an erythrocytogenesis.
5. Role of vitamin B₁₂, folic acid in an erythrocytogenesis.
6. Age dynamics (curve) Hb and erythrocytes.
7. Normal hematologic (laboratory) characteristic of an erythrocyte system.

VII. List of questions for check of final level of knowledge:

1. What is anemia?
2. What types of anemias do you know?
3. What risk factors of developing an iron deficiency anemia do you know?
4. What key clinical laboratory indicators of iron deficiency anemias at children?
5. What treatment is performed at a zhelezodefitsita? Give characteristic to the main ferriferous drugs.
6. What does prevention of iron deficiency states consist in? For what contingent of the children's population similar prevention is necessary?
7. Give basic reasons of development of megaloblastny anemia.
8. What changes of laboratory indicators at the same time are observed?
9. Call methods of treatment and prevention of anemias at a vitaminodefitsita.

Information block

To anemias according to the WHO classification, carry states at which a hemoglobin content lower than 110 g/l at children up to 6 years are reduced and – at children 6 years are more senior than lower than 120 g/l.

There are many options of classification of anemias. It is essentially important that causes of anemias only four:

- 1. blood loss;**
- 2. hemolysis;**
- 3. decrease in products of erythrocytes;**
- 4. deposition (sequestration) of blood.**

The most frequent option of anemias at children are ***microcytic iron deficiency anemias***.

Iron participates in activity of each cell of an organism. The main part of iron is included in hemoglobin (60%) and a myoglobin (9%).

Reduction of content of iron brings in blood serum, marrow and fabric depots to:

- to decrease in rates of synthesis and disturbance of formation of hemoglobin;
- to accumulation of free protoporphyrin in erythrocytes;
- to development of hypochromia anemia;
- trophic to disorders in fabrics.

At children at the highest tension of a metabolism and its anabolic orientation at quite often available immaturity of fermental systems the deficiency of iron is felt most sharply. The

deficiency of iron leads to decrease in ferriferous euzymatic systems – cytochrome, a catalase, peroxidase. It considerably influences on fabric and cellular metabolism.

Features of exchange of iron in a children's organism.

Reserves of iron begin to be formed at receipt through a placenta. At normally proceeding pregnancy mother gives to a fruit about 300 mg. gland. Most actively this process proceeds from 28-32 weeks of pregnancy.

To a placenta iron is delivered by transferrin. The placenta is not surmountable for the pregnant woman's transferrin. The exact mechanism of transfer of iron through a placenta is not clear. It is known that iron transport – the active process which is carried out against a concentration gradient only in one direction – from mother to a fruit. It is supposed that in a placenta there is a highly active fermental system extracting iron from maternal transferrin, and transferring to its placentary ferritin or fetal transferrin. Transferrin of a fruit delivers iron in marrow where erythrocytes are synthesized, and in fabric where iron is a part of various fermental systems. The excess of iron is deposited in a liver and muscles in the form of ferritin.

After the birth sources of iron are:

- exogenous iron of foodstuff;
- utilization of iron from endogenous stocks.

Antenatal stocks are quickly exhausted. The daily physiological need for iron at children makes 0.5-1.2 mg/days. If preventive therapy of a sideropenia is not carried out, then even at natural feeding by 3 months at premature and by 5-6 months at full-term development of an iron deficiency anemia is possible.

In exchange of iron in the post-natal period it is possible to allocate the following stages:

- absorption of iron in a GIT;
- iron transport in an organism;
- deposition of iron in an organism.

Absorption of iron in a GIT.

- a) bivalent iron is taken cells of a mucous membrane of a small intestine;
- b) oxidation of bivalent iron in trivalent in a membrane of microvillis of cells mucous a small intestine;
- c) the future of iron depends on iron reserves in an organism:
 - at excess of iron it is late in epithelial cells of a mucous membrane in connection with ferritin. Then with the exfoliated epithelium is removed from an organism;

- at a lack of iron the speed of its absorption increases. Its most part is soaked up in a blood stream, connects to transferrin.

Within the first 3-4 months of life, maternal milk is the only product which provides balance of exchange of iron. And, iron of female milk is acquired more effectively – 38-49%. From cow's milk the absorption of iron does not exceed 10%.

When choosing food it is necessary to consider not only quantity, but also a qualitative form of its connections. In iron dried fruits - 15 mg / 100 gr., in beef meat - 2.6 mg / 100 gr., but iron from meat is acquired much more actively that is explained by more effective absorption of heme iron. The heme iron complex (heme) connects to receptors of a mucous membrane of a small intestine and is soaked up in not changed look. Absorption of a heme in intestines does not depend on acidity of the environment and food factors of an inhibition. At the same time utilization of iron from cereals, fruit and vegetables significantly decreases in the presence of oxalates, phosphates and other inhibitors. Therefore coefficient of absorption of iron from beef meat – 17-22%, from dried fruits – 3%.

Iron transport in an organism.

Transferrin provides:

- delivery of iron from a GIT to erythrocytes of marrow and in fabric depots;
- iron transport in marrow from fabric depots;
- iron transport from macrophages where there is its reutilization from the collapsing erythrocytes.

Humoral regulation of an erythropoiesis is carried out by erythropoietin which is synthesized in kidneys. In anemia of 10-15% of erythropoietin it is synthesized in addition in a liver. Erythropoietin accelerates release of reticulocytes from marrow, supports a proliferative pool of erythroidal predecessors, promotes their differentiation.

Deposition of iron in an organism.

The excess amount of iron is deposited practically in all fabrics. Ferritin is most intensively laid in a liver and muscles; hemosiderin – in macrophages of marrow and parenchymatous bodies.

Losses of iron with urine, a stake, then, hair, nails are 0.1-0.3 mg/days. They increase in the pubertal period, in gastrointestinal diseases, bleedings, vasculites.

IRON DEFICIENCY (SIDEROPENIC) ANEMIA

– the morbid condition which is characterized by decrease in a hemoglobin content because of deficiency of iron in an organism as a result of disturbance of its receipt, assimilation or pathological losses.

The reasons of iron deficiency states at children:

1. Antenatal:

- disturbance of uteroplacental blood circulation, placentary insufficiency (toxicoses, threat of interruption and perenashivany pregnancies, hypoxemic syndrome, exacerbation of somatic and infectious diseases);
- fetomaterinsky and fetoplacental bleedings;
- syndrome of fetal transfusion at polycarpous pregnancy;
- pre-natal melena;
- prematurity, multiple pregnancy;
- deep and long deficiency of iron in the pregnant woman's organism.

2. Intranatal:

- fetoplacental transfusion;
- premature or late bandaging of an umbilical cord;
- intranatal bleedings as a result of traumatic obstetric grants or anomaly of development of a placenta and vessels of an umbilical cord.

3. Post-natal:

- insufficient intake of iron with food
- the increased needs for iron at children with the accelerated growth rates.
- the increased losses of iron because of bleedings of various etiology; disturbances of intestinal absorption;
- disturbances of exchange of iron in an organism because of hormonal changes, disturbance of transport of iron because of insufficient activity and (or) decrease in content of transferrin in an organism.

Development of clinical manifestations of an iron deficiency anemia is preceded **the period of latent deficiency of iron** which is characterized by decrease in the deposited iron and its transport pool. The activity of ferriferous enzymes decreases. Speed of synthesis of hemoglobin and saturation of erythrocytes by it are not broken.

Laboratory criteria of deficiency of iron.

Indicator	Norm	Latent	ZHDA
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		deficiency of iron	
OZhSS, $\mu\text{mol/l}$	44.6 - 56.8	>58.0	>58.0
KHT, %	30 - 50	<22	<20
Ferritin of blood serum, mkg/l	32 - 68	<20	<12
Free protoporphyrins of erythrocytes, mkg/l	200 - 400	>500	>600
Sideroblasts in a miyelogramma, %	20 - 90	<10	<10

Clinical manifestations of deficiency of iron are shown **by a sideropenic syndrome**:

- epithelial changes (trophic disturbances of skin, nails, hair, mucous);
- faddism of food and sense of smell;
- asthenic - vegetative disturbances;
- disturbances of processes of intestinal absorption;
- dysphagia and dyspepsic changes;
- decrease in local immunity (increase in incidence of ORZ, intestinal infections).

Decrease in level of erythrocytes in volume of blood happens at disappearance of stocks of gland. Hemoglobin synthesis decreases, concentration of protoporphyrins in erythrocytes increases. Erythrocytes gain the morphological features characteristic of ZhDA – *a microcythemia, an anisocytosis, a poikilocytosis, a hypochromia*.

In clinic the all-anemic symptoms caused by development of an anemic hypoxia join a sideropenic syndrome. Changes join from CCC – tachycardia, muting of tones, anemic systolic noise, a tendency to hypotonia, dystrophic changes on the ECG. Asthenoneurotic disturbances accrue.

ZhDA can lead to a delay of psychomotor development in children of early age. School students with low indicators of hemoglobin have indicators of intelligence and speed of reactions considerably conceded to that at healthy children. The above-mentioned changes of central nervous system connect with a depression of activity of monoaminooxidases and aldoksidaz – the enzymes playing the main role in destruction of false neurotransmitters.

Calculation **of quantity of erythrocytes** occur by two unified methods: under a microscope in the cytometer of Goryaev and by means of the automatic counter.

The color indicator reflects the relative average content of hemoglobin in erythrocytes. It is calculated empirically on a formula of "three":

$$\text{Tsv. pok.} = (N_v \times 3): \text{erythrocytes};$$

where N_v - hemoglobin in g/l; erythrocytes - the first three figures of erythrocytes without comma. Normal values – **0.85 - 1.0**.

Average volume of erythrocytes (MCV).

Is defined automatically in hematologic counters or it is calculated by a formula:

$$MCV = (Ht : Ayr) \cdot 1000,$$

where Ht – a hematocrit of the patient (%), Ayr – the first three figures of erythrocytes without comma. Normal values **75 - 100 mkm³**.

The average content of hemoglobin in an erythrocyte (MSN) – reflects an absolute hemoglobin content in one erythrocyte.

$$MSN = N_v : Ayr,$$

where N_v - hemoglobin of the patient (g/l); Ayr - the first three figures of erythrocytes from a comma. Normal values – **24-33 personal computers**.

Average concentration of hemoglobin – reflects erythrocyte saturation rate **in an erythrocyte (MSNS)** hemoglobin. Is determined automatically or by a formula:

$$MSNS = (N_v : Ht) \cdot 10$$

Normal values – **30-38%**.

Serumal iron reflects amount of the negeminovy iron which is in serum. Is defined by the unified technique with batofenantroliny.

Normal value:

newborn – 5.0 - 19.3 $\mu\text{mol/l}$;

children, are more senior 1 month – 10.6 - 33.6 $\mu\text{mol/l}$.

Indicator of reserves of iron in an organism:

Desferalovy test. It is based on ability of desferal to form connections with the iron which is a part of ferriferous proteins of a stock (hemosiderin and ferritin) and it is removed with urine in the form of complexes.

Normal daily excretion of iron according to the test with desferal is:

full-term – 0,1640,19 $\pm\text{mg/days}$;

premature – 0,0920,19 $\pm\text{mg/days}$;

up to 4 years – 0,410,03 $\pm\text{mg/days}$;

5-6 years – 0,570,09 $\pm\text{mg/days}$;

7-11 years – 0.71 ± 0.05 mg/days;

12 years – 0,730,07 $\pm\text{mg/days}$ are more senior.

Classification of anemia by severity:

Easy – Hb of 90-120 g/l; erythrocytes – to $3.5 \times 10^{12}/l$;

Moderately severe – Hb of 70-90 g/l; erythrocytes – $2.5-3.4 \times 10^{12}/l$;

Heavy – Hb less than 70 g/l; erythrocytes – less than $2.5 \times 10^{12}/l$.

Treatment of ZhDA

The basic principles of therapy of ZhDA formulated in 1981 by L.I. Idelson are program also today:

1. It is impossible to compensate deficiency of iron without medicinal ferriferous drugs.
2. Therapy of iron deficiency states has to be carried out mainly by drugs for oral administration.
3. Therapy of ZhDA should not stop after normalization of level of hemoglobin.
4. Hemotransfusions at ZhDA have to be carried out only according to vital indications.

Diet

Women of fertile age need observance of adequate food and compensation by iron preparations of the increased its losses.

When choosing food, both for pregnant women, and for children it is recommended to give preference to the products containing iron in the form of a gem (neat's tongue, meat of a rabbit, turkey, chicken, beef). From the products containing iron in the form of hemosiderin and transferrin (a liver, fish) its absorption is much less. Products from meat increase absorption of iron from vegetables and fruit at simultaneous use. The soy protein, polyphenols reduce digestion of negemovy iron (tea, coffee, nuts, bean).

From vegetable products the bigger amount of iron contains in a sea cabbage (16 mg./100 gr.), fresh dogrose (11.5 mg.), buckwheat, oat-flakes (7.8 mg.), pear, apples, apricot (2.3 mg.). At a vegetarian diet no more than 17% of iron are acquired.

The full-fledged and balanced diet allows to cover only the physiological need for iron, but not to eliminate deficit. Therefore in treatment of ZhDA ferriferous medicines are surely appointed.

At ZhDA the contents in an organism of vitamins of group B and their metabolism is not violated. This group of vitamins initiates synthesis of porphyrines which at ZhDA is accelerated. Therefore use of these vitamins B of therapy of iron deficiency states is not justified.

Iron preparations.

Advantage of oral administration of iron preparations is:

1. Oral administration increases hemoglobin level only for 2-4 days later, than parenteral administration.
2. Oral administration extremely seldom, unlike parenteral, results in side effects.
3. Oral administration at wrong interpretation of anemia as iron deficiency does not lead to development of a hemosiderosis.

The greatest number of active iron contains in drugs with fumaraty gland (33% of the general content of iron in drug), ferrous sulfate (20%), an iron gluconate (12%).

Distinguish:

1. **Monocomponent** drugs – Haemoferum, ferrograd, a ferrogradumet.
2. **The combined** drugs:
 - a) gland sulfate + serine (Aktiferrin);
 - b) gland sulfate + vitamin C (Ferroplexum, Ferrograd With, Sorbifer Durules, Ferroplekt);
 - c) gland sulfate + vitamin C + mukoproteaza (Tardiferon);
 - d) gland sulfate + vitamin C + mukoproteaza + folic acid (Ginotardiferon);
 - e) gland sulfate + folic acid (Ferrograd folik, Fefol).

Besides, there are numerous vitamin drugs as a part of which the amount of iron exceeds a dose of daily physiological requirement.

Recently the drugs containing in the basic bivalent iron salts are used. Utilization of trivalent iron is strictly limited by level in gastric juice.

Inclusion in composition of ascorbic acid along with increase in absorption of iron contributes to more frequent development of side effects of a ferroterapiya.

The phytin included in complex drugs for stimulation of exchange processes worsens iron absorption.

Wide use of syrup of an aloe with iron therapeutic is a little effective since in 5 ml. drug only 2.5 mg contain. active iron and often the dispepsichesky phenomena develop.

At children of early age the prescribing of ferriferous drugs in a liquid form – drops and syrup is preferable (Haemoferum, Maltofer, Aktiferrin).

At disturbance of a microbiocenosis of intestines the activation of gram-negative opportunistic siderophile flora and development of dispepsichesky disturbances is possible. To these children are justified together with iron preparations to use eubiotik.

For children of advanced age it is better to appoint Tardiferona drugs and Ferrogradumet. They are slowly soaked up, provide the prolonged and uniform absorption of iron in intestines, are well transferred.

It is reasonable to appoint iron preparations in 1-2 hours prior to or after a meal. At bad shipping appoint during meal, but iron absorption at the same time worsens. It is impossible to wash down ferriferous drugs with tea, milk because of decrease in efficiency of digestion of iron.

Some medicines – tetracyclines, levomycetinum, antacids, calcium drugs break iron absorption.

For definition of a necessary medical dose of drug the calculation it is carried out only on elementary (active) iron.

Children up to 3 years have **5-8 mg/kg/days** of elementary iron.

Children up to 7 years have **100-120 mg/days**.

At children 7 years are more senior – to **200 mg/kg/days**.

So, **Haemoferum** contains in 1 drop 7.8 mg. ferric chloride and 2.2 mg. elementary;
aktiferrin in 1 drop of 47.2 mg. ferrous sulfate and 9.8 mg. elementary;
tardiferon in 1 tab. - 256 mg. ferrous sulfate and 80 mg. elementary;
ferro-gradument in 1 tab. - 525 mg. ferrous sulfate and 105 mg. elementary.

Daily amount of drug = a therapeutic daily dose of elementary iron / quantity of elementary iron in drug.

The starting dose is equal 1/2 - 1/4 from a full dose, therapeutic with the subsequent achievement, within 7-14 days. It is necessary for assessment of individual tolerance of drug the child and reduction of risk of side effects.

The therapeutic effect is shown gradually. In the beginning clinical improvement, then hemoglobin normalization is noted. From clinical symptoms muscle weakness disappears in the beginning (iron is a part of the enzymes participating in reduction of myofibrils). For 8-12 day of treatment the maintenance of reticulocytes in peripheral blood increases. Normalization of hemoglobin happens on 4-5 week of treatment.

After achievement of normal level of hemoglobin it is reasonable to continue a ferroterapiya within 2-3 months the daily dosage decreases on 1/2 from a therapeutic dose.

At premature the ferroterapiya in a maintenance dose is carried out until the end of the 2nd year of life.

Indications to parenteral administration of iron preparations:

1. states after a resection of a stomach, a small intestine;
2. syndrome of the broken absorption;
3. nonspecific ulcer colitis;
4. chronic coloenteritis.

Daily doses of drugs for parenteral iron

Age	Daily dose of elementary iron of mg/days.
1 - 12 months.	to 25
1 - 3 years	25 - 40
3 years are more senior	40 - 45

Course dose of elementary iron (mg) = MT (kg) x (78 - 0.35 x child's Hb).

Course dose of drug on a course (ml) = KJ: SZhP, where KZhD – a course dose of iron (mg); SZhP - the content of iron (mg) in 1 ml. drug.

Quantity of injections on a course = KDP: SDP, where KDP - a course dose of drug (ml); SDP – a daily dose of drug (ml).

Parenterally the drug is administered with an interval of 1-2 days.

Side effects of ferriferous drugs: skin itching, dermahemia, allergic dermatitis, nausea, vomiting, loss of appetite, diarrhea.

Hemotransfusions at ZhDA are carried out only according to vital indications. Preference is given to a hemotransfusion of a packed red cells or the washed erythrocytes in a dose of 10-15 ml/kg of body weight; for children of advanced age from 150 to 250 ml.

Indications to a hemotransfusion:

a) Critical level of Nv in g/l:

The 1st day of life <130

The 2-6th day of life - heavy respiratory disturbances <130

- heavy disturbances of a hemodynamics <110-120

- without disturbance of breath and CCC <100

The 7-28th day of life - with disturbances of breath and CCC <100

- without disturbance of breath and CCC <80

Is more senior than one month <60

b) Disturbance of the central hemodynamics, hemorrhagic shock, anemic prekoma, hypoxemic syndrome.

Scheme of inspection of the patient:

When collecting the anamnesis to pay attention on:

- genetic predisposition;
- presence of anemia at mother during pregnancy;

- features of the obstetric and gynecologic anamnesis of mother;
- features of the perinatal period;
- defects of food of the child;
- symptoms of a disease: their character, terms of emergence, loudspeaker.

At an objective research to pay attention on:

- condition of weight of the child;
- color of integuments and visible mucous;
- condition of a cardiovascular system;
- sizes of a liver and spleen, lymph nodes;
- condition of other bodies and systems.

At interpretation of datas of laboratory to pay attention on:

- characteristic of an erythrocyte system: quantity, form, size, coloring of erythrocytes;
- amount of hemoglobin, contents and concentration of hemoglobin in an erythrocyte;
- gematokritny number;
- quantity of reticulocytes, thrombocytes;
- existence of young forms;
- biochemical analysis of blood (content of serumal iron, general iron-binding ability of serum (GIBAS), LZhSS saturation level transferrin).

Tasks for independent work:

1. Solve situational problems and test control tasks.
2. Write in a workbook prescriptions on drugs for treatment of scarce anemia.

Situational tasks.

Task No. 1

The girl of 10 months came to clinic with complaints to pallor, a loss of appetite, slackness. Was born full-term (body weight 3300 gr., length of 49 cm), from the first pregnancy proceeding with toxicosis. From 3.5 months was on artificial feeding, practical did not receive fruit and berry juice, from 5 months it is raised mainly by porridges. Prevention of rickets was not carried out. Began to hold the head from 4 months, to sit from 9 months, the first teeth were cut through in 8 months, only 4 teeth.

At survey: pallor, tearfulness, a hypomyotonia, a stomach it is increased in volume, the umbilical ring is expanded, frontal and occipital hillocks act, at a palpation of a thorax costal

beats are defined. Pulse of 142 beats/min., symmetric, rhythmical, satisfactory filling. The upper bound of warm dullness at the level of II edges, right – the right sternal line, left – the left mamillar line. Cardiac sounds are moderately muffled, on a top gentle systolic noise. The liver acts from under edge of a costal arch on 3 cm, edge equal, smooth. The spleen is not palpated.

Complete blood count test: Ayr – $2,8 \cdot 10^{12}/l$, Hb – 76 g/l, Ley – $13,2 \cdot 10^9/l$, Tsv. pok. – 0.6, reticulocytes – 2.8%, with / I am 29%, p/ya-2%, l/c of 57%, m/c of 8%, e/f 1%.

Questions:

1. Your diagnosis?
2. What possibilities of a cause of illness and risk factors?
3. Offer the plan of additional inspection, a balanced diet and medicamentous therapy.

Task No. 2

The girl of 14 years came to clinic with complaints to weakness, dizziness, periodically arising pains in epigastric area for 3 years. At first the tendency to constipations was noted, then – to a diarrhea. Recently the language sizes increased, there were sites of reddening, the appetite decreased.

Moderately severe state, sluggish, emotionally labile. Subnutrition, astenik. Integuments pale with a citreous shade, a subjikterichnost of scleras, mucous clean. Brightly red sites of inflammation are always on the lips. Pulse of 94 beats/min., rhythmical, satisfactory filling. Cardiac sounds are muffled. A stomach painful at a palpation in epigastric area. A liver at edge of a costal arch. The spleen is not increased.

Complete blood count test: Ayr – $3,2 \cdot 10^{12}/l$, Hb – 130 g/l, Tsv. the item – 1.1, Ley – $4,0 \cdot 10^9/l$, e/f – 1%, p.b. – 2%, with / I am 35%, l/c – 55%, m/c – 5%, SOE – 8 mm/h.

In a smear: reticulocytes ++, schizocytes ++, Kebbot's rings ++, Howell-Jolly bodies ++, a significant amount of neutrophils with the polysegmented kernels.

Level of serumal iron is 750 mkg/l, the content of vitamin B₁₂ is 10 mkg/ml.

Result of a fibrogastroskopichesky research – symptoms of atrophic gastritis.

Questions:

1. Your presumable diagnosis?
2. What causes and mechanisms of a course of a disease?
3. Appoint treatment.

Test control:

1. The iron deficiency anemia is characterized by decrease:
 - a) hemoglobin

- b) color indicator
 - c) hematocrit
 - d) quantities of erythrocytes
 - e) quantities of reticulocytes
2. Most intensively iron collects in a fruit organism:
- a) the first trimester of pregnancy
 - b) the second trimester of pregnancy
 - c) the third trimester of pregnancy
3. The iron deficiency anemia on the saturation rate of erythrocytes hemoglobin is:
- a) normochromic
 - b) hyperchromic
 - c) hypochromia
4. A marrowy hemopoiesis at ZhDA is characterized:
- a) hypoplasia
 - b) aplasia
 - c) tension of an erythrogenesis with the advent of a reticulocytosis in peripheral blood.
5. At ZhDA the increase comes to light:
- a) transferrin saturation percent
 - b) level of serumal iron
 - c) ferritin level in blood serum
 - d) concentration of hemoglobin in an erythrocyte
 - e) iron-binding ability of blood serum
6. Are the reasons of development of iron deficiency anemias in children all listed below
EXCEPT:
- a) alimentary
 - b) sprue
 - c) infectious diseases
 - d) marrow aplasia
 - e) the increased need of an organism of the child for iron during certain age periods
 - e) chronic blood losses
7. The principles of treatment of ZhDA are:
- a) replacement therapy by blood preparations
 - b) vitamin therapy by vitamin C

- c) vitamin therapy by group B vitamins
 - d) use of the products rich with iron
 - e) prescribing of iron preparations
 - e) glucocorticoid therapy
8. What products does the child need to appoint with an iron deficiency anemia?
- a) milk
 - b) rice
 - c) meat
 - d) fermented milk products
 - e) buckwheat cereal
 - e) fruit
9. ¹²Is the reason of V-scarce anemia more often:
- a) blood loss
 - b) helminthic invasion
 - c) disturbance of secretion of an internal factor of Kastl or disturbance of absorption
 - d) insufficient intake of vitamin B₁₂ with food
10. At deficiency of folic acid anemia:
- a) hypochromia
 - b) normochromic
 - c) hyperchromic
11. V-scarce ¹²anemia is characterized:
- a) microcytic anemia
 - b) megaloblastny type of a hemopoiesis
 - c) decrease in number of reticulocytes
 - d) hyperchromic anemia
 - e) increase in serumal iron

Class in a subject:
"CHRONIC DISORDERS OF FOOD"

I. Scientific and methodical justification of a subject.

Chronic disorders of food are frequent pathology of children of early age, reducing body resistance and increasing incidence of children of early age. In this regard it is important to know the reasons, prevention and treatment of this pathology. It is also necessary to consider that the

foundation in development of heavy degrees of obesity in adults is quite often laid on the first year of life. In this regard knowledge of this problem is important for doctors of endocrinologists, therapists.

II. Purpose of activity of students.

The student has to know:

- definition of a normotrofiya;
- classification of chronic disorders of food;
- etiology of chronic disorders of food;
- disturbances in a metabolism at various forms chronic disorders of food;
- clinical manifestations and diagnostic methods in chronic disorders of food;
- basic principles of treatment and prevention of chronic disorders of food;
- value of chronic disorders of food in pathology of children of early age.

The student has to be able:

- to purposefully collect the anamnesis, allocating from it the factors of internal and external environment contributing to the development of chronic disorders of food;
- to conduct anthropometry and an objective research of the child;
- to make the diagnosis according to classification;
- to make the treatment plan of the child;
- to write prescriptions on the main medicines;
- to define preventive actions.

III. Content of training:

1. The factors of external and internal environment contributing to development of chronic disorders of food
2. Clinical forms of chronic disorders of food.
3. Role of chronic disorders of food in pathology of children of early age.
4. Hypotrophy (definition, etiology, pathogenesis, clinic, diagnostics, prevention, treatment).
5. Paratrofiya (definition, etiology, pathogenesis, clinic, diagnostics, prevention, treatment).
6. Obesity (definition, etiopathogenesis, clinic, diagnostics, prevention, treatment).

IV. Educational material security.

1. Visual aids: tables, schemes, multimedia presentations, videos, audiogramma.
2. Educational medical documentation (case histories, laboratory researches,

roentgenograms).

3. Technical means of training.
4. Literature.

V. The list of the recommended literature.

1. Children's diseases: the textbook / under the editorship of A.A. Baranov. – M.: GEOTAR-media, 2009. – 1008 pages.
2. Pediatrics: The textbook for medical schools. Under the editorship of N.P. Shabalov. – SPb: SpetsLit, 2006. – 895 pages.
3. Propaedeutics of children's diseases / to N.A. Geppa. – M.: GEOTAR-media, 2009. – 464 pages.
4. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Background diseases of children of early age. Manual for students. – Vladikavkaz, 2011. – 64 pages.
5. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Technique of a research of the child. The study guide for students. – Vladikavkaz, 2011. – 51 pages.
6. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Scheme of a case history. The study guide for students. – Vladikavkaz, 2011. – 38 pages.
7. Lectures on pediatrics.
8. Methodical instructions for out-of-class work of students 4 courses of medical faculty on discipline "Pediatrics".

VI. List of questions for check of initial level of knowledge:

1. Features of the structure of a digestive tract at children.
2. Features of process of digestion at children.
3. Types of feeding at children of the first year of life.
4. What is a hypogalactia? Reasons of its emergence?
5. Types of obesity, development reason.

VII. List of questions for check of final level of knowledge:

1. Give definition of a concept a normotrofiya.
2. List the factors of external and internal environment contributing to development of chronic disorders of food
3. Give the reasons for a congenital hypotrophy.
4. Call clinical forms of chronic disorders of food.

5. List the main disturbances in a metabolism at: a) hypotrophy; b) paratrofiya; c) obesity.
6. What role of chronic disorders of food in pathology of children of early age?
7. Call changes from blood in a hypotrophy.
8. Call the basic principles of treatment: a) hypotrophy; b) paratrofiya; c) obesity.
9. Call the main preventive actions of chronic disorders of food.

Information block.

DYSTROPHIES at children represent the chronic disorders of food connected with the insufficient or excess intake of nutrients, disturbances of their assimilation and a metabolism conducting to a delay of physical and psychological development. Dystrophic states weaken the child's organism, contributing to developing of acute and chronic bacterial and other diseases. The children having dystrophies have to be carried in risk groups on many diseases.

In our country, according to G.N. Speransky's proposal (1945), 3 main types of dystrophy at children are allocated: *hypotrophy, gipostatura and paratrofiya*.

Is later than G.I. Zaytsev, L.A. Stroganov (1969, 1981) considerably added this classification at the expense of discharge of severity (I, II, III), the disease periods (initial, progressing, stabilization, a convalescence), origins (prenatal, post-natal, prenatal and post-natal) and indications of the reasons (exogenous and endogenous).

HYPOTROPHY

The chronic disorder of food resulting from starvation of an organism when there is no opportunity to compensate the expenses spent for vital processes. The deficiency of feedstuffs can be caused by insufficient receipt them with food or to be result of disturbance of assimilation and utilization of food. The hypotrophy develops at children of the first 2 years of life more often.

Etiology and pathogenesis. Development of a hypotrophy is the cornerstone alimentary, infectious and constitutional factors. From alimentary factors, first of all, it is necessary to allocate disturbances of feeding of quantitative or qualitative character. Insufficient receipt of food can be connected with the reasons depending on mother (a hypogalactia, defect of chest glands – "a hard breast", the flat or pulled-in nipples, etc.) and from the child (an underdevelopment of a sucking reflex at premature children, the birth trauma, defects of development and ugliness complicating suction: splitting of a lip, hard palate, pylorostenosis, etc.).

The hypotrophy can develop also from disturbance of qualitative structure of a diet, at the wrong ratio of proteins, fats and carbohydrates, insufficient receipt any of the main food ingredients (proteins, vitamins). Leaving disturbance, chaotic feeding, insufficient input of the

fluid, etc. are of great importance. The hypotrophy arises at the children who are on the mixed and artificial feeding more often.

Diseases of the child, especially acute and persistent infections (intestinal, respiratory and virus, pyoinflammatory, etc.), influence of a number of toxic factors (hypervitaminoses, medicinal poisonings), congenital pathology (mucoviscidosis, a Gee's disease, an enzymopathy) can also be the causes of a hypotrophy. The most frequent cause of a hypotrophy at children of early age – intolerance of proteins of cow's milk, gluten (protein of cereals). The hypotrophy can develop as a result of some hereditary endocrine, immunodeficient diseases, damages of the central nervous system.

The hypotrophy can have congenital character, the disease of mother, a fetopathy, defects of pre-natal development can be its reason.

In development of a hypotrophy the decrease in secretory function of digestive glands and evacuation of food is of great importance. Reduction of secretion of digestive glands leads to disturbance of processes of absorption and decrease in comprehensibility of feedstuffs. Band and pristenochny digestion is broken, dysbacteriosis develops. Special changes are undergone by protein metabolism, there is a disintegration of fabric protein. The hypoproteinemia, disturbance of a normal ratio of protein fractions, the increased removal of amino acids with urine, negative nitrogenous balance are noted. There is a disappearance of stocks of a glycogen, fat, mineral substances, standard metabolism changes, exhaustion develops.

Clinical picture. The leading clinical symptoms in a hypotrophy are increase delay, a stop of an increase or decrease in body weight. Depending on deficit of body weight distinguish a hypotrophy of the I degree (deficit of the body weight of 10-20%), the II degrees (deficit of the body weight of 20-30%) and the III degrees (deficit of body weight more than 30%).

In a hypotrophy of the I degree the general condition of the child remains satisfactory, coloring of integuments is a little pale, the hypodermic fat layer decreases, the elasticity of skin decreases a little.

In a hypotrophy of the II degree along with lag in body weight occur lag in growth (from 2 to 3-4 cm); the hypodermic fat layer disappears on a trunk and extremities; skin loses elasticity, become dry, easily gathers in folds, on its certain sites there can be a peeling, pigmentation, hair become more rigid and rare, the turgor of fabrics considerably goes down, develops a hypomyotonia.

Disturbances of activity of a number of bodies and systems are characteristic of a hypotrophy of the III degree, besides sharper exhaustion: eyes sink down, the person takes a

senile form, wrinkled, skin dry, shelled, with pigmentation, easily gathers pleated and long does not finish, mucous membranes dry, bright, vulnerable that often leads to development of a thrush, stomatitis. There is a hyperexcitability, nervousness or apathy, slackness, a dream uneasy, the appetite is reduced. Development of motor functions (children begin to sit, stand, go later) lags behind or they are lost. Muscles become atrofichny. Changes from a cardiovascular system are noted: dullness of cardiac sounds, the slowed-down pulse, a lowering of arterial pressure. Thermal control, extremities constantly cold is broken. The stomach is involved or blown up, an atony of intestines and a meteorism. Anemia develops.

At children with a hypotrophy the reactivity is lowered, they are ill more often, are susceptible to infections; diseases at them quite often accept difficult character, often there are complications.

Diagnostics. The diagnosis is made on the basis of assessment of clinical symptoms, lag in body weight.

Treatment. Consider a disease etiology, degree of exhaustion and presence of associated diseases. Treatment has to be complex, include correction or elimination of an etiological factor, a dietotherapy, the rational mode, careful leaving, detection and treatment of the centers of an infection, rickets, anemia and other associated diseases.

At all forms of a hypotrophy particular importance is attached to food. It has to be constructed correctly, taking into account age need of the child for the main feedstuffs and his physiological opportunities. The proper correlation of the main food ingredients has positive impact on normalization of the broken exchange processes and also on physical and psychological development. In a hypotrophy of the I degree often happens to eliminate enough defect of feeding of the child, to adjust the mode and leaving then the deficit of body weight is quickly liquidated. To children with a hypotrophy of II and especially the III degrees, often suffering from the lowered tolerance to food, in the first days of treatment reduce the daily volume of food to $\frac{3}{4}$, $\frac{2}{3}$ and even up to $\frac{1}{3}$ from the relying norm (depending on a condition of the patient).

The missing volume of food is filled with liquid (tea, dogrose infusion, glucose solution, fruit juice, vegetable and fruit broths). At improvement of a condition of the child the amount of food is gradually brought to physiological norm.

Purpose of food to the child having a hypotrophy and also all changes in food have to be made under control of calculations of the chemical composition of a diet. In a hypotrophy of the I degree the calculations and correction of food, as a rule, are made on 1 kg. the must body weight

which consists of the body weight of the child at the birth and average norms of increases of body weight for the lived life period. In a hypotrophy of II and III degree when disturbances of exchange processes are significant, and, therefore, and digestion of the main feedstuffs, especially fat, is required accurate individualization of dietary actions. In a hypotrophy of the II degree the amount of proteins and carbohydrates in a daily diet is expected 1 kg. the must body weight, and amount of fats — on 1 kg. the actual body weight or at rather satisfactory condition of the child — on the body weight which is average size between actual and forced. In a hypotrophy of the III degree the necessary amount of proteins and carbohydrates is expected 1 kg. approximately must body weight (the actual body weight + 20% of the actual body weight). The amount of fats is calculated only on the actual body weight as the tolerance to fat at these children is sharply reduced. The specified calculations cannot use in a hypotrophy with the profound toxicosis. In this case it is necessary to eliminate the phenomena of intoxication and in the subsequent it is very careful, observing the principle of gradualness, to increase amount of ingredients and to expand a diet of the child.

In process of normalization of a condition of the child, permanent increase of body weight the food is calculated on the must weight. First of all korrigitur proteinaceous and carbohydrate structure of a diet and in the last — fat. In the period of a reparation the amount of carbohydrates can be slightly higher than physiological norm as in connection with restriction of fats they represent the main source of energy.

It is important that the nutrition of the children having a hypotrophy was good. It is desirable to provide children of the first months of life with breast milk (in the absence of milk at mother — donor with the corresponding correction). At artificial feeding prefer as the mixes adapted milk and sour-milk. Fermented milk products stimulate production of digestive juices, reduce the dysbacteriosis phenomena, are easier digested and acquired by the child's organism.

For restoration of normal intestinal microflora it is recommended to use the biological products containing natural protective factors: bifidobacteria, lysozyme, lactobacilli.

It is reasonable to carry out correction of a proteinaceous component at the expense of natural products (cottage cheese, a yolk, meat mash) and also specialized canned meat.

Correction of a carbohydrate component is carried out by means of sugar syrup, fruit juice and puree. The fat structure of a diet is recommended to be expanded at full adaptation of the child to the protein arriving with food by inclusion in food of creamy and vegetable oil.

The feeding up to children with a hypotrophy is usually appointed carefully, against the background of positive dynamics of body weight, in the absence of acute diseases. As the first

feeding up milk porridge from various grain is entered, then in 1-1.5 weeks appoint the second feeding up in the form of vegetable puree. All types of a feeding up enter gradually, since small doses, and within 7-10 days bring to necessary volume.

At all forms of a hypotrophy to children appoint a complex of vitamins B to a medical age dosage. In a stage of metabolic adaptation fermental drugs are shown (solution of hydrochloric acid with pepsin, Pancreatinum, panzinorm, festal, abomin, etc.). The apilak is applied to stimulation of trophic functions. In the heavy hypotrophy which is badly responding to treatment the hormonal therapy (retabolil) is shown.

Along with medicamentous therapy perform massage and gymnastics. The great value is attached to child care and a raising of its emotional tone.

Prevention has to include rational feeding, the organization of the correct mode, sufficient use of fresh air, careful leaving, physical training and hardening. Also periodic control of increase of body weight, carrying out calculations of food and its timely correction are of great importance.

Tasks for independent work:

1. Solve independently tasks of test control.
2. Examine the patient with chronic disorders of food, using the scheme below. Describe the changes in the state of health of your patient revealed by you in a workbook.
3. Solve situational problems.
4. Write prescriptions in a workbook:
 - a) festal
 - b) mezy forte
 - c) abomin
 - d) apilak
 - e) motilium

Scheme of inspection of the patient.

When collecting the anamnesis pay attention on:

- pregnancy course at mother;
- the body weight of the child at the birth;
- the nature of feeding of the child since the birth;
- existence of a hypogalactia at mother;
- timeliness and sequence of introduction of a feeding up;

- to the loudspeaker of an increase of body weight;
- the diseases transferred the child;
- features of child care.

At objective survey to pay attention on:

- color of integuments;
- expressiveness of a hypodermic and fat layer;
- turgor of soft tissues;
- elasticity of skin;
- presence of symptoms of exudative diathesis;
- condition of a muscle tone;
- deficit or surplus of body weight as a percentage;
- presence of symptoms of rickets;
- timeliness of development of static functions;
- psychological development of the child;
- changes from internals;
- appetite of the child;
- nature of a chair.

At interpretation of laboratory analyses to pay attention on:

- complete blood count test (maintenance of erythrocytes, Hb, leukocytes, eosinophils);
- biochemical analysis of blood (protein content and protein fractions);
- clinical analysis of urine.

Situational tasks.

Task No. 1

Mother of the girl of 1 month on reception at the pediatrician with complaints to the increased concern of the child remaining after feeding between feedings maintains no more than 1 hour.

From the anamnesis it is found out that the girl was born from the first pregnancy, physiological childbirth. Body weight at the birth 3050 gr., body length – 49 cm. Is on breastfeeding.

At objective survey: body temperature 36.5°C, body weight 3150 gr., length of a body is 51 cm. Skin with hyperaemia in inguinal and axillary folds. Hypodermic fatty tissue is thinned:

thickness of a skin fold at the level of a navel – 0.5 cm, breasts – 0.5 cm, shoulders – 1 cm, a hip – 1.5 cm. Internals without pathology. A chair dense gruel 1-2 times a day. About 10 times a day urinate. The angry shout, emotional, greedy is enough a pacifier and a horn from 5% with glucose. The neurologic status within norm.

Questions:

1. Make the preliminary diagnosis?
2. What development of this state is connected with?
3. Appoint treatment.

Task No. 2

On reception girl of 1.5 months. Mother shows complaints to the frequent vomiting of the child arising both right after food and through some time after feeding and also before meal. The volume of the belched masses non-constant (from 1-2 teaspoons before vomiting by "fountain"), belches stvorozhenny milk more often. Eats greedy, a chair of usual consistence, without pathological impurity, a diuresis sufficient. The body weight increase for the first month of life made 650 gr., in 2 weeks of the second – 300 gr.

From the anamnesis it is known that the girl from the II pregnancy proceeding with toxicosis, interruption threat, childbirth was prompt. The girl is observed by the neuropathologist concerning perinatal encephalopathy, hyperexcitability. Belches from the first days of life.

Questions:

1. Make the preliminary diagnosis.
2. What development of this state is connected with?
3. Appoint treatment.

Test control:

1. Duration of the period of determination of tolerance to food in a hypotrophy of the I degree is:
 - a) 1-2 days
 - b) 3-7 days
 - c) 10 days
 - d) up to 14 days
2. Duration of the period of determination of tolerance to food in a hypotrophy of the II degree is:
 - a) 1-2 days
 - b) 3-7 days
 - c) 10 days

- d) up to 14 days
3. Duration of the period of determination of tolerance to food in a hypotrophy of the III degree is:
- a) 1-2 days
 - b) 3-7 days
 - c) 10 days
 - d) up to 14 days
4. The deficit of body weight at the I degree of a post-natal hypotrophy is:
- a) 5-8%
 - b) 5-15%
 - c) 10-20%
 - d) 20-30%
5. The deficit of body weight at the II degree of a post-natal hypotrophy is:
- a) 5-8%
 - b) 5-15%
 - c) 10-20%
 - d) 20-30%
6. The deficit of body weight at the III degree of a post-natal hypotrophy is:
- a) 5-15%
 - b) 10-20%
 - c) 20-30%
 - d) more than 30%
7. States belong to a paratrofiya of the I article with:
- a) deficit of weight more than 10%
 - b) surplus of weight from 5% to 10%
 - c) surplus of weight from 10% to 20%
 - d) surplus of weight and height more than 10%
8. Food volume in a post-natal hypotrophy of the I degree during determination of tolerance to food is:
- a) $\frac{2}{3}$ from norm
 - b) $\frac{1}{2}$ from norm
 - c) $\frac{1}{3}$ from norm

9. Food volume in a post-natal hypotrophy of the II degree during determination of tolerance to food is:
- a) $\frac{2}{3}$ from norm
 - b) $\frac{1}{2}$ from norm
 - c) $\frac{1}{3}$ from norm
10. Food volume in a post-natal hypotrophy of the III degree during determination of tolerance to food is:
- a) $\frac{2}{3}$ from norm
 - b) $\frac{1}{2}$ from norm
 - c) $\frac{1}{3}$ from norm
11. Uniform significant deficit of weight and growth is called:
- a) paratrofiya
 - b) hypotrophy
 - c) gipostatura
12. Exchange disturbance is a basic reason of oppression of immune responsiveness in a hypotrophy:
- a) proteins
 - b) fats
 - c) carbohydrates
13. Enzymatic drugs and anabolic hormones are shown at treatment of a hypotrophy:
- a) are not shown
 - b) The I degrees
 - c) The II degrees
 - d) The III degrees
14. Can lead to developing of a post-natal hypotrophy:
- a) alimentary factors
 - b) infectious diseases
 - c) untimely vaccinal prevention
 - d) genetic factors
 - e) diabetes at mother
 - e) iron deficiency anemia

Class in a subject:
"ANOMALIES OF THE CONSTITUTION"

I. Scientific and methodical justification of a subject.

Anomalies of the constitution are widespread pathology of children of early age in which forming the big role belongs to heredity and environmental factors. Existence at the child of one of diathesis contributes it to diseases of the respiratory organs, digestive tract, nervous and urinary system getting a heavy and long course.

At the senior children and adults who had diathesis at early age the bent to allergic diseases (urticaria, a Quincke's edema, bronchial asthma, etc.), urolithic, cholelithiasis, gout, etc. to serious diseases is also noted. In this regard acquaintance to anomalies of the constitution is important not only for pediatricians, but also for obstetricians-gynecologists, therapists, allergists, dermatologists.

II. Purpose of activity of students.

The student has to know:

- determination of the anomaly of the constitution (AC);
- forms of joint stock company;
- role of factors of external and internal environment in forming of joint stock company;
- symptomatology and clinical forms of exudative and catarrhal diathesis;
- clinical manifestations of limfatiko-hypoplastic diathesis;
- clinical manifestations of neuroarthritic diathesis;
- changes from internals at anomalies of the constitution;
- changes from blood at joint stock company;
- role of joint stock company in pathology of early and advanced children's age;
- treatment and prevention of different types of joint stock company.

The student has to be able:

- to purposefully collect the anamnesis, allocating from it the factors contributing to the development of joint stock company;
- to examine children with joint stock company;
- to appoint the mode, food and drug treatment to the child with joint stock company taking into account etiological factors and a clinical picture of a disease;
- to make recommendations for preventive inoculations.

III. Content of training:

1. Concept about anomaly of the constitution.
2. The environmental factors influencing forming of exudative and catarrhal diathesis.

3. Forms of exudative and catarrhal diathesis.
4. Clinic, laboratory diagnostics, treatment (the basic principles of the mode, food, list methods of medicinal therapy) exudative and catarrhal diathesis.
5. Limfatiko-gipoplastichesky diathesis (definition, etiology, pathogenesis, clinical picture).
6. Laboratory diagnostics (changes from blood), treatment of limfatiko-hypoplastic diathesis.
7. Neuroarthritic diathesis (general characteristic, etiology, pathogenesis).
8. Clinical picture, influence of neuroarthritic diathesis on development of mentality of the child.
9. Laboratory diagnostics, characteristic changes from blood.
10. Methods of medicinal therapy, tactics of the doctor when holding preventive actions in neuroarthritic diathesis.

IV. Educational material security.

1. Visual aids: tables, schemes, multimedia presentations, videos, audiogramma.
2. Educational medical documentation (case histories, laboratory researches, roentgenograms).
3. Technical means of training.
4. Literature.

V. The list of the recommended literature.

1. Children's diseases: the textbook / under the editorship of A.A. Baranov. – M.: GEOTAR-media, 2009. – 1008 pages.
2. Pediatrics: The textbook for medical schools. Under the editorship of N.P. Shabalov. – SPb: SpetsLit, 2006. – 895 pages.
3. Propaedeutics of children's diseases / to N.A. Geppa. – M.: GEOTAR-media, 2009. – 464 pages.
4. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Background diseases of children of early age. Manual for students. – Vladikavkaz, 2011. – 64 pages.
5. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Technique of a research of the child. The study guide for students. – Vladikavkaz, 2011. – 51 pages.
6. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Scheme of a case history. The study guide for students. – Vladikavkaz, 2011. – 38 pages.
7. Lectures on pediatrics.

8. Methodical instructions for out-of-class work of students 4 courses of medical faculty on discipline "Pediatrics".

VI. List of questions for check of initial level of knowledge:

1. List the main anatomico-physiological features of skin, hypodermic fatty tissue at children.
2. What features of nervous system of children of younger age do you know?
3. Call features of exchange at children.
4. List the main features of the immune system at children's age.

VII. List of questions for check of final level of knowledge:

1. Give definition to a concept of "anomaly of the constitution".
2. Call forms of diathesis.
3. What role of heredity in forming of joint stock company?
4. List the environmental factors influencing forming of exudative and catarrhal diathesis.
5. Call forms of exudative and catarrhal diathesis.
6. List the main clinical laboratory diagnostic criteria of exudative and catarrhal diathesis.
7. Call the basic principles of the mode, food, methods of medicinal therapy of exudative and catarrhal diathesis.
8. Characterize the main etiopatogenetichesky mechanisms of limfatiko-hypoplastic diathesis.
9. Give the description of the main clinical symptoms of limfatiko-hypoplastic diathesis.
10. Laboratory diagnostics (changes from blood), treatment of limfatiko-hypoplastic diathesis.
11. General characteristic of neuroarthritic diathesis, etiopatogenetichesky mechanisms.
12. Development of mentality of the child against the background of neuroarthritic diathesis.
13. Laboratory diagnostics, characteristic changes from blood.
14. List methods of medicinal therapy, preventive actions in neuroarthritic diathesis.

Information block.

DIATHESIS – states at which the organism of the child answers usual external irritants not with the absolutely proper responses contributing to development of some diseases. Therefore diathesis is considered as the borderline cases which are formed against the background of hereditary features of an organism, lag in maturing of a number of structural and functional systems and also in formation of neurohumoral regulation. With age at the correct organization of leaving, the mode, food and education of manifestation of diathesis at children almost disappear. However and at mature age certain constitutional features of the human body which transferred

diathesis in the childhood remain, and the predisposition to developing of any given pathology is to a certain extent also connected with them.

LIMFATIKO-GIPOPLASTICHESKIY DIATHESIS

– a state at which decrease in adaptation reactions of the chromaffin device of adrenal glands to influence of various vneshtresredovy factors is observed. At such children the hyperplasia of adenoid tissue at a simultaneous relative hypoplasia of some closed glands and internals is noted. Abnormal development of a thymus and a deviation in forming of cell-mediated and antibody-mediated immunity quite often are found in them. Meets at early and preschool age more often.

Clinical picture. Externally children look pale, pastose, body weight at them exceeds average age values. Friable hypodermic fatty tissue, more often externally excess, muscular system is expressed poorly. A trunk rather short, the extremities extended, a thorax it is narrowed. Big, friable tonsils. Often considerable adenoid growths in this connection, nasal breath is at a loss are found and a peculiar adenoid face type is formed. The numerous bulked-up hypodermic lymph nodes are probed. The thymus is increased that can be established at thorax percussion, by means of a X-ray analysis and ultrasonography. The moderate leukocytosis, often an eosinophilia, a lymphocytosis and a monocytosis are characteristic of a picture of blood. The children having limfatiko-hypoplastic diathesis are inclined to slow inflammatory diseases of mucous membranes (rhinitises, nasopharyngites, a tracheobronchitis, otitises, conjunctivitis).

At them quite often during inflammatory diseases of upper airways the obstructive syndrome develops. During an acute respiratory infection, flu, at stressful situations there can be a syndrome of sudden death. Earlier such death was explained with a timiko-lymphatic state. Now believe that in pathogenesis of this syndrome the large role is played by insufficiency of function of adrenal glands.

Treatment. Sufficient stay in the fresh air, observance of the mode is recommended. For increase in a tone of a muscular system it is important to carry out improving massage and gymnastics systematically. In a diet it is necessary to limit several amount of fats, easily assimilable carbohydrates, salts and liquids. Food has to be rather caloric and contain necessary amount of vitamins: With, And, E, groups B due to maintenance them in vegetables and fruit. In addition in cold season and in the spring prescribing of complex vitamin drugs is recommended. It is important to carry out in due time sanitation of a pharynx, in the presence of adenoides of the II—III degree the adenectomy is shown. In cases when against the background of limfatiko-hypoplastic diathesis arise and it is long inflammatory diseases proceed, except antibacterial the immunomodulatory therapy is shown.

NEUROARTHRITIC DIATHESIS.

According to Maslov's data, this diathesis from 3.6 to 8.2% of children, mainly preschool and younger school age have. The instability in lipid metabolism with bent to a ketosis and functional shifts from nervous system is found in them. Excess consumption of the food rich with purines and fats, the wrong mode which is under construction without presence of diathesis belong to the vneshnesredovy reasons leading to exacerbations of diathesis. Children with neuroarthritic diathesis are predisposed to development of diskineziya in them of biliary and uric tract, a cholelithiasis, obesity, arthralgias.

Clinical picture. At children of chest age the neuroarthritic diathesis can be shown by the increased nervous irritability, reduced appetite, unevenness in increase of body weight, unstable temperature. At the children of preschool and school age suffering from this form of diathesis more often than at others, neurotic reactions in the form of hyperkinesias, nightmares, enuresis are observed. Periodically repeating attacks of atsetonemichesky vomiting are especially characteristic. At the same time along with frequent vomiting the expressed smell of acetone from a mouth is felt, acetone content is also considerably increased in blood and urine. Neuroarthritic diathesis can be combined with exudative and catarrhal diathesis. At such children allergic diseases (asthmatic bronchitis, bronchial asthma, a dry form of eczema, neurodermatitis) quite often take place.

Children with neuroarthritic diathesis ripen mentally, quite often with unilateral endowments quicker. In blood they increased the level of uric acid, in urine periodically expressed uraturia, an oksaluriya, an acetonuria, a glycosuria. The persons which had symptoms of neuroarthritic diathesis in the childhood at mature age get sick with gout, diabetes, exchange arthritises, a hypertension, atherosclerosis more often.

Treatment. Children with neuroarthritic diathesis need providing them with sufficient rest in the fresh air. It is necessary to avoid overloads occupations at various specialized schools, than parents are fond, early noticing the accelerated development of such children. In a diet has to be in enough vegetables, fruit, with the moderate content of fats and restriction of proteins. It is not recommended to children to cook with neuroarthritic diathesis dishes from a liver, kidneys, bean, spinach, to give the chocolate, cocoa rich with purines and oxalic acid. For improvement of processes of exchange and stimulation of removal of urates and oxalates with urine periodic use of mineral water is shown (Yessentuki No. 4, 20, Smirnovskaya). At exacerbations of neuroarthritic diathesis, quinophan, cocarboxylase, ATP, vitamin B₆, pyridoxal phosphate, in hard cases – Miluritum (Allopyrinolum) are appointed. At an attack of atsetonemichesky

vomiting of the child it is necessary to hospitalize. In the conditions of a hospital isotonic solutions are intravenously entered. It is necessary to consider that the acetonemia is often observed in a disease of the child of an acute respiratory infection, pneumonia and other diseases. Repeated vomitings can be at a syndrome of an acute abdomen, meningitis, food poisoning, etc.

EXUDATIVE AND CATARRHAL DIATHESIS.

This state is frequent is called as allergic diathesis in connection with big tendency of such children to allergic diseases. However completely it is impossible to agree with this term as, first of all, deviations in a metabolism and hereditarily the caused shifts in some fermental systems are the cornerstone of exudative and catarrhal diathesis. In an organism of children the raised delay of sodium, chlorine and water is found that is caused by strengthening of mineralokortikoidny function and rather low glucocorticoid function of adrenal glands. The tendency to acidosis, a disproteinemia with rather small level of albumine and increase in globulins is inherent to them. Glikogenoobrazuyushchy function is reduced. The reduced gistaminopektichesky ability of blood, small activity of carboxypeptidase, acetylcholinesterase, a monoaminoxidase are noted that promotes increase in permeability of digestive tract for proteins. The predisposition to an allergy in exudative and catarrhal diathesis is connected, on the one hand, with the increased products of immunoglobulin E (reagins) participating in forming of an atopic allergy another – with tendency to accumulation of the biogenous amines (a histamine, serotonin, kinin, etc.) emitted by mast cells of friable connective tissue, basophiles, thrombocytes, lymphocytes in a patokhimichesky phase of allergic reactions. The excess use of carbohydrates contributes to the development of allergic reactions.

Clinical picture. The predisposition to an exudative catarrh of skin comes to light very much early, already in the period of a neonatality. Such children easily have limited erubescences in an intertrigo. On a hairy part of the head gray and fat scales (gneiss) are found. Children of the second half of the year have lives and after a year erythematic and vesicular rashes, typical exudative children's eczema are observed. Two types of exudative diathesis differ: *pastose and heretical*. At *pastose* the increased hydrophily of fabrics, tendency to excess body weight take place. Skin and mucous membranes at such children pale, rash elements on skin juicy, on their basis can develop the becoming wet eczema. In *heretical type* of diathesis of an enanthesis mainly dry, pruritic in this connection, children uneasy, sleep badly, the body weight increase at them is reduced. At any form of diathesis near affected areas of skin and mucous swelling of lymph nodes is often noted.

The children having exudative and catarrhal diathesis are inclined to a disease of blepharitis, conjunctivitis, pharyngitis, laryngitis, bronchitis, pneumonia. They quite often observe "geographical" language, an unstable chair, girls have a vulvovaginitis, boys have balanitis. At such children, especially at girls, urinary tract infection (urethritis, cystitis, pyelitis, pyelonephritises) easily develops. At exacerbations of diathesis in blood the moderate leukocytosis, an eosinophilia, lympho- and a monocytosis, moderate increase in SOE, sometimes hypo- or normochromic anemia are found.

Exudative and catarrhal diathesis can be combined with limfatiko-hypoplastic or with neuroarthritic diathesis. In such cases, allergic manifestations are more expressed. This form of diathesis can develop against the background of hereditary diseases of exchange of tryptophane, a pyridoxine, phenylalanine, at disakharidazny insufficiency, an exudative enteropathy.

Treatment. Breastfeeding as allergic manifestations, inflammatory diseases of skin and mucous develop at artificial feeding much more often is shown. The feeding up in the form of vegetable puree is recommended to be entered about 4-4.5 months, to avoid giving excess amount of carbohydrates (porridges, kissel, jam, sugar, etc.). The nursing mother should not use the foodstuff rich with allergens (natural coffee, chocolate, strawberry, a wild strawberry, a citrus, spices, canned food, eggs). Every time at introduction to a diet of the child of new food product of mother needs to be noted within the next week in the food diary reaction of the child to new food. It is so possible to reveal individual intolerance of the child to the certain feedstuffs leading to exacerbations of exudative diathesis. About 30% of need for fats at the expense of the vegetable oil rich with unsaturated fatty acids are recommended to cover.

At exacerbations of exudative and catarrhal diathesis with strengthening of allergic manifestations antihistaminic drugs (Dimedrol, tavegil, Suprastinum, klaritin, tsiterezin, etc.), stabilizers of membranes of mast cells (Ketotifenum, Zaditenum), drugs of Sa, vitamins are appointed (With, In₁, In₂, In₆, In₁₂, C₁₅).

At strongly disturbing itching zinc paste with anaesthesin locally is applied, 0.5% solution of bromic sodium or barbiturates inside are given. Children with more significant and often recurrent allergic manifestations need to be advised at the allergist or to direct for treatment in a hospital. It is necessary to consider also that with exudative and catarrhal diathesis the allergic reactions can cause various medicamentous drugs in children. In such cases it is important to cancel in due time the means causing adverse effect.

Tasks for independent work:

1. Solve situational problems and tasks of test control.

2. Using the scheme below, examine the patient with manifestations of diathesis. Write down the revealed changes in the state of health of your patient in a notebook.
3. Write prescriptions:
 - a) Suprastinum
 - b) tavegil
 - c) levamisole
 - d) Allopyrinolum

Scheme of inspection of the child.

When collecting the anamnesis to pay attention on:

- course of pregnancy of mother;
- disease of mother during pregnancy;
- intake of medicines during pregnancy;
- pregnant woman's food;
- features of feeding of the child from the moment of the birth;
- reactions to preventive inoculations;
- the diseases transferred the child;
- features of child care.

At an objective research to pay attention on:

- condition of food of the child;
- color of integuments;
- character and localization of skin rashes and other changes of skin (macerations, erythema);
- sizes of a liver and spleen, lymph nodes;
- child's language.

At interpretation of laboratory analyses to pay attention on:

- in complete blood count test: maintenance of erythrocytes, Hb, lymphocytes, eosinophils, monocytes, SOE);
- changes in a proteinogramma;
- changes in the clinical analysis of urine.

Situational tasks

Task No. 1

The boy B., 6 months, came to a hospital. Anamnesis of life: the child from the second pregnancy proceeding with toxicosis, births in time. Body weight at the birth of 2950 g, length – 50 cm. Assessment on a scale Apgar of 7/8 points. To a breast it is attached in the delivery room, sucked well. On natural feeding up to 2.5 months, then it is transferred to artificial feeding in connection with a hypogalactia at mother. Vaccination of BCZh in maternity hospital, other inoculations were not carried out.

Anamnesis of a disease: after transfer to artificial feeding (mix "Nan") the boy on skin of cheeks had sites of reddening with elements of microvesicles which were exposed further a moknutiya with formation of pruritic crusts. At the age of 3 months on a hairy part of the head diffusion grayish-yellow scales appeared. From the 4th monthly age the frequent change of milk mixes (Frisolak, Enfamil, "Similac", Human, etc.) was carried out against the background of what skin manifestations of a disease amplified, involving a face, upper and lower extremities, a trunk in process. Further skin of flexion surfaces of hands and legs, area of buttocks was involved in process. Use of external medicamentous means ("talkers", creams, ointments, grass bathtubs) and antihistaminic drugs gave short-term effect. In 5.5 months the feeding up – porridge is entered then the expressed concern was noted, there was a diluted chair with slime and undigested lumps, sometimes with blood streaks. The child practically does not sleep recently. For inspection and treatment the child was sent to a hospital.

Family anamnesis: mother – 29 years, has eczema (in an aggravation stage now); the father – 31 years, has a pollinosis.

At receipt condition of the child heavy. It is sharply uneasy. On a hairy part of the head manifestations of seborrheal peeling are expressed as "cap". Integuments almost everywhere (except for a back) are covered with the becoming wet erythematic vesicles, places are covered with crusts. Behind ears, in the field of cervical folds, in elbow and popliteal bends, on a scrotum and in a crotch sites with moknutiy and macrolaminar peeling are noted. Peripheral lymph nodes up to 0.5 cm in the diameter, painless are palpated, elastic consistence, are not soldered to surrounding fabrics. Puerile breath, there are no rattles. Rhythmical cardiac sounds, clear, ChSS of 114 beats/min. The stomach is blown a little up, painless at a palpation in all departments; rumbling on the intestines course. A liver + 3 cm from under a costal arch. The spleen is not palpated. The chair diluted, flavovirent color, with undigested lumps and slime. Light urine. There are no all-brain, focal and meningeal symptoms.

General blood test: Hb - 104 g/l, Ayr. - 3.5 kh 1012/l, C. the item - 0.8, Leyk. - 11.2 $\cdot 10^9$ /l, p/ya-7%, with-33% e e-9, l-41 of %, m-10 of %, mm/hour SOE-12.

General analysis of urine: color is light yellow, specific weight – 1010, the squirrel does not, glucose does not, the epithelium flat – is a lot of, leukocytes – 6-8 in p/z, erythrocytes do not, cylinders do not, slime – is a lot of.

Biochemical analysis of blood: crude protein – 68 g/l, urea – 3.6 mmol/l, bilirubin the general – 16.7 μ mol/l, potassium – 4.2 mmol/l, sodium – 139 mmol/l, the calcium ionized – 0.95 mmol/l (norm 0.8-1.1), phosphorus – 1.0 mmol/l (norm 0.6-1.6), iron – 8.1 μ mol/l (norm 10.4-14.2), zhelesvyazyvayushchy ability of serum – 87.9 μ mol/l (norm 63.0-80.0), free hemoglobin – is not defined (the norm – is not present), IgE – 830 ME/l (norm – to 100 ME/l).

Task:

1. Prove your diagnosis?
2. List the main pathogenetic mechanisms of development of a disease in this patient.
3. Draw up the plan of further inspection.
4. List the risk factors promoting implementation of a disease at this child.
5. Basic principles of therapy of this disease.

Task No. 2

The child of 6 years came to vaccination, treats group of often ill.

At survey sluggish, pale, a skin fold the flabby, muscular system is poorly developed. Growth – 106 cm. Weight – 24.5 kg.

Mucous light pink, back wall of a throat of a mouth with the expressed follicles, palatine tonsils are hypertrophied the II-III degrees, breath through a nose is complicated because of adenoid growths of the III degree. Peripheral l / at: submaxillary, inguinal, axillary l / nodes are palpated: small, multiple, mobile, elastic consistence.

In heart – muting of warm tones, functional systolic noise over all warm area, ChSS – 90 in min., the ABP – 95/65 mm Hg.

Task:

- 1) Define the state of health of the child.
- 2) Of what diathesis it is possible to think in this case?
- 3) What complications of infectious diseases often arise against the background of this anomaly of the constitution?
- 4) Whether treatment is necessary for this child? If yes, that what?

Task No. 3

Child of 8 years. The child's mother not the first time addressed with complaints to an abdominal pain, frequent headaches, a bad dream, small appetite, frequent vomitings.

The boy perfectly studies at school, occupations come to him easily, except school visits a fine art studio and a chess circle. The boy is observed at the neuropathologist.

The condition of the child worsened (there was numerous vomiting with an acetone smell) after the use of a large number of chocolates during the birthday at the friend.

Genealogical anamnesis: the grandfather has a gout, the aunt has an urolithiasis, mother has a migraine.

At survey: weight – 26.5 kg. Growth – 128 cm

On bodies and systems at the time of survey without pathology.

Task:

- 1) Estimate the state of health of the child.
- 2) Of what diathesis in this case it is possible to think?
- 3) What examinations need to be performed? What results are expected?
- 4) What treatment is necessary for the child?

Test control:

1. Most often meets at children of early age:
 - a) infectious form of an allergy
 - b) food allergy
 - c) medicinal allergy
 - d) inhalation allergy
2. The diathesis which is characterized genetic by the determined disturbances of a number of the enzymes which are taking part in purine exchange and accumulation of uric acid is called:
 - a) limfatiko-hypoplastic
 - b) neuroarthritic
 - c) exudative and rolling
3. Genetically determined feature of a metabolism defining an originality of adaptive reactions of an organism and contributing to a certain group of diseases is called:
 - a) anomaly of the constitution
 - b) disease
4. Diathesis in which the congenital generalized immunopatiya which is combined with insufficiency of function of bark of adrenal glands is observed is called:
 - a) neuroarthritic

- b) limfatiko-hypoplastic
 - c) exudative and catarrhal
5. Development of atsetonemichesky vomiting is most probable in diathesis:
- a) neuroarthritic
 - b) limfatiko-hypoplastic
 - c) exudative and catarrhal
6. Development of acute adrenal insufficiency is most probable in diathesis:
- a) neuroarthritic
 - b) limfatiko-hypoplastic
 - c) exudative and catarrhal
7. Manifestation of children's eczema at children are caused by genetically determined hyperproduction:
- a) IgA
 - b) IgD
 - c) IgM
 - d) IgE
8. Increase in a thymus is characteristic of diathesis:
- a) neuroarthritic
 - b) limfatiko-hypoplastic
 - c) exudative and rolling
9. At treatment of the child with neuroarthritic diathesis in the last turn it is appointed:
- a) Allopyrinolum
 - b) plentiful alkaline drink
 - c) sedative therapy
 - d) diet with restriction of the products rich with purines
10. The most significant food-borne allergens are:
- a) cow's milk
 - b) protein of egg
 - c) egg yolk
 - d) fish
 - e) semolina
 - e) corn grits
11. The main markers of limfatiko-hypoplastic diathesis are:

- a) congenital generalized immunopatiya
 - b) excess synthesis of a histamine
 - c) genetically determined hyperproduction of IgE
 - d) hyperadenosis
 - e) thymus hyperplasia
 - e) hypofunction of bark of adrenal glands
12. In the absence of primary prevention the limfatiko-hypoplastic diathesis can lead to development of such states as:
- a) sprue
 - b) syndrome of sudden death
 - c) gout
 - d) autoimmune diseases
 - e) neurotic syndrome
13. In the absence of primary prevention the neuroarthritic diathesis can lead to development of such states as:
- a) urolithiasis
 - b) syndrome of sudden death
 - c) gout
 - d) autoimmune diseases
 - e) neurotic syndrome

Class in a subject:
"DISEASES OF NEWBORNS"

I. Scientific and methodical justification of a subject.

The perinatal injury of a brain which is quite often followed by developing of diencephalic pathology can be the reason of development not only destructive and dystrophic processes in the leading departments of central nervous system, but also a number humoral somato - endocrine disturbances which can remain not only during the subsequent periods of the childhood, but also at adults.

In terms of unity of mother and a fruit, the solution of extremely complex complex problems of the perinatal period depends not only on activity of the pediatrician, but also the therapist, the obstetrician. These circumstances explain need of serious studying diseases of the period of a neonatality of students of medical faculty.

II. Purpose of activity of students on occupation:

The student has to know:

- The contributing factors to development of perinatal pathology, birth trauma of newborns;
- Main etiopatogenetic mechanisms of development of perinatal encephalopathy;
- Clinical manifestations of perinatal pathology of central nervous system, birth trauma;
- Main diagnostic methods of a birth trauma, perinatal damage of central nervous system;
- Basic principles of treatment and prevention of perinatal pathology;
- Main aspects of an etiopathogenesis of a hemolytic disease of newborns;
- Clinical picture GBN and differential diagnostics with other jaundices of the neonatal period;
- Basic principles of treatment and prevention of GBN.

The student has to be able:

- To purposefully collect the anamnesis;
- To perform objective examination of the newborn;
- To interpret laboratory and paraclinic data;
- To make the diagnosis;
- To make the treatment plan of the patient;
- To write prescriptions on the main medicines;
- To define preventive actions.

III. Content of training:

1. Perinatal damage of central nervous system (etiology, pathogenesis, clinic, treatment, prevention).
2. Remote effects of perinatal encephalopathy
3. Dispensary observation of children with perinatal damage of central nervous system.
4. Types of birth trauma.
5. Injuries of the central and peripheral nervous system (clinic, treatment, remote effects)
6. Patrimonial damages of a bone and articulate system (clinic, treatment).
7. Prevention of patrimonial traumatism.
8. Hemolytic disease of newborns (etiology, pathogenesis, clinic, treatment, prevention).

IV. Educational material security.

1. Visual aids: tables, schemes, multimedia presentations, videos, audiogramma.
2. Educational medical documentation (case histories, laboratory researches,

roentgenograms).

3. Technical means of training.
4. Literature.

V. The list of the recommended literature.

1. Children's diseases: the textbook / under the editorship of A.A. Baranov. – M.: GEOTAR-media, 2009. – 1008 pages.
2. Pediatrics: The textbook for medical schools. Under the editorship of N.P. Shabalov. – SPb: SpetsLit, 2006. – 895 pages.
3. Propaedeutics of children's diseases / to N.A. Geppa. – M.: GEOTAR-media, 2009. – 464 pages.
4. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Background diseases of children of early age. Manual for students. – Vladikavkaz, 2011. – 64 pages.
5. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Technique of a research of the child. The study guide for students. – Vladikavkaz, 2011. – 51 pages.
6. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Scheme of a case history. The study guide for students. – Vladikavkaz, 2011. – 38 pages.
7. Lectures on pediatrics.
8. Methodical instructions for out-of-class work of students 4 courses of medical faculty on discipline "Pediatrics".

VI. List of questions for check of initial level of knowledge:

1. Anatomico-physiological features of central nervous system of newborns.
2. The organization of work in chambers and departments of newborns in maternity hospital.
3. Toxicoses of pregnant women.
4. Anomalies of development and disease of a fetus and its appendages.
5. Pathology of childbirth (childbirth by a large fetus, pre-natal asphyxia of a fetus and so forth).

VII. List of questions for check of final level of knowledge:

1. Risk factors of perinatal damage of central nervous system.
2. Clinical signs perinatal hypoxic - traumatic damage of nervous system at children.
3. The plan of treatment in perinatal encephalopathy.
4. Remote effects of perinatal encephalopathy.

5. Dispensary observation of children with perinatal damage of central nervous system.
6. Risk factors of patrimonial traumatization.
7. Give definitions to the concept "birth trauma". What types of birth trauma do you know?
8. Describe clinical manifestations of birth trauma of the central and peripheral nervous system, bone damages.
9. Call the basic principles of treatment of birth trauma.
10. What does prevention of patrimonial traumatism consist in?
11. Etiopatogenetichesky mechanisms of development of the hemolytic disease of newborns (HDN).
12. Call the main clinical manifestations of GBN.
13. With what diseases it is necessary to differentiate GBN?
14. What treatment is held by sick GBN?
15. What forecast of this disease?
16. What does prevention of GBN consist in?
17. How medical examination of sick GBN on the site is carried out?

Information block.

HEMOLYTIC DISEASE OF NEWBORNS (HDN)

– this serious congenital illness arising in utero or in the first days after the birth as a result of immunological incompatibility of blood of mother and a fruit on a Rhesus factor, group antigens of blood and is very rare – on other factors of blood (Kel, Duffy, Lyuteran, Lewis's system, etc.).

The Rhesus factor is a protein, is mainly in erythrocytes, but also contains in smaller quantities in other blood cells (leukocytes, thrombocytes). There are following types of a Rhesus factor which qualitatively differ from each other: antigens D, S and E. Most often GBN develops owing to immunological incompatibility of blood of mother and a fruit on factor.

Pathogenesis.

Getting to blood of the Rh-negative person, antigen D causes an immune response with development of anti-Rh antibodies. GBN develops when the Rh-negative woman is pregnant a Rhesus factor with a positive fruit which inherited a Rhesus factor from the Rh-positive father. A fruit Rhesus factor, getting through a placenta into mother's blood stream, immunizes it, causing development of anti-Rh antibodies. Immunization of women about a Rhesus factor negative accessory of blood can come also after administration of Rh-positive blood by it irrespective of a method and time of introduction (intramuscularly or intravenously) and also after artificial or a misbirth, an extrauterine pregnancy.

The anti-Rh antibodies developed in mother's organism in response to a fruit Rhesus factor get to blood of a fruit and cause reaction antigen antibody with its Rh-positive erythrocytes that brings to hemolysis of the last and accumulation in blood of newborn indirect bilirubin in toxic quantities. At normally proceeding pregnancy the placenta is impenetrable for anti-Rh antibodies, performing barrier function. However at a number of the morbid conditions causing disturbance of integrity of chorion or their permeability: toxicoses of pregnant women, threat of termination of pregnancy, extragenital pathology, anti-Rh antibodies pass through a placenta and, getting to fruit blood, cause reaction antigen antibody with hemolysis of its erythrocytes and development of GB in utero. The most considerable transplacental transfusions of antigens and antibodies are observed during parturient activity, especially at long childbirth and at surgery (manual separation of a placenta, version, etc.).

At the first pregnancy the immunization of the woman begins generally only after the delivery and therefore at a fruit and newborn GB does not develop. Each subsequent pregnancy strengthens a condition of a sensitization to a Rhesus factor, is followed by increase in a titer of anti-Rh antibodies during pregnancy and increase of danger of defeat of a fruit and the newborn.

Reaction leads antigen antibody to the strengthened hemolysis of erythrocytes therefore a significant amount of free (indirect) bilirubin which has toxic effect on the child's organism is formed, breaking fabric metabolism due to switching off of respiratory enzymes from phosphorylation processes. Indirect bilirubin is insoluble in water and cannot be removed from an organism with urine and a stool. Thanks to good solubility of free bilirubin in lipids it collects in large numbers in the bodies rich with lipids – a brain, adrenal glands, a liver, etc., causing disturbance of their function. Indirect bilirubin enters temporary combination with albumine and is transported by blood to a liver where it is taken a hepatic cell and under the influence of enzyme of glucuronosyltransferase connects to two molecules of glucuronic acid, turns into the non-toxic water-soluble connected (direct) bilirubin – bilirubin diglucuronide. Direct (connected) bilirubin from a hepatic cell gets to biliary capillaries, further to biliary ways and a gall bladder from where it is allocated in intestines. One of the basic reasons promoting accumulation of indirect bilirubin in blood and tissues of the newborn and appearance of jaundice is considerable decrease of the activity of glucuronosyltransferase. At premature children, in connection with a bigger immaturity of fermental systems of a liver and the slowed-down their maturing, process of a bilirubin conjugation is delayed and therefore the hyperbilirubinemia is expressed more considerably. Besides, the hyperpermeability of capillaries of premature children and histologic

immaturity of their fabrics facilitate transition of indirect bilirubin to nervous tissue at its lower concentration (150-170 $\mu\text{mol/l}$), especially in combination with a hypoxia and a hypoproteinemia.

Clinical picture GBN. Distinguish 3 clinical forms of a disease

1) hydropic

2) icteric

H) anemic

The GBN hydropic form is the most severe form of a hemolytic disease. The disease develops still in utero. Most often the birth of children with an edematous form is preceded by the birth of children with the GB icteric form or spontaneous abortions. Children with an edematous form are very pale, anemic, but without jaundice or the last is expressed very slightly. The absence of jaundice is connected with the fact that the indirect bilirubin which is formed at hemolysis of erythrocytes passes transplacental into blood of mother and is conjugated by her liver. Partially function of binding of indirect bilirubin is probably undertaken by a placenta, than it is possible to explain the big sizes it at the GB hydropic form.

The most expressed symptoms of a disease along with anemia are considerable hypostasis of hypodermic cellulose, presence of free liquid in cavities, increase in a liver and spleen. In blood the hypoproteinemia and existence of young forms of erythrocytes – normoblasts and erythroblasts are characteristic. At this form of a disease the hemorrhagic syndrome is quite often observed. Pronounced anemia in combination with a hypoproteinemia and damage of a vascular wall leads to development of the heart failure which is most often a proximate cause of death. However use of fractional zamenny transfusions of small amounts of a packed red cells under control of venous pressure in certain cases gives the chance to save these children.

The GBN icteric form is the most frequent, but less severe form of a hemolytic disease. Its most characteristic symptom – the jaundice which is shown sometimes already at the birth, however most often arising in the first day of life. Since the birth at the child the icteric coloring of vernix caseosa and amniotic waters is quite often observed. At this GBN form the fast increase of jaundice is noted. At healthy newborns the bilirubin content in umbilical blood averages 25-30 $\mu\text{mol/l}$, without exceeding 51 $\mu\text{mol/l}$. Further increase of indirect bilirubin in blood should not exceed 3.2 $\mu\text{mol/l}$ an hour. At the GBN icteric form can take place as the increased content of indirect bilirubin in umbilical blood due to pre-natal accumulation, and its fast hourly gain in the post-natal period because at the time of delivery antibodies from mother plentifully get to a fruit.

At the GBN icteric form the hourly gain of bilirubin fluctuates from 5 to 17 $\mu\text{mol/l}$. Increase in indirect bilirubin in blood above critical level – 300-342 $\mu\text{mol/l}$ leads to damage of the central nervous system and development of "kernicterus". Symptoms of bilirubinovy intoxication are shown in aggravation of symptoms of the child, decrease in physiological reflexes of the period of a neonatality, appearance of hypotension, a lethargy.

Premature children can have apnoea attacks passing into asphyxia.

Against the background of sharp hypotension and an areflexia, short-term tonic spasms, stiff neck, "mask-like face" with widely open eyes develop. At deeply premature children the phase of oppression can last several hours. At more mature and full-term it quickly passes into a spastic phase, with a heavy picture of defeat of kernels of a brain: shrill "brain" shout, an extensive hyper tone, stiff neck, an oculogyric crisis, a nystagmus, a symptom "setting the sun". Predictively emergence of a syndrome of respiratory disorders is adverse. Further these children have permanent motive disturbances, spastic paresis, vestibular and oculomotor disorders. Damage of hearing, lag in physical and psychological development.

With a hemolytic disease it is important to distinguish children with the greatest risk of developing bilirubinovy encephalopathy from newborns. Treat them:

- 1) children with rough hemolysis of erythrocytes at the birth,
- 2) premature children with a hemolytic disease in combination with extensive hemorrhages and hemorrhages in internals,
- 3) premature and full-term children with big loss of weight,
- 4) hypotrophicities,
- 5) children who were born in asphyxia.

Eritroblastoz is not constant sign at the GBN icteric form, however serves as a reliable indicator of disease severity.

The anemic form – most the GBN easy form, meets seldom. Its main manifestation – anemia and pallor of integuments. Increase in a liver and spleen is significant slightly and meets seldom.

The diagnosis is established on the basis of presence of Rh incompatibility of blood of mother and a fruit, a clinical picture and definition of hemoglobin and erythrocytes in the newborn's blood.

As the GBN various forms, in effect, are stages of one process, three severity of a disease are distinguished: easy, moderately severe and the heavy Severity of GBN is estimated on

expressiveness of such main symptoms of a disease as puffiness, jaundice and anemia at the time of the birth.

If the main symptoms belong to the I degree, the GB should be considered an easy form (assessment 1-3 points) if there is at least one of signs of the II degree, this condition of moderate severity (4-6 points). With at least one sign of the III degree – a severe form of a disease (7-9 points).

Main clinical symptoms	Severity of GB		
	I	II	III
Anemia (the maintenance of Nv in umbilical blood, g/l)	150	150-100	100
Bilirubin content in umbilical blood, $\mu\text{mol/l}$	85.5	85.5-136	137
Edematous syndrome	Pastosity of hypodermic cellulose	Pastosity and ascites	Universal hypostasis

Treatment of GBN has to be organized in due time and directed to the fastest removal from an organism of toxic decomposition products of erythrocytes, indirect bilirubin and also antibodies promoting hemolysis. *The zamenny blood transfusion (ZBT)* remains the most effective method of treatment at a severe form of GBN. Recently there are attempts instead of zamenny blood transfusion to carry out hemosorption or a plasma exchange with administration of the defrozen plasma.

Indications to the first zamenny blood transfusion in the 1-2nd day of life at the full-term newborn are:

- 1) developing of jaundice right after the birth or during the first hours life;
- 2) bilirubin content in umbilical blood is higher than $68 \mu\text{mol/l}$;
- 3) hourly gain of bilirubin during the first hours lives more than $6.8 \mu\text{mol/l}$;
- 4) hemoglobin level at the birth - 150 g/l and less.

Premature and unripe newborns have indications to zamenny blood transfusion, except early appearance of jaundice and low level of hemoglobin – the content of indirect bilirubin in umbilical blood – $60 \mu\text{mol/l}$ and more and hourly gain of bilirubin of $5 \mu\text{mol/l}$ and more.

Indications to the repeated zamenny blood transfusion (ZBT) is the same hourly gain of bilirubin at the full-term newborns, as well as at the first transfusion, but hourly gain is estimated in relation to the figures received after the first ZPK.

Absolute indications to ZPK (both to the first, and repeated) on the 3rd, the 4th and later day of life – increase in level of indirect bilirubin to 300 $\mu\text{mol/l}$ at the full-term child and up to 270 $\mu\text{mol/l}$ at the premature or unripe newborn. Emergence of symptoms of bilirubinovy intoxication, the increasing slackness, decrease in physiological reflexes of the period of a neonatality, apnoea attacks – indications for ZPK irrespective of bilirubin level in blood.

At GBN caused a Rhesus factor conflict the Rh-negative blood odnogruppy with the child's blood, in number of 150-180 ml/kg is transfused. At AVO-incompatibility the zamenny transfusion is carried out by a packed red cells 0(I) of the group compatible on a Rhesus factor with blood of a fruit and AV (IV) plasma of group or odnogruppy with the child's blood in the ratio 3:1. In 20-30 minutes prior to operation to the child plasma or 10% albumine solution in number of 5-8 ml/kg of weight for mobilization of bilirubin from fabrics is entered into a vascular bed. Operation begins with removal of blood of the child, fractional doses, on 20 ml. blood of the child is removed and donor blood is entered (premature transfusions become on 10 ml. blood). After each 100 ml. 2 ml are necessary for the transfused donor blood. 10% of solution of a gluconate of calcium and 2 ml. 5% of solution of glucose. At the end of operation 25 ml are entered. plasmas or 10% of albumine and 5-10 ml. 4-5% of solution of hydrosodium carbonate.

Infusional therapy is performed in addition to ZPK or at not severe forms of GBN independently. The following solutions are used: Haemodesum – 10 ml/kg, albumine – 10-20% on 5-8 ml/kg, plasmas – 8-10 ml/kg, glucose of 10%, a reopoliglyukina – 10 ml/kg in various combinations. In a low hourly diuresis (less than 0.5 ml/hour) diuretic drugs are used.

The phototherapy (phototherapy), is directed to destruction of indirect bilirubin in blankets of skin of the newborn. The faltering phototherapy for 2 hours every 2 hour (6 photosessions) or on one hour in an hour (12 photosessions) is most effective. The phototherapy course – 24-48 hours, for strengthening of photoeffect is recommended vitamin B₂ on 5 mg 2 times a day inside.

For improvement of conjugation and excretory function of a liver are appointed phenobarbital at the rate of 10 ml/kg of body weight a day. Also antioksidazny therapy is recommended: 20-30% tocopherol solution (vitamin E) on 0.2 ml. intramuscularly, 1 time a day; Riboflavinum (vitamin B)₂, calcium pangamat (vitamin B)₁₅ inside.

At a syndrome of "pachycholia" also antispasmodics are appointed bile-expelling (10% solution of sulfate magnesia). Removal of bilirubin from intestines is promoted by prescribing of Carbolenum, agar-agar.

Feeding of newborns with GBN is carried out by the decanted milk for an exception of physical activity which can strengthen hemolysis of erythrocytes. Existence of antibodies in milk

of mother is not a contraindication for feeding by breast milk as in digestive tract of the child of an antibody collapse. However it is reasonable to put the child to a breast only after reduction of jaundice and decrease in content of bilirubin in blood.

Prevention of GBN.

Specific method of prevention a Rhesus factor sensitization – introduction to Rh-negative women after any termination of pregnancy (first labor by a Rh-positive fruit, repeated childbirth without manifestations of a sensitization, abortions, operations for an extrauterine pregnancy) an anti-Rhesus factor immunoglobulin. The anti-Rhesus factor immunoglobulin is entered on one dose (200 mkg) intramuscularly once no later than 72 hours after the delivery. At abortion – at once after the end of operation. After operation of Cesarean section, manual department of an afterbirth, placental presentation, premature amotio of normally located placenta, drug is necessary in a double dose.

HYPOXIA

– it is symptom complex which arises in the conditions of oxygen insufficiency.

The most frequent reasons of perinatal hypoxemic cerebral damages following:

1. The reasons connected with the state of health of the woman – somatic, gynecologic, infectious diseases of the woman, including specific (a cytomegaly, toxoplasmosis, a herpesviridae infection), the prof. of a harm (work in the conditions of a lack of oxygen, influence of noise, vibration, fields of high frequency, intensive magnetic fields, contact with chemical and bioorganic substances).
2. The reasons connected with the complicated pregnancy course – toxicosis of the II half, threat of termination of pregnancy.
3. The reasons connected with a complication of a course of childbirth – long or rapid childbirth, weakness of patrimonial activity, prenatal izlitiye of waters.
4. The reasons connected with a condition of the newborn – the hypoglycemia, a serious illness of respiratory organs, a cardiovascular system, immaturity of the child, phrenic hernia promoting preservation of a heavy hypoxia after the child's birth.

Distinguish primary (inborn) and secondary (the post-natal, arising during the first hours lives) a hypoxia of newborns. On emergence time primary asphyxia can be anti- and (or) intranatal. Depending on duration of influence allocate sharp (more often intranatal) and chronic (is more often pre-natal) a hypoxia. The severity depends on duration, time of emergence and severity of a hypoxia.

Pathogenesis of an antenatal hypoxia. Pathology of pregnancy leads to fall of the contents of oxygen in blood and to a hypoxia. At the same time the compensatory mechanisms directed to improvement of supply of bodies and fabrics by oxygen turn on: increase in number of the circulating erythrocytes, increase of a warm rhythm, increase in emission of glucocorticoids, some increase in systolic pressure. However in a long hypoxia these compensatory mechanisms are exhausted. Energetically unprofitable pathway of carbohydrates – anaerobic glycolysis joins.

The circulating blood is redistributed in such a way that first of all vitals – a brain, heart are supplied with blood. It, in turn, leads to impoverishment of supply with blood of intestines, muscle tissue, kidneys and developing of a metabolic acidosis which promote disturbance of permeability of a vascular wall and cellular membranes. It mediates an exit of a liquid part of blood from a vascular bed (wet brain), a gipovolyumiya, increase in haemo concentration up to formation of intravascular blood clots, development of diapedetic and big large hemorrhages in a brain. There is a death of neurons of bark and a subcortex. At the same time cells come out potassium, there is a hypocalcemia, a hypomagnesiemia, it aggravates damage of heart and central nervous system. Systolic pressure decreases that is followed by sharp falling of intensity of a brain blood-groove. There is brain ischemia which is the main mechanism of hypoxemic encephalopathy and also ischemia of heart, papillary muscles.

Pathogenesis of an intranatal hypoxia. As a result of action of harmful factors in labor, in an organism of the child the hypoxia, a hypercapnia, acidosis which cause a spasm of pulmonary arterioles and hypostasis of an alveolar wall develops, slow down surfactant synthesis. There are atelectases which aggravate a hypoxia, acidosis. The spasm of arterioles leads to increase in pressure in vessels of lungs, to increase in permeability of a vascular wall. Elements of plasma transude in alveoluses, fibrin drops out that leads to formation of hyaline membranes. In vessels there is a haemo concentration, intravascular coagulation, i.e. the hydropic and hemorrhagic syndrome up to hemorrhages develops in the central nervous system, in tissue of lungs and other bodies. There is a syndrome of respiratory disorders. Development of a post-natal (secondary) hypoxia is the cornerstone of disorder of breath and blood circulation.

The intracranial birth trauma unlike a hypoxia is a local damage of fabrics of a fruit in the course of childbirth as a result of the mechanical influences causing compression of a brain, crush, ruptures of fabrics, hemorrhage in tissue of a brain or a cover. It is promoted by discrepancy of patrimonial ways of mother and the sizes of a head of a fruit, anomaly of presentation, too rough or long patrimonial activity.

Encephalopathy understand not classified damages of the central nervous system as the term.

Clinical picture. At the child's birth the state assessment on a scale Apgar is carried out twice – in the 1st and 5th minutes of life. At premature newborns assessment on a scale Apgar should be combined with assessment of severity of respiratory disorders.

In an acute period the hypoxia, depending on severity, conditionally is divided into easy, medium-weight and heavy.

Easy degree – assessment on a scale Apgar of 6-7 points, is characterized by increase in neuroreflex irritability. The emotional and motive concern, a spontaneous reflex of Moro, start, a melkoamplitudny tremor, sometimes a non-constant horizontal nystagmus, passing squint is noted. Congenital reflexes are moderately strengthened. Muscular dystonia, sometimes – changes of a respiratory rhythm, moderate tachycardia is noted. By 15th day the symptoms begin to disappear.

Medium-weight degree – assessment on a scale Apgar – 4-5 points. In the first 7-10 days of life the damage of nervous system is characterized by the following syndromes: general oppression, gipertenzionny, gipertenzionno-hydrotsefalny, convulsive syndrome or their combination.

The syndrome of the general oppression is shown by considerable slackness of newborns, weak reaction on surrounding, decrease in congenital reflexes, including sucking and deglutitory, a hypomyotonia which can soon be replaced by increase in a muscle tone. In the first 5-7 days the convulsive syndrome (kloniko-tonic spasms), a horizontal nystagmus, passing focal symptoms of damage of the central nervous system (dysfunction of cranial nerves of the 3, 6, 7 couples, an anizorefleksiya, asymmetry of a muscle tone), disturbances of a warm rhythm and breath is sometimes observed.

At a gipertenzionny syndrome the concern of newborns, the general hyperesthesia, a sleep disorder, sometimes screaming, a tremor a century, a chin, increase in reflexes of oral automatism, tendon jerks, emergence of a spontaneous reflex of Moro prevails. Protrusion and tension of fontanel, a protrusion of eyeballs, a moderate symptom Gref is possible.

The divergence of cranial seams, increase in the sizes of the head and fontanel, change of a shape of a skull is typical for a gipertenzionno-hydrotsefalny syndrome along with the above symptomatology.

The convulsive syndrome is characterized more often by generalized or local spasms of clonic or kloniko-tonic character. Spasms can arise during the first hours – days after the child's

birth.

Heavy degree – assessment on a scale Apgar 1-3 points. It is characterized by symptoms pre- and coma of the newborn. The sharp weakness, slackness, an adynamia, shout weak is noted or is absent, a hypomyotonia or an atony. Reaction to pain stimulation is sharply reduced or is not caused. Tendon and congenital reflexes, including suction and swallowing, are oppressed or are absent, the vertical nystagmus, the floating movements of eyeballs, an anisocoria is quite often observed. In the most hard cases the pupils are narrowed, the photoharmose sluggish or is absent, corneal reflexes are sharply reduced. Arrhythmic breath, with a frequent apnoea, repeated attacks of cyanosis. Bradycardia, pulse arrhythmic is noted, arterial blood pressure is lowered. Repeated spasms with prevalence of a tonic component are observed. Depending on the nature of damage and localization of the center there is paresis of cranial nerves, motive disturbances. Defeats most part are diffusion, taking a trunk, subcrustal departments and a cerebral cortex. Generalized wet brain is the cornerstone of these changes. Weight of a state can be caused by hemorrhages owing to a heavy hypoxia.

In the recovery period the following syndromes are observed: motive disturbances, a delay of psychomotor and prespeech development, Epi - and hydrotsefalny, vegeto-visceral dysfunctions, the increased neuroreflex irritability and a tserebroastenichesky syndrome.

The syndrome of motive disturbances can be shown by increase or decrease in physical activity, hypo - or a hypertension. In a hypomyotonia the spontaneous activity, a muscle tone, tendon jerks is reduced. In a muscular hypertension the physical activity is also sharply reduced, the child accepts an embryonic pose, the general constraint appears from behind a high muscle tone. Congenital reflexes have paradoxical dynamics: oppression of reflexes of suction, swallowing, a support, crawling along with strengthening and a delay of involution of reflexes of oral automatism, Robinzona, Babkina. Mono - both a hemiparesis spastic and sluggish and also hyperkinesias are possible.

It is necessary to remind that the physiological hyper tone appears from 2nd week of life and disappears by 3 months. At unripe and premature the hyper tone appears later and keeps long (up to 5 months). If the muscle tone by 5 months is not normalized, it is necessary to exclude cerebral palsy.

Outcomes: recovery (normalization of a tone of muscles from proximal departments to distal within 3-6 months), children's cerebral palsy.

The syndrome of a delay of psychomotor and prespeech development can develop at

children with a convulsive syndrome and also at the children who transferred coma. Mental development usually advances motor.

At prevalence of disturbances of statiko-motor functions the child begins to hold later the head, to sit, go at normal mental development.

Primary disturbance of the speech is characterized by weak shout or its absence, disturbance of rates of development of the speech (delay of forming of an articulation).

At prevalence of disturbance of mentality the child fixes later a look, traces a toy, is guided in the environment. Outcomes: mental retardation.

Episindr it is characterized by presence of the repeating spasms of polymorphic character from small attacks to the developed general spasms of toniko-clonic character. Spasms become paroxysmal, serial, are followed by consciousness disturbance. Emergence of foam in a mouth, an urine upuskaniye is sometimes noted. The outcome is better if spasms arise in the period of a neonatality or early chest age when they can be the child's manifestation of metabolic disturbances with a somatopathy, in later terms there are more data for development of epilepsy.

The Hydrotsefalny syndrome is shown in the form of open external hydrocephaly in which the divergence of seams of a skull, protrusion and a pulsation of fontanel, significant increase in the head, increase in an occipital hillock, a symptom "setting the sun" is noted, a protrusion of eyeballs, the nystagmus, paresis of 3-6 pairs of cranial nerves is possible. The diagnosis is made by results the ECHO encephalography and neurosonografiya. At a research of an eyeground hemorrhages, changes of caliber of vessels can come to light. The X-ray analysis of a skull allows to exclude volume formations in a head cavity, to find kaltsinata owing to the postponed pre-natal infection.

The course of a hydrotsefalny syndrome, can be favorable (during treatment symptoms disappear) and adverse: forming of true dekompensirovanny hydrocephaly, epilepsy, cerebral palsy.

The syndrome of vegeto-visceral dysfunctions is characterized by emergence of the "marble" drawing of skin, a Crocq's disease, thermal control disorders, gastrointestinal diskineziya with the pylorospasm phenomena, increase in a vermicular movement of intestines, rumbling, constipations, vomiting, the vomiting, lability of cardiovascular and respiratory systems increased by perspiration, a sialosis, disturbance of appetite. Outcomes: favorable (at unripe children since similar symptoms are caused by immaturity of central nervous system), adverse – forming of vegeto-vascular dystonia, dyskinesia of digestive tract, a bile-excreting system, enuresis.

Syndrome of the increased neuroreflex irritability. At children the bad dream, concern, the

increased physical activity, starts, revival of tendon jerks and reflexes of newborns, vomiting, a horizontal nystagmus, the non-constant meeting squint is noted.

Outcomes: recovery by 4-6 months or later, forming of a convulsive syndrome (at 15-20% of children), transition to an asthenoneurotic syndrome (after a year).

Tserebroastenichesky syndrome (asthenoneurotic syndrome).

Against the background of normal psychophysical development the emotional lability, motive concern at minor changes in the environment is observed. The imbalance of the processes of excitement and braking proceeding on two types is noted: hypersthenic and hyposthenic. The general in symptomatology: fears, tics, logoneurosis, sleep disorder, difficult adaptation to a new situation.

Outcome: recovery, psychopathic personal development.

Diagnosis. At diagnosis consider diseases of mother, the obstetric and gynecologic anamnesis, data of a clinical laboratory research. As additional diagnostic methods carry out USI, a computer tomography (allow to determine the area and localization of damage), an electroencephalography (emergence of pathological potentials), radio isotope scanning with technetium isotope (allows to define localization of sites of necrosis, near-ventricular softening). The ekhoentsefalografiya which allows to establish localization of hemorrhage, an occlusal syndrome has auxiliary value. The condition of cerebral blood circulation can be specified by means of a rheoencephalography, doppler sonography. To perform magnetic resonance imaging at newborns still difficult because of technical difficulties.

As additional methods of a research carry out a lumbar puncture, an oftalmoskopiya, a skull X-ray analysis. In an acute period define an acid-base condition and gas composition of blood, glycemia level, a gematokritny indicator, electrolytes (potassium, sodium, calcium, magnesium), creatinine, a kinase, LDG. In a heavy hypoxia reasonably monitor observation of a condition of the child. Complete blood count test allows to diagnose early anemia and to suspect development of an infection.

The differential diagnosis of a perinatal hypoxia is carried out with the following states:

1. pre-natal neuroinfection which can develop if mother during pregnancy had an infectious disease or has a specific infection,
2. intracranial or spinal birth trauma,
3. acute adrenal insufficiency,
4. injury of parenchymatous bodies,
5. phrenic hernia,

6. congenital heart diseases,
7. posthemorrhagic anemia,
8. inherited disorders of a metabolism (phenylketonuria, galactosemia),
9. congenital malformations of the central nervous system.

Treatment. Reasonably step-by-step treatment of children with heavy options of a hypoxia.

1 stage – in the delivery room at the child's birth the treatment is directed to restoration of warm activity and breath.

2 stage – in chamber of resuscitation and intensive care – it is directed to restoration of a hemodynamics, elimination of a metabolic acidosis, preservation of functions of the central nervous system.

Observance of temperature condition of the newborn in the course of resuscitation (the child is located under heat source) as overcooling is followed by generalized vasoconstriction is of special importance, aggravating acidosis.

Recovery of passability of airways begins with the head birth moment by suction of contents of a mouth and a throat a suction machine through a thin catheter. Evacuation of aspirirovanny masses from airways after the birth is carried out under control of the laryngoscope, assisted ventilation of easy 60% About₂ through a mask is carried out further. In a heavy hypoxia after recovery of passability of a tracheobronchial tree start artificial ventilation of the lungs by means of an intubation as mask breath for such patients is insufficient. In the course of IVL the sanitation of a tracheobronchial tree by introduction through a catheter is carried out to airways of 0.5-1 ml. 5% of solution of sodium bicarbonate, 2-3 drops of 2.4% of solution of Euphyllinum with the subsequent suction of aspirate each 5-10 min. For prevention of inflammatory changes in lungs through an endotracheal tube antibiotics in combination with corticosteroid drugs are entered. Appoint oxygenation of 40% of the Lake₂.

When carrying out IVL at premature it is reasonable to apply the oxygen-helium mix having anti-atelectatic effect, in the ratio 1:1 or 2:1, IVL duration - up to 1 h.

At the II stage of resuscitation in hard cases of respiratory insufficiency sometimes use a method of hyperbaric oxygenation. For the purpose of restoration of warm activity at its absence or bradycardia (less than 100) carry out indirect cardiac massage together with IVL and administration of antiatsidotichesky drugs to min. In 2-3 min. at its inefficiency vnutriserdechno or enter 0.5 ml into a vein of an umbilical cord. 10% of a gluconate of calcium and 1 ml. adrenaline.

Infusional therapy which main objectives is elimination of acidosis, normalization of a

hemodynamics, metabolic disturbances, restoration of volume of the circulating blood, its rheological properties, correction of electrolytic disturbances is at the same time carried out. Infusion of medicinal solutions is usually carried out usually by a puncture of an umbilical vein or its catheterization.

Treatment of children with a hypoxia is carried out depending on weight of a state. Correction of cardiovascular disturbances is important. After full oxygenation and elimination of acidosis, enter strophanthin or Korglykonum in a dose of 0.03 ml on 1 kg. masses in 0.9% sodium chloride solution intravenously slowly.

In bradycardia 0.05-0.1 ml of 0.1% Atropini sulfas solution are intravenously entered. If bradycardia does not disappear, the drug is administered repeatedly in combination with adrenaline. For improvement of a trophicity of a cardiac muscle include vitamins of group B, C, potassium drugs – Pananginum, the polarizing mix, Sulfocamphocainum.

For restoration of a vascular tone, appoint Prednisolonum or a hydrocortisone, in arterial hypotonia intravenously it is necessary to enter a dopamine (10 mkg/kg/min.) or 0.2% noradrenaline solution (20 mkg/kg/min.) into 10% glucose solution.

When assigning infusional therapy it is necessary to define daily need of the newborn for liquid – no more than 50-60 mg/kg, the speed of intravenous injections of 6-8 drops in a minute.

For restoration of microcirculation dextrans are used, at the same time it is necessary to enter saluretics (lasixum of 1 mg/kg) for prevention of an overhydration.

Fight against wet brain by administration of albumine, native plasma (10-15 ml/kg) in combination with diuretics is important. At decrease in level of sugar of blood lower than 2 mmol/l are necessary introduction of 10-15 ml. 20% of glucose and Prednisolonum of 0.5 mg/kg. Children with a hypotrophy and premature are inclined to a hypoglycemia in the first 3-6 hours of life.

It is reasonable to include intravenous administration of Cavintonum in a dose of 2.5-5 mg/days in complex treatment of children, a sermion of 4 mg/days, for the purpose of improvement of power exchange and increase in resistance of brain cells to a hypoxia, trental of 0.5 ml/days for improvement of microcirculation, nootropil of 1-3 ml/days for maintenance of exchange in neurons.

Vitamins of group B, E, C, lipoic acid, coenzymes – KKB are shown, to ATP.

Newborns with perinatal encephalopathy are subject to the further treatment in department of rehabilitation providing continuation of the course appointed in maternity hospital with its individual correction depending on the syndromal pathology which is formed at this stage.

At a syndrome of motive disturbances appoint 1% glyutaminovy acid on 1 tsp 3 times, ATP on 1 ml in oil, KKB on 25 mg in oil, 1% vitamin B₆ on 0.5 in oil, Dibazolum on 0.5-1 mg in day of 1-1.5 months, massage 3 courses (1 month, 3 months and in the 2nd half-year), physiotherapy exercises to 2 months of life, exercises in water from 2-3rd week of life (water temperature at the raised tone of 38-39 °C, at lowered 35-34 °C, in the 2nd half-year up to 32 °C).

At a spastiko-hypotonic syndrome it is recommended to appoint Mydocalmum in a dose of 5 mg/kg a day (a course of treatment up to 2 months).

At a syndrome of an arrest of development Encephabolum in syrup of 1/2 tsps in the 1st half-year is recommended, to 1 tsp in the 2nd half-year 1-2 months; Pyriditolum 0.25 x 2 times a day, piracetam (nootropil) in oil or in 30-50 mg/kg a day.

In treatment of patients use Cavintonum, Euphyllinum, B₁, B₆ vitamins, glyutaminovy acid, Dibazolum. Very sluggish patient appoint Cerebrolysinum in oil of 0.3-0.5 mg, Aminoaloniun of 1/4 t. 2-3 times in day of 1-2 months, mkg B₁₂ 50-100 vitamin a day 10-15 days, Cobamamidum (B₁₂ vitamin coenzyme) in a hypotrophy of 125-250 mkg in day in oil 7-10 days or 3 weeks inside in the same dose.

At an episindroma appoint phenobarbital of 1 mg/kg 2-3 times a day not less than 6 months against the background of dehydrational therapy (Diacarbum of 0.02 g/kg a day courses 3 days, a break 1 day) within 1-1.5 months. Dehydration is repeated short courses (7-8 days) – during critical periods (against the background of an acute respiratory disease, at a teething, receipt in children's collective). Resorptional therapy begins in 1 month after spasms. Appoint an aloe of 0.3-0.5 12-15 injections in oil, a lidaza p / to 8 units-16 units-32 units of 10-12 injections of 10-14 days.

At a gipertenzionno-hydrotsefalny syndrome recommend Diacarbum of 0.02 g/kg in day of 1 times in the morning 3 days, a break 1 day, a course of 1-1.5 months, in combination with potassium drugs – an asparkam, Pananginum. At intake of Diacarbum the metabolic acidosis therefore it is useful to appoint soda inside or in candles of 50-80 mg/days can develop. Appoint veroshpiron 2 mg/kg 2 days, a break 1 day, give 1-2 months, appoint with potassium drugs.

Apply also glitserol (brakes products of liquor) 1 tsp 3 times of 1-2 months, mixture with tsitralyyu, sulfate magnesia.

Use vascular means: a Vinpocetine or Cavintonum of 1 mg 2 times, cinnarizine or Stugeronum of 5 mg 2 times in day of 1.5-2 months, sernion 2 mg 2 times a day, komplamin 1/4 t. 2 times a day, trental of 1/4 t. 2 times a day. Appoint the means improving exchange in a neuron: vitamin B₆ of 1% 0.5 in oil or in 1-2 mg/kg in day of 1 month, pyridoxal phosphate of 2.5 mg

(1/4 tablets 1-2 times in day of 1-2 months), glyutaminovy acid of 5 mg (1/4t. 2 times a day.)

At a syndrome of the increased nervous reflex irritability appoint Relanium of 0.5 mg/kg a day, Seduxenum of 1 mg/kg a day, Tazepamum of 1 mg/kg a day, 1% bromic sodium of 1 tsp 3 times a day; mixture with tsitralyyu 1 tsps 3 times, broths of plants with sedative actions (root of a valerian, peony, a grass of a marjoram, a sage, mint, a motherwort).

At an asthenoneurotic syndrome it is shown: to excitable children – vitamins B, E, bromine drugs, carbonic and sulphidic bathtubs, meprobamate, Theralenum, Tazepamum, Elenium, to sluggish children – bathtubs with sea salt, vitamins B₆, With, caffeine, an eleuterococcus, Pantocrinum.

Consecutive landmark long-term treatment of children with perinatal encephalopathy promotes prevention of development of organic pathology and allows to cope with functional damages of the central nervous system successfully.

Forecast. The forecast depends on the reason which caused a hypoxia and from weight of clinical manifestations. In an acute intranatal hypoxia on condition of rational treatment the forecast, as a rule, favorable. At the children who had a chronic pre-natal hypoxia, the forecast for an absolute recovery should be done with care.

At modern methods of landmark treatment after the postponed hypoxia of 60-70% of children by 1 year have no neurologic deviations, 20-25% need treatment continuation, and at 1-6% heavy organic disturbances (cerebral palsy, deafness, a blindness, hydrocephaly) come to light.

Prevention. Prevention of a perinatal hypoxia comes down to antenatal protection of a fruit, timely and rational treatment of late toxicoses of pregnancy, decrease in frequency of not wearing out, timely delivery, the correct tactics of conducting childbirth.

Tasks for independent work:

1. Solve situational problems.
2. Make test tasks on a subject.
3. Examine the newborn child with a birth trauma or GBN. Describe in a workbook the deviations revealed by you in the state of health of your patient.

Scheme of inspection of the patient.

When collecting the anamnesis pay attention on:

- the centers of persistent infection at mother. Diseases during pregnancy.
- features of a course of pregnancy and childbirth (toxicoses, a fruit hypoxia, the newborn's

assessment on a scale Apgar);

- features of the period of adaptation in the early neonatal period;
- weight at the birth, features of feeding, an increase in weight.

At an objective research to pay attention on:

- physical and psychological development of the child;
- color of integuments and mucous;
- condition of a musculoskeletal system (existence of deformations, pathological mobility, pain at a palpation and the movement, restriction of mobility of one of extremities);
- existence of an edematous syndrome;
- tachycardia, circulator disorders;
- increase in a liver and spleen.

At interpretation of datas of laboratory:

- blood type and Rhesus factor of the child and mother;
- decrease in Hb;
- increase in maintenance of indirect fraction of bilirubin, dynamics of hourly gain of indirect bilirubin;
- hypoproteinemia (hypoalbuminemia), increase in level of globulinovy fractions;
- indicators of cerebrospinal liquid.

At additional inspection to pay attention on:

- X-ray inspection of a skull and bones of extremities;
- Ultrasonography of a liver and spleen;
- EEG;
- to neurosonogramm.

Situational tasks.

Task No. 1

The child of 4 days, from the I pregnancy, births in time. Body weight at the birth 3200 gr., length – 50 cm. At the birth integuments icteric, jaundice accrued by 4th day of life, the slackness and drowsiness was noted. Heart borders: upper – the II edge, left – on 1 cm of a knaruzha from the left mamillar line, right – the right parasternal line. Cardiac sounds are muffled. Pulse of 130 beats/min, satisfactory filling. Over a top of heart the gentle systolic noise is listened. In lungs – without features. The liver acts from under edge of a costal arch on 3 cm, a spleen – the muscle tone is reduced by 1.5 cm.

Mother's blood – B(III) Rh (-); blood of the child of O(I) Rh (+).

General blood test: Hb - 106 g/l, erythrocytes – $3.4 \cdot 10^{12}/l$, a color indicator – 0.9, reticulocytes – 3.5%, thrombocytes – $204 \cdot 10^9/l$, leukocytes – $9.7 \cdot 10^9/l$, eosinophils – 1%, p.b. - 20%, with / I am 69%, lymphocytes – 10%, monocytes – 10%, SOE – 8 mm/h.

Biokhimichesy blood test: straight line bilirubin – 10 $\mu\text{mol}/l$, indirect – 211 $\mu\text{mol}/l$, ALT – the 126th nmol/l.

Questions:

1. Make the diagnosis.
2. What pathogenesis of a disease?
3. Draw up the plan of additional inspection and treatment.

Task No. 2

The boy A., 4 days, came to department of pathology of newborns from maternity hospital concerning the profound jaundice.

From the anamnesis it is known that the child from the woman of 23 years having O(1) Rh-negative blood type. The father of the child has And (II) Rh-negative blood type.

The first pregnancy ended with medical abortion at the term of 10 weeks. The real pregnancy the second, proceeded with a gestosis in the second half. Births in time. Body weight at the birth 3200 gr., length of a body is 52 cm. Shout loud cried at once. By the end of the first day the ikterichnost of integuments and scleras is noted. On second day the jaundice amplified.

At receipt in a hospital serious condition, the expressed ikterichnost of integuments and scleras, the child sluggish, is marked out a hypomyotonia, hyporeflexia. The liver acts from under edge of a costal arch on 2.5 cm, a spleen at costal edge. Transitional chair.

General blood test: Hb - 141 g/l, Ayr. - $3.9 \cdot 10^{12}/l$, C. the item - 0.99, Leyk. - $9.4 \cdot 10^9/l$, p/ya-7%, with-53%, e e-1, l-32 of %, m-7 of %, SOE – 1 mm/hour.

Biochemical analysis of blood for the 2nd day of life: crude protein – 54.4 g/l, bilirubin: indirect – there are no 180 $\mu\text{mol}/l$, a straight line –.

Task:

1. Make and prove the diagnosis.
2. What examinations should be performed in addition for specification of the diagnosis?
3. To properly feed this child?
4. Appoint treatment.
5. What it is necessary to do zamenny blood transfusion in case of its need by? How to choose a blood type and Rh a factor?

Task No. 3

The girl L, came to a hospital at the age of 6 days.

From the anamnesis it is known that the child from the woman of 26 years, from the first pregnancy proceeding with toxicosis in the first trimester, a nephropathy. Childbirth in time, weakness of patrimonial activity, stimulation by oxytocin. the 1st period of 12 hours, the 2nd – 25 minutes, an anhydrous interval – 10 hours, in labor noted difficulty of removal of a coat hanger. Body weight at the birth 4200 gr., length of a body is 54 cm. Assessment on a scale Apgar of 7/8 points.

After the birth the child uneasy, is marked out hyper excitability, muscular dystonia, the volume of active movements in the left hand is reduced. In maternity hospital to the child treatment by sulfate magnesia of 25%, 5 ml in oil, phenobarbital 0.005 x 2 times, Vikasolum of 0.3 ml in oil No. 2 was carried out. For the 6th day the child is transferred to a hospital for further treatment.

At receipt condition of the moderately severe child. Pink integuments, marbling of the drawing. Umbilical wound dry. In lungs breath puerile. Rhythmical cardiac sounds. The soft stomach, a liver acts from under costal edge on 1.5 cm, the spleen is not palpated. Yellow, kashitseobrazny chair. A head circle – 37 cm, a big fontanel of 2x2 cm. A craniocereberal innervation without features. Reflexes of newborns: the oral automatism +, but palmar and oral is not caused at the left, grasping and Moro's reflex are reduced at the left. The muscle tone distonichen, in the left hand is reduced, the hand is brought to a trunk, the rotirovana inside in a shoulder, a brush in palmar bending is unbent in all joints. Active movements are limited in humeral and elbow joints. Movements in fingers are kept. The tendon jerk from a biceps is not caused at the left. On a support sits, the automatic gait is caused. Reflexes: crawlings +, protective +, spinal reflexes +.

General blood test: Hb - 221 g/l, Ayr. - 6.5kh 1012/l, C. the item - 0.97, blood clot. - 310.0Ö109/l, Leyk. - 8.2Ö109/l, p/ya-6%, with-56%, e-1%, l-30 of %, m-8 of %, mm/hour SOE-2.

Biochemical analysis of blood: crude protein - 55.0 g/l, bilirubin: there are no indirect -98 µmol/l, direct - urea - 4.0 mmol/l, potassium - 6.0mmol/l, sodium - 136 mmol/l, calcium - 1.05 mmol/l.

Neurosonogramma: not numerous echodense inclusions in subcrustal gangliya, echogenicity of periventrikulyarny areas, depth of a big occipital tank of 8 mm is raised (norm - 6 mm).

Task:

1. Your preliminary diagnosis?
2. What additional researches need to be conducted for specification of the diagnosis?
3. Appoint treatment.

Class in a subject:

"POLICLINIC. WORK IN THE OFFICE OF THE HEALTHY CHILD AND ON RECEPTION OF BABIES".

I. Scientific and methodical justification of a subject.

In modern pathology of children of chest age the problem of pre-natal infection, the neonatal sepsis which is characterized by special weight and high lethality did not lose relevance. Infection of newborns is promoted substantially: existence of the chronic centers of an infection at mother, an adverse course of pregnancy and childbirth, development of the perinatal pathology leading to a decompensation of homeostatic systems.

In terms of unity of mother and a fruit, the solution of extremely complex complex problems of the perinatal period depends not only on activity of the pediatrician, but also the therapist, the obstetrician. These circumstances explain need of serious studying diseases of the period of a neonatality of students of medical faculty.

II. Purpose of activity of students on occupation:

The student has to know:

- The contributing factors to development of perinatal pathology, contamination, sepsis in newborns;
- Main etiopatogenetichesky mechanisms of development of sepsis;
- Clinical manifestations of neonatal sepsis, local purulent defeats;

- Laboratory diagnostic methods of pyoinflammatory diseases and sepsis;
- The basic principles of treatment and prevention it is purulent - septic diseases of newborns.

The student has to be able:

- To purposefully collect the anamnesis;
- To perform objective examination of the newborn;
- To interpret laboratory and paraclinic data;
- To make the diagnosis;
- To make the treatment plan of the patient;
- To write prescriptions on the main medicines;
- To define preventive actions.

III. Content of training:

1. Nosological forms of local it is purulent - a septic infection.
2. Etiology and pathogenesis it is purulent - septic diseases in modern conditions.
3. Sepsis (definition, clinical forms, laboratory diagnostics).
4. Features of treatment in pyoinflammatory diseases and sepsis of newborns.
5. Prevention and dispensary observation of the sick transferred GVZ and sepsis.
6. Pre-natal infections (major etiological factors, features of a clinical picture).
7. Additional methods of a research of pre-natal infections at newborns.
8. Medical and preventive actions in pre-natal infections.

IV. Educational material security.

1. Visual aids: tables, schemes, multimedia presentations, videos, audiogramma.
2. Educational medical documentation (case histories, laboratory researches, roentgenograms).
3. Technical means of training.
4. Literature.

V. The list of the recommended literature.

1. Children's diseases: the textbook / under the editorship of A.A. Baranov. – M.: GEOTAR-media, 2009. – 1008 pages.
2. Pediatrics: The textbook for medical schools. Under the editorship of N.P. Shabalov. – SPb: SpetsLit, 2006. – 895 pages.

3. Propaedeutics of children's diseases / to N.A. Geppa. – M.: GEOTAR-media, 2009. – 464 pages.
4. N.P. Shabalov. Neonatology: Manual. – M.: MEDpress-inform, 2009.
5. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Technique of a research of the child. The study guide for students. – Vladikavkaz, 2011. – 51 pages.
6. Z.D. Kaloyeva, K.M. Dzilikhova, S.K. Karyaeva, etc. Scheme of a case history. The study guide for students. – Vladikavkaz, 2011. – 38 pages.
7. Lectures on pediatrics.
8. Methodical instructions for out-of-class work of students 4 courses of medical faculty on discipline "Pediatrics".

VI. List of questions for check of initial level of knowledge:

1. The Anatomico-physiological features of newborns contributing to the development of purulent - septic diseases.
2. The organization of work in chambers and departments of newborns in maternity hospital.
3. Asepsis and antiseptics in obstetrics.
4. Toxicoses of pregnant women.
5. Assessment of a condition of newborns on a scale Apgar.
6. Causes of a hypoxia of a fruit.
7. Course of pregnancy, childbirth and puerperal period in acute and persistent infections and diseases of the major bodies and systems.
8. Postnatal infectious diseases.

VII. List of questions for check of final level of knowledge:

1. Sepsis etiology it is also purulent - septic diseases in modern conditions.
2. Clinic of an omphalitis.
3. Clinical signs of a vezikulopustulez.
4. Concept about "presepsis", treatment. Risk group on sepsis.
5. Definition and scheme of the diagnosis of sepsis.
6. Clinical forms of sepsis (kliniko-morphological, biochemical characteristic).
7. Features of ways of infection of "entrance gate" and metastasis in sepsis in modern conditions.
8. Additional methods of a research, laboratory diagnosis of sepsis, it is purulent - septic diseases.

9. Basic principles of therapy of sepsis.
10. The choice of antibacterial therapy in sepsis.
11. Replacement therapy.
12. Principles of inspection and treatment of mother of the child sick with sepsis.
13. Recommendations about maintaining a healthy lifestyle during pregnancy.
14. Processing of an umbilical wound, skin care and mucous newborn.
15. The main requirements imposed to the medical staff servicing chambers of newborns.

Information block.

LOCAL PYOINFLAMMATORY DISEASES.

Etiology of the majority of these diseases – gram-positive microorganisms (stafilo- and streptococci), in 1/4-1/3 cases – gram-negative microbes (a klebsiella, colibacillus, psevdomonas, etc.).

Omphalitis – the purulent or serous inflammation of an umbilical wound, skin and a hypodermic fat layer around a navel, umbilical vessels which is followed serous or purulent separated, the infiltration and hyperaemia of an umbilical ring which is slowed down by wound epithelization. On a lobby of a wall of a stomach the red strips characteristic of the attaching lymphangitis, expanded superficial veins are visible, and at a palpation signs of defeat of umbilical vessels (are defined in thrombophlebitis of an umbilical vein round tyazh it is palpated on the average line of a stomach over a navel, and at a trombarteriita – from two parties: below a navel and sideways). The combination to incomplete fistula and fungusy a navel is possible.

Treatment. Locally – processing by aqueous and alcohol solutions of antiseptic agents (Furacilin, chlorofillipt, diamond green, permanganate potassium solution), lysozyme, use of the helium-neon laser, at considerable infiltration – Vishnevsky's ointment, at necrotic changes – oil sea-buckthorn and a dogrose. Fungus of a navel cauterize a stick of a lyapis once a day.

Taking into account sensitivity of the flora sowed from an umbilical wound and expressiveness of inflammatory process, antibiotics apply as locally (irrigations, ointments), and parenterally, at the same time hold events for stimulation passive (γ -globulin, anti-staphylococcal plasma, etc.), and later and active immunity. In toxicosis the active infusional therapy is important.

The forecast at early the begun therapy favorable, but in the remote period the portal hypertension is possible.

Navel fistula – congenital anomaly of development, is a consequence of not fusion of a vitelloduct or uric course, existing in the early embryonic period and obliterated by the time of

the birth. Fistula is full and incomplete. Full fistulas can be two types (from a nezarashchenny uric channel and a nezarashchenny channel between a navel and an intestinal loop (in the embryonic period the channel connects intestines to a vitellicle).

Clinical picture. After falling away of the funic rest find a fistular opening from which the mucous membrane of bright red color is stuck out and intestinal contents (full fistula of a vitellicle) follow. In incomplete fistula of the uric course at the bottom of an umbilical pole there is no spherical protrusion of a mucous membrane, and there is a site of the becoming wet surface with a fistular opening in the center. From this opening at a natuzhivaniye the stream emits urine. Incomplete fistulas proceed with the phenomena insignificant a moknutiya of a navel around which skin can be matserirovanny.

Treatment. Full fistula is subject to surgical treatment after establishment of the diagnosis, incomplete – 6 months since at the correct navel care, its perhaps independent closing are aged more senior.

Vezikulopustulez – a superficial staphyloderma of newborns. Process is localized in the mouth of ekkrinny sweat glands. In pathogenesis the astenisation and immune insufficiency at artificially raised children matter. The promoting factors most often are overheating, the increased perspiration, maceration.

Clinical picture are characterized by emergence of superficially located bubbles of 1-3 mm. filled in the beginning with transparent, and then muddy contents. Pustules more often multiple, are localized on buttocks, the lower part of a stomach, a hairy part of the head, in natural folds. Skin around elements is not changed, or is slightly hyperemic, the basis not infiltrirovano, and infectious process takes skin not more deeply than malpigiyevy network. Dynamics of development of a vezikulopustulez is characteristic: in 2-3 days after emergence the bubbles burst and the superficial erosion is formed, and then it dries up and is epithelized without the subsequent pigmentation. At early diagnostics and active treatment the general condition of children is not broken, the appetite of patients is kept, body temperature normal. However sometimes the disease progresses, the bladderwort, abscesses of sweat glands, pseudo-furuncles, metastatic suppurative focuses - otitis, pneumonia, osteomyelitis, etc. develop.

Treatment. It is not recommended to wash, bathe children in the period of a disease. The centers of defeat and probably healthy leather are processed antiseptic microbicides: solution of Furacilin, 0.1% solution of Rivanolum, 0.1-0.2% potassium permanganate solution, aniline dyes. Directly apply pastes with 1% of erythromycin, 1% of lincomycin on the centers of pustulous elements, to ointment with antibiotics.

Bladderwort of newborns (pempigus) – more severe form of the staphylococcal pyoderma proceeding it is benign or zlokachestvenno.

The benign form of a bladderwort is characterized by emergence at the end of the first week of life or is later against the background of erythematic spots of the bubbles and sluggish bubbles of 0.2-0.5 cm in size filled with serous and purulent contents. Localization – the lower part of a stomach, extremity, inguinal, cervical, etc. folds. Elements of a bladderwort are polymorphic, surrounded with hyperaemia nimbus, have the infiltrirovanny basis (all layers of skin are struck). More often pustules multiple, pouring out pushes. The general state is not broken, or moderately severe – slackness, appetite reduced, subfebrile temperature. At active and early the begun treatment in 2-3 weeks the recovery is noted though at first and it is benign the proceeding bladderwort can lead to metastatic suppurative focuses, sepsis.

The malignant form of a bladderwort which is called also piokokkovy pemphigoid develops in the same terms, as benign, but at it bubbles between which skin is exfoliated are observed sluggish, from 0.5 to 2-3 cm in size and more in the diameter. Febrile temperature, the general serious condition with prevalence in a clinical picture of the phenomena of intoxication and anemia. In peripheral blood are available the anemia of different degree of manifestation, a leukocytosis with a neutrocytosis and shift to the left increased by SOE. The disease highly kontagiozno and, as a rule, comes to an end with sepsis.

Exfoliative dermatitis of newborns (Ritter's disease) – a severe form of an epidemic bladderwort of newborns.

The clinical picture is characterized by a condition of an erythrosis with multiple bubbles, extensive erosive surfaces. Nikolsky's symptom positive. The sites of skin deprived of epidermis remind a burn of the II degree. Distinguish three stages of a disease: eritromatozny, exfoliative and regenerative. In hard cases the process proceeds septic with decrease in body weight, toxicosis, gastrointestinal disorders, anemia, a disproteinemia.

Treatment. Parenteral administration of the semi-synthetic penicillin having property to inhibit production of epidermolitichesky toxin and microbic flora, resistant to a penitsillaza, is obligatory. As antibiotics of specific anti-staphylococcal action use Fusidinum-sodium, lincomycin a hydrochloride and cephalosporins III-IV of generations. Along with antibiotics apply gamma-globulin, anti-staphylococcal plasma, anti-staphylococcal gamma-globulin, staphylococcal anatoxin. For the purpose of a detoxication enter by drop infusion in into albumine, reopoliglyukin, Polyglucinum, native plasma, 20% glucose solution, carry out hemosorption or a plasma exchange. In an intestinal dysbiosis appoint eubiotik

(bifidumbacterium, bifikol, baktisubtit, laktobakterin, etc.). Vitamin therapy is shown (ascorbic acid, pyridoxal phosphate, pantothenate or pangamat calcium, vitamins A and E).

External treatment and child care are of particular importance owing to contagiousness of process. Make daily shift of linen. Recommend daily bathtubs with solution of potassium of permanganate (1:10000). Bubbles open or suck away their contents the syringe. Leather around bubbles is processed aniline dyes, by 0.1-0.2% in alcohol r-m of a sangviritrin, 1-2% salicyl alcohol. The formed erosion subject Ural federal district with further processing by the ointments and pastes containing antibiotics.

Prevention. Careful observance of the hygienic mode. Obligatory quartz treatment of chambers. Whenever possible the children having staphylodermas are placed in boxes. Breastfeeding remains or at a hypogalactia at mother of the child transfer to donor breast milk.

Staphylococcal pyoderma. Distinguish superficial and deep forms. Carry an osteofolliculitis, a folliculitis to superficial; to deep – a hydradenitis, a furuncle, an anthrax.

Osteofolliculitis – purulent inflammation of the mouth of a hair follicle with formation of the superficial conic pustule penetrated in the center by a hair. At spread of suppuration deep into of a follicle there is a folliculitis. Deeper is purulent - necrotic inflammation of a hair follicle and surrounding fabrics with formation of a necrotic core is called a furuncle. The furuncle of an upper part of the face is dangerous because of a possibility of metastasis of an infection with emergence of a septic state with the meningitis phenomena.

Hydroadenitis – purulent inflammation of the apocrine sweat glands which are located more often in an anus, genitals. The same pathogenetic factors, as for all staphylococcal processes, but additional impact is made by the increased perspiration, alkali reaction of sweat.

The streptococcal pyoderma is shown by the basic primary pustulous element – a phlyctena. The most often found types of a pyoderma at children are superficial streptococcal defeat – impetigo and deep – an ecthyma. Streptococcal impetigo is shown by a superficial bubble – a phlyctena. Localization: face, skin of a trunk, extremity. In corners of a mouth of a phlyctena are quickly opened, and the erosive surface is transformed to a longitudinal crack (perleche). On nail phalanxes of brushes of a phlyctena podkovoobrazno surround a nail, form periungual impetigo (turniol). At the combined superficial streptostafilokokkovy infection there is vulgar impetigo differing in considerable contagiousness, tendency to dissimulation on various sites of an integument.

Osteomyelitis sharp – purulent inflammation of elements of a bone. Any pyogenic microorganism can be the activator.

Clinical picture. The disease begins sharply. The first symptom is sharp extremity pain from which the child shouts and avoids any movements. At newborns it is shown by concern when they are taken on hands or shifted. Body temperature rises up to 39-40 °C. Vomiting, a diarrhea are observed. External symptoms of osteomyelitis can be absent in the beginning. In process of process development when it passes to soft tissues, there is a local swelling, the extremity configuration changes. Skin becomes hydropic and hyperemic. The next joint is deformed.

The clinical course of acute osteomyelitis depends on a variety of reasons: virulence of a microorganism and reactivity of a macroorganism, etc. Distinguish three forms of a disease: toxic, septicopyemic, local. The first is characterized by the rough beginning, the sepsis phenomena prevail, and the patient quite often dies earlier, than local changes managed to be shown. The second form is observed more often than others; the local manifestations which are combined with all-toxic reaction sometimes are accurately significant several bones are surprised at once, purulent metastases in other parts of the body are observed. The third form differs in an easy course with primary expression of local reaction.

Diagnosis. Recognition is complicated at newborns. At suspicion of osteomyelitis at them especially carefully investigate the ends of tubular bones and joints. Diagnostics is specified by X-ray inspection. Precursory radiological symptoms appear at children for 7-10 day of a disease. In an onset of the illness in blood – a leukocytosis with deviation to the left; however in hard cases quite often there is a leukopenia.

Surgical treatment.

Dacryocystitis of newborns – inflammation of a dacryocyst. The reason – incomplete disclosure of a nasal duct by the time of the birth. It is shown by a slezostoyaniye, mucopurulent separated at an internal corner of an eye. When pressing the purulent contents are allocated for area of a dacryocyst from lacrimal openings.

Treatment. Massage of area of a dacryocyst in the direction from top to down for a rupture of a film and recovery of passability of a nasal duct. If the passability is not restored within a week, the child needs to be directed to the oculist for sounding and washing of plaintive ways.

Paraproctitis – pararectal fat inflammation. At children it is observed less than at adults, and usually has the nature of hypodermic abscess. Implementation of an infection is promoted by maceration of skin and an intertrigo.

Clinical picture. On the limited site of skin about an anus there are a consolidation and hyperaemia which is followed by pain during defecation. The child becomes uneasy, the general state is sometimes broken, body temperature increases up to 38 - 39 °C. Pains gradually accrue.

The diagnosis a paraproctitis without effort is recognized during survey of a crotch. Sharp strengthening of pain when pressing is characteristic. Sometimes at the same time pus from an anus is emitted.

Treatment consists in perhaps earlier opening of abscess. Sometimes there is a spontaneous opening of an abscess. Thus after subsiding of the inflammatory phenomena there is fistula with purulent separated. In such cases speak about a chronic paraproctitis.

Phlegmon of newborns – the peculiar inflammation of hypodermic cellulose observed in the first month of life. Any pyogenic microorganism can be the activator, staphylococcus is more often.

Clinical picture. The disease begins sharply. The child is uneasy, loses appetite, body temperature rises up to 39-40 °C. On skin there is a limited site of reddening and consolidation, painful to the touch. The most frequent localization – sacrococcygeal area, a breast, a neck. The inflammatory center quickly increases on the area. Skin over it gets a crimson, then cyanochroic shade. For the 2-3rd day in the center of defeat there is a site of softening. In this place the skin melts quickly and formed fistula through which nekrotizirovanny fabrics are torn away. At the edges there is amotio of skin which is exposed to fusion, and the deckle-edged extensive wound is formed. Quite often necrosis extends deep into and in breadth, bones are bared.

Surgical **treatment.**

Sepsis of newborns.

Sepsis is a generalized polyetiological infectious disease with an acyclic course, existence of primary pyoinflammatory center, emergence and which course are defined by features of a macroorganism and properties of the activator.

Clinical classification. In classification of sepsis by time of emergence allocate pre-natal and post-natal sepsis.

Pre-natal sepsis is understood as the disease which already developed in utero i.e. is antenatal, at in utero the infected fruit. And in this case primary pyoinflammatory center is out of the child's organism (the chorionitis, horioamnionit, a placentitis, etc.).

Pre-natal infection is the state which is characterized by microbic contamination (planting) of a fruit. Contamination by microorganisms of a fruit can antenatal happen (that is noted seldom) or intranatalno (during childbirth) that meets considerably more often.

Depending on a condition of reactivity of a macroorganism, virulence and extent of colonization (microbic settling) at intranatal pre-natal infection in post-natal the period of life of the child there are processes gradually of replacement of this microflora by saprophytes, or the carriage of this flora is formed, or at failure of compensatory protective mechanisms of a macroorganism the pyoinflammatory disease (localized or sepsis develops). It is necessary to emphasize that the disease, per se, develops in the post-natal period and if it is sepsis, is considered as post-natal sepsis.

In post-natal sepsis there is always primary pyoinflammatory center. Depending on its localization allocate umbilical, otogenic, skin, pulmonary, intestinal, an urosepsis, etc. At newborns and babies the umbilical sepsis meets with primary center in the field of an umbilical wound and/or umbilical vessels more often. In recent years the infected blood clots, thrombophlebitises arising in connection with catheterization of veins for performing infusional therapy and also mucous a gullet and intestines are primary septic center quite often. Almost all types uslovnopatogenny and some pathogenic microorganisms can be the cause of sepsis: staphylococcus, streptococci, bacteria of intestinal group, group of pseudo-monads, anaerobe bacterias, etc.

In **an etiology** of sepsis of newborns the leading place is taken (to 50%) borrow staphylococcus (36%) gram-negative flora, generally enterobakteriya is on the second place. The mixed disease etiology has a certain value (up to 10%).

The etiology of a disease leaves a certain mark on a clinical picture of a disease, its outcome, defines the choice of antibacterial therapy. Therefore establishment of the etiological diagnosis of sepsis is obligatory.

Clinical forms. Most of authors distinguishes two clinical forms of sepsis – a septicaemia and a septicopyemia.

Septicaemia – sepsis without metastases. Clinical symptoms can not have strict specificity since depend on properties of a microorganism and also on extent of disturbance of parameters of a homeostasis.

Bacteremia, in the course of settling of an organism of the newborn child microbic flora, can arise as a short-term episode practically at each child, including at almost healthy (the so-called "symptomless bacteremia" revealed on average at 15% of newborns of the first week of life).

Development of sepsis (septicaemia) depends not so much on the fact of circulation of bacteria, and on ability to clarification and speed of clarification of blood and a lymph from

microbes the eliminiruyushchy systems of an organism. Dysfunction of the last is the cornerstone of development of sepsis.

The septicaemia which developed as a result of pre-natal infection in the first days of life is followed by serious general condition, the progressing central nervous system function oppression, a hypothermia, is more rare – a hyperthermia, pale or dirty-gray coloring of the skin which early is shown and quickly accruing jaundice, the progressing edematous syndrome, increase in a liver and spleen, respiratory insufficiency in the absence of the significant radiographic changes. Vomiting, vomiting, a hemorrhagic syndrome can be noted.

The sepsis which developed after the birth is more often characterized by more gradual beginning. After implementation of the infectious agent the eclipse period is 2-5 days, at premature children it is extended up to 3 weeks. Therefore in a clinical picture of sepsis conditionally allocate disease harbingers, early symptoms and a heat of process.

It is possible to refer decrease of the activity, appetite of the child, vomiting, local symptoms to harbingers of a disease. Assessment of an umbilical wound as places of primary implementation of an infection, presents the greatest difficulties since the gram-negative flora prevailing now does not give the significant local inflammatory reaction. Late falling away of the umbilical rest (after the 6th day at full-term and the 10th at premature), a condition of a bottom of a wound (consolidation, protrusion or sharp retraction), character and duration separated, preservation of a dense crust after the 16-18th day of life is considered. Existence of inflammatory process is demonstrated pastosity of fabric in the lower segment of an umbilical ring, by emergence or strengthening of venous network on an anterior abdominal wall, especially on the right, tension of a direct muscle of a stomach above an umbilical ring or direct muscles is lower than it. In thrombophlebitis it is possible to propalpirovat the condensed umbilical vein. At a palpation of vessels from the periphery to the center at the bottom of an umbilical wound the purulent discharge can appear. Symptoms of damage of an umbilical wound and vessels, being almost continuous manifestation of umbilical sepsis, in itself do not serve, however, as criterion of generalization of an infection.

Development of sepsis is characterized by appearance of infectious toxicosis – slackness or concern, temperature reaction, vomiting, dyspepsia, an edematous syndrome or, on the contrary, eksikozy, a disrefleksiya, dystonia.

In the period of a disease heat the toxicosis even more amplifies. The toxic defeat of separate bodies which is not manifestation of a septicopyemia comes to light. So, for example, the abnormal liver function (hepatomegalia, jaundice, increase in direct bilirubin and

transaminases) is noted; dispeptic disorders; reaction from kidneys (oliguria, a proteinuria, a leukocyturia, an erythrocyturia).

Accession of pneumonia as independent intercurrent disease with an aero bronchogenic way of infection can complicate a septicemia. Against the background of a septicemia the respiratory disturbances caused by disturbances of microcirculation in lungs, metabolic changes in a myocardium are also possible.

The septicopyemia is characterized by presence of purulent metastases (elimination centers). The most frequent localization of the pyemicheskyy centers are a meninx, lungs, bones, a liver, other bodies are more rare. Now change of the activator with gram-negative flora affected features of metastasis: cases of development of purulent meningitis with poor kliniko-likvorodinimicheskyy symptomatology at the beginning of a disease became more frequent that demands repeated spinal punctures. At reduction of frequency of such metastatic centers as epiphyseal osteomyelitis that is more typical for staphylococcus, metafizarnyy and epiphyseal defeats, slow, difficult for diagnostics, proceeding quite often without distinct arthritis became more frequent, with poor and indistinct radiological changes. In similar cases become the leading symptoms overextension of extremities in a joint and a pain syndrome at passive movements. Only on the third week there can be a periosteal reaction defined to the touch and radiological.

Sepsis course. In a course it is possible to allocate the lightning course of sepsis leading to a fatal outcome within 3-7 days sharp (lasting 4-8 weeks) and long.

Development of septic shock is characteristic of a lightning course of sepsis, as is a proximate cause of death of the patient.

Clinically shock is shown by catastrophic increase of weight of a state, sharp pallor of the skin amplifying in distal departments, fall of temperature of a body to subnormal figures, fast change of tachycardia with bradycardia, increase of dullness of cardiac sounds, emergence and fast generalization of a sclerema, emergence of an oliguria, bleeding, the progressing respiratory insufficiency, emergence of a picture of a fluid lungs ("a shock lung") owing to deep disturbances of microcirculation, microcirculator blockade of kidneys, lungs that is expressed in development of an acute renal failure and fluid lungs.

At the heart of pathogenesis of this option of sepsis – inadequate reaction of an organism to infectious process, failure of protective mechanisms. The role of a hormonal imbalance (a hypophysis – a thyroid gland – adrenal glands) in origin of septic shock is established.

At sharp a course of sepsis the initial stage, the period of a heat, restoration and recovery are allocated.

The clinic of an initial stage at a sharp course of sepsis is characterized by existence of primary center and gradually accruing general changes. In the period of a heat of manifestation of toxicosis are most expressed, its characteristic features are disturbance of thermal control, the central nervous system function, breath, a hemodynamics and a GIT.

The period of restoration is characterized by sanitation of the metastatic centers, gradual subsiding of toxicosis, at the same time increase in a liver and spleen, a flat weight curve remains. In the period of a convalescence the restoration of function of all bodies and systems, normalization of coloring and turgor of skin, a body weight increase is noted.

Laboratory diagnosis of sepsis. The most informative are changes of peripheral blood. In early terms of sepsis as imparted, the moderate leukocytosis, anemia most often insignificant takes place or is absent. In the heat of a disease the number of leukocytes accrues, the deviation to the left is noted, myelocytes, toxic granularity of neutrophils appear. During this period a constant symptom – anemia. At premature newborns, especially at a gram-negative etiology of a disease, the leukopenia can be observed (up to $4,0 \times 10^9/l$).

Thrombocytopenia is also not absolute criterion of sepsis.

Some importance is represented by researches of the fermental status of leukocytes, especially alkaline phosphatase of neutrophils which indicators raise in sepsis. However this test has no absolute importance as in local purulent infections also is positive.

Implementation of immunological diagnostic methods in clinical practice allowed to develop methods of early diagnosis of sepsis. Treat them:

- decrease in percent of digestion in neutrophils and monocytes of peripheral blood (at a research of phagocytal activity) is more than twice lower than norm;
- increase in quantity a neutrophil and the monocytes giving positive reaction to nitro-blue tetrazoly (NST-test) over 70%;
- decrease in quantitative and functional indices of T lymphocytes more than twice in comparison with norm.

Crops of blood in diagnosis of sepsis play an important role and define a disease etiology.

Treatment.

1. Patients are placed in the separate box for an exception of cross infection.
2. Rational antibiotic treatment taking into account sensitivity of the activator (a course about 3 weeks). In the first days the antibiotics enter intravenously.
3. To children with manifestation of a thrush surely appoint antifungal drugs (fluconazole, ketokonazol).

4. Food native breast milk.
5. Disintoxication therapy:
 - plasma exchange with replacement of plasma of the patient by freshly frozen;
 - infusion of a reopoliglyukin, 10% of solution of glucose, plasmas (5-10 ml/kg), 5-10% albumine solution.
6. A specific passive immunotherapy – in/in administration of the drugs Ig (immunoglobulin human normal) on a course of 5-7 infusions.
7. In staphylococcal sepsis the administration of hyperimmune anti-staphylococcal gamma globulin in oil on 1 dose, 3-6 injections within 2-3 days is shown, also repeated administration of anti-staphylococcal plasma in a single dose of 5-8 ml/kg is possible.
8. Infusional therapy under control of KSS and electrolytic composition of blood, Ht, the ECG.
9. Glucocorticoids a short course (5-7 days including the dose decline period) appoint at septic shock. At the rate of 1-2 mg/kg/days.
10. At signs of hypercoagulation antiagregant (Dipiridamolum), in/in middlemolecular dextrans (reopoliglyukin), heparin sodium in a dose of 150-300 Pieces/kg/days appoint.
11. Prevention of complications of antibiotic treatment – group B vitamins, vitamin C, E, bifidumbacterium.
12. After subsiding of the sharp phenomena carry out the stimulating and antioxidant therapy (orotovy acid, a carnitine, thioctic acid, taktivin), massage, remedial gymnastics.

Scheme of inspection of the patient.

When collecting the anamnesis to pay attention on:

- the centers chronic infections (especially urogenital), mastitis at mother. Diseases during pregnancy, a puerperal period (temperature);
- features of a course of pregnancy and childbirth (toxicoses, a fruit hypoxia, the newborn's assessment on a scale Apgar, an inoculation of test of tuberculosis);
- it is purulent - septic diseases of skin, an umbilical wound, eyes in maternity hospital and in the period of a neonatality, an intertrigo;
- feeding, increase in weight. Weight at the birth, anorexia;
- temperature curve, incidence (frequency);
- nature of a chair.

At an objective research to pay attention on:

- psychological and physical development of the child;
- presence of intoxication, infection, toxicosis. Nature of a temperature curve;
- color, purity of skin, mucous, umbilical wound, nature of a chair, condition of umbilical veins, arteries;
- edematous syndrome;
- tachycardia, circulator disorders, intestines paresis;
- increase in a liver, spleen of lymph nodes;
- existence of metastatic suppurative focuses (osteomyelitis, pyelonephritis, encephalomeningitis);
- IDCS (hyper - hypocoagulation).

When reading datas of laboratory:

1. To pay attention on:

- neutrophilic or lymphocytic leukocytosis, acceleration of SOE, decrease in Hb;
- disproteinemia (a gipoalbumiyemiya, increase in level of globulinovy fractions by 1.5 - 2 times and the maintenance of fractions within age norms);
- shifts in mesenchymal reactions (SRB,DFA);
- change in a koagulogramma;
- bacteriological researches (urine crops, a calla, from skin, blood, an umbilical wound, mucous, etc.);
- serological researches with the allocated activator.

2. To pay attention on:

- X-ray inspection of a skull (signs of the increased intracranial pressure);
- bones of extremities (osteomyelitis);
- kidneys (symptoms of pyelonephritis);
- lungs (pneumonia).

Tasks for independent work:

1. Solve situational problems.
2. Make tasks for test control on a subject.
3. Examine the newborn with pyoinflammatory diseases, describe the changes revealed by you. Make the plan of inspection of your patient.

Situational tasks.

Task No. 1

Sick K. came to department of pathology of newborns at the age of 1 day.

From the anamnesis it is known that the child from mother of 19 years having pyelonephritis. The first pregnancy, proceeded with toxicosis in the first and exacerbation of chronic pyelonephritis in the third trimesters. Births in time, the 1st period of childbirth of 13 hours, the 2nd – 25 minutes, an anhydrous interval – 7 hours. Back waters greenish, with an unpleasant smell. Body weight at the birth of 2850 gr., length of a body is 49 cm. Assessment on a scale Apgar of 6/7 points.

At primary survey the decrease in physical activity, repeated attacks of asphyxia attracted attention. Integuments pale with a grayish shade, marbling of the drawing, a Crocq's disease, cyanosis of a nasolabial triangle, an asthma with retraction of intercostal spaces, department of foamy slime from a mouth. In maternity hospital the infusional and antibacterial therapy is begun, for further treatment the child is transferred to a hospital.

At survey by the end of the first day of life serious condition, shout weak, sucks inertly. Hypothermia. Gray integuments, the profound cyanosis of a nasolabial triangle, wings of a nose are strained. Shallow breathing, 80 in a minute, with the apnoea periods. Retraction of intercostal spaces, epigastric area. Percussionly over lungs the shortening a sound is defined, auskultativno – breath is weakened, on a deep breath crepitant rattles are listened. Cardiac sounds are muffled, rhythmical, by PS – 170 1 minute. The soft stomach, is available to a palpation. The liver acts from under costal edge on 2 cm, the spleen is not palpated. In the neurologic status: the slackness, an adynamia, a hypomyotonia, unconditioned reflexes are reduced.

General blood test: Hb–180 of g/l, Ayr. - 5.5•10¹²/l, C. item-0.9, blood clot. - 208.0•10⁹/l, Leyk. - 23.1•10⁹/l, % myelocytes-2, % metamyelocytes-4 p/ya-13%, with-6%, e e-5, l-11 of %, % m-15, SOE-4mm/chas.

Acid-base condition of blood: ro-55 ₂mm Hg, rSO-70 ₂mm Hg, rn rn-7.21 BE-18mmol/l, AB-9mmol/l, SB-8mmol/l, BB-19mmol/l.

Task:

1. Make the diagnosis to this child.
2. List the contributing factors which contributed to the development of this disease.
3. Appoint treatment.

Task No. 2

The boy Yu., 1 month, is in a hospital.

From the anamnesis it is known that the child from mother of 24 years. The first pregnancy, proceeded with toxicosis in the first trimester, in 24-26 weeks of a gestation the temperature increase without the catarrhal phenomena was noted, it was not treated. Childbirth on the 37th week, in head presentation. the 1st period - 8 hours, the 2nd - 25 minutes, an anhydrous interval - 9 hours, amniotic waters light. Body weight at the birth of 2700 gr., length of a body is 48 cm, a head circle - 35 cm, a thorax - 32 cm. Discharged from maternity hospital for the 7th day of life.

Houses the child was sluggish, sucked on 60-70 ml for one feeding. The increase in weight for the first month made 200 g. At visit of polyclinic the increase in the sizes of the head is revealed, and for inspection the child was hospitalized.

At receipt serious condition. Light pink, dry skin. Food is lowered, the hypodermic and fat layer is pierced. Head of a hydrotsefalny form. A head circle - 40 cm, a thorax - 34 cm, the sagittal seam is open on 1 cm, coronal - on 0.2 cm, a big fontanel of 4x4 cm, is executed, a small fontanel - 0.5x0.5 cm. The symptom Gref, a horizontal nystagmus is expressed. The tone of extensors prevails. The liver acts from under costal edge on 2.5 cm, a spleen - on 1 cm.

Research of cerebrospinal fluid: the transparency is muddy, protein of-1660 g/l, a cytosis - 32 in 3 mkl: neutrophils - 5%, lymphocytes - 27%.

RSK with toksoplazmenny antigen: at the child - 1:64, at mother - 1:192.

Task:

1. Of what disease it is possible to think in this case?
2. What additional examination should be performed for specification of the diagnosis? Possible results?
3. Estimate results of a research of liquor?
4. What principles of treatment of this disease?

Task No. 3

The girl D, 13 days, is in department of pathology of newborns.

From the anamnesis it is known that the child from mother of 24 years having genital herpes. The first pregnancy, proceeded with exacerbation of herpes in 36-37 weeks of a gestation. Births in time, in head presentation. 1 period - 7 hours, the 2nd - 25 minutes, an anhydrous interval - 12 hours. Near-water waters light. Assessment on a scale Apgar of 7/8 points. Body weight at the birth of 2950 gr., length of a body is 51 cm, a head circle - 35 cm, a thorax - 32 cm. In the period of early neonatal adaptation the hyperexcitability, a krupnorazmashisty tremor of hands, a divergence of a sagittal seam on 0.3 cm, a big fontanel 2x2, small 0.3x0.3 were noted.

For the 3rd day of life there was jaundice with a tendency to increase in this connection for the 5th day of life the being able moderately severe child is transferred to a hospital.

For 13 day of life on a trunk, extremities, mucous a mouth vesicular rashes with a dense tire and transparent contents developed. In 3 days the state worsened to heavy, temperature rise to 38.3 °C, a hyperesthesia, kloniko-tonic spasms was noted.

The general blood test for the 6th day of life: Ayr. - 4.6kh 1012/l, Hb - 172g/l, C. the item - 0.94, Blood clot. - 310.0Ö109/l, Leyk. - 10.0Ö109/l, p/ya-1%, with-30%, l - 54%, m-15 of %, mm/hour SOE-2.

Biochemical analysis of blood: crude protein – 60.0 g/l, bilirubin: the general –310 µmol/l, indirect – 298 µmol/l, a straight line – 12mkmol/l, urea – 4.2 mmol/l, cholesterol – 3.6mmol/l, potassium – 5.1 mmol/l, sodium of-141 mmol/l.

Research of cerebrospinal fluid for 12 day of life: the transparency is muddy, protein – 1650 g/l, Pandi's reaction +++, a cytolysis – 350 in 3mkl: neutrophils of-25%, lymphocytes of-75%.

Task:

1. Of what disease more likely it is possible to think in this case?
2. What additional examination should be performed for specification of the diagnosis?
3. Estimate results of the general blood test, a liquor research?
4. Estimate result of biochemical analysis of blood. What can the revealed changes be caused by?
5. What medical tactics in this case?