MINISTRY OF PUBLIC HEALTH OF UKRAINE
O.BOHOMOLETSNATIONALMEDICALUNIVERSITY

PEDIATRIC DEPARTMENT N5

"Approved"
on the methodical conference
Department of Pediatrics № 5
protocol №2 from 07.09.2016

GUIDELINES FOR STUDENTS

Academic discipline	Pediatrics
Study subject	Clinical examination in Pediatrics. Physical development. Feeding of the children. Newborn baby
Course	4
Faculty	Dental

Kyiv-2016

Actuality of the topic:

1. Despite the introduction to the practice of laboratory and imaging studies, the clinical examination of patients is the most important in the diagnosis. Competently and completely collected complaints, anamnesis of disease and life are of great importance in the formulation of the preliminary diagnosis and determining the amount of paraclinical examination of the patient.
2. Physical development is one of the integrative health indicators of biological maturity of all body systems. The study of the formation of the nervous system in children and reflex activity of a healthy child are very important in the work of the pediatrician. These skills facilitate to assess the psychological development of children of various ages, identify rapidly any pathologies and eliminate them.
3. Feeding of the children is one of the most important problems in pediatrics. Special attention should be paid to the proper organization of nutrition of infants. The feeding habits of the child in the first year of life largely determines the future health and even his mental development and resistance to inflectious and somatic diseases.
4. Neonatal period i the most important and critical period of life. In the adaptation of the child to the conditions of extrauterine life involves all the major functional systems. A transient reorganization of their activities is the essence of the neonatal period. Knowledge of these features allows the doctor to interpret correctlythe processes occurring in the neonatal period.

Questions for self– checking

1. The main indicators of physical development.
2. The main indicators of psychological development.
3. Methods of basic anthropometric measurements and the necessary equipment
4. Regularity of increase in the mass and body length, chest circumference and head in different age periods
5. Factors affecting the physical development of children
6. Anatomical and physiological characteristics of the formation of the central nervous system in children.
7. Formation of physical activity.
8. The disruption and central nervous system malformations.
9. Natural, mixed and artificial feeding.
10. Regime diet of young children.
11. Complementary food for infants
12. The main products for artificial feeding of children.
13. The benefits of breastfeeding.
14. The differences between breast milk and cow's milk.
15. What is the term delivery?
16. Gestational age of a full-term baby.
17. Gestational age of a premature baby
18. Gestational age of a post-term baby
19. Morphologic criteria of maturity newborn.
20. Criteria for functional maturity newborn.

21. What are the transient states?
22. What are normal vital signs of a newborn?
23. What is the Apgar score?

Topic 1

1. **ANAMNESIS**
   During the examination of the child certain guidelines should be observed.
   1) The room in which the examination is carried out, should be warm (temperature 24, 25), free from drafts and outside noise, preferably with natural light or fluorescent light.
   2) Examination is carried out in the presence of parents.
   3) Hands of a doctor should be clean, warm, with close-cropped nails.

Questioning

Examination always begins with questioning. Young children should be interested toy. Typically, the physician receives data about the disease from the child's parents. On questioning certain sequence must be followed.

PASSPORT PART. Date and time of admission to the hospital the patient
surname, first name, patronymic of the child, age, date of birth, home address.

ANAMNESIS MORBI
• child complaints
• When a child gets sick.
• Characteristics of the disease
• Common manifestations of the disease (fever, lethargy, anxiety, sleep, appetite, thirst, chills).
• Manifestations of the disease on the part of all organs and systems:

The conclusion of anamnesis of the disease: the assumption of the defeat of certain systems and organs.

HISTORY OF LIFE (ANAMNESIS VITAE) -
History of the child's life.

- Features of the pregnancy,
- information about previous pregnancies,
- features of delivery,
- newborn characteristic.
- Diseases in the newborn period.
- The physical development of the child in the first, second and third year of life (weight gain and body length).
- The development of motor skills and statics.
- Mental development.
- Type of feeding in the first year of life: natural, artificial, mixed.

- Postponed diseases.
- Vaccinations.
- Allergic reactions

Family history. The age of the parents. The health status of the parents and close relatives on the mother and father. Presence of infectious (tuberculosis, venereal disease, toxoplasmosis, etc.), psychiatric, cancer, nervous, endocrine, and other allergic diseases in the family. The presence of these unfavorable factors, such as alcoholism, smoking, potential occupational hazards.

The general conclusion by anamnesis vitae: the defeat of what the system we can assume an acute or chronic disease, what negative factors of the history of life can contribute to the development of this disease or that can aggravate it.

2. OBJECTIVE EXAMINATION (STATUS PRAESENS objectives) (The present state of patient).

(inspection, palpation, percussion, auscultation).

The child is face to light source at the examination..

General examination (inspection): state of health, posture, gait disorder, the presence of minor abnormalities of development.

State of health–

- **satisfactory** - a clear conscience, active, no signs of intoxication or they are minor, there are no functional impairment of body systems.
- **Moderate** condition - clear consciousness, decreased activity, distinct signs of intoxication, the presence of functional disorders of the body systems.
- A **serious** condition - the various stages of impaired consciousness, severe intoxication, decompensation of systems.
- Condition was **extremely grave** - the aggravation of these phenomena and the signs of threatening the life of a child.

The position of the body–

- The **active** - patient easily change the position of the body, makes active movements, takes a relaxed pose.
- **Passive** position - patient can not change its position without assistance.
- Position **forced** - a special position, which takes the child to relieve their condition (for example, pose a gundog with meningitis, the position of orthopnea in bronchial asthma).

Body temperature

The nervous system.

Consciousness:

- **clear**- the child is easily oriented in time and space.

- **Stuporous** consciousness (somnolence) - a state of stupefaction, the reaction of the surrounding is slowed down, the child answers the questions a little, responding to severe irritation by cry.
- **Soporous** consciousness - no reaction to his surroundings, but is retained response to pain stimuli.
- **Coma** - loss of consciousness, significant inhibition of the cerebral cortex.

Neuropsychological development (for young children).

- **Mood** (smooth, calm, elated, excited, unstable), contact with others kids, interest in toys, environment, and behavior.
- **Reaction** to the examination - adequate, inadequate.
- The width of eye slits, strabismus, nystagmus, the size of the pupils, their reaction to light;
- **Appetite** is satisfactory (good, high - bulimia, low – anorexia)
- **Mood** is good (quiet, depressed, excited).
- **Sleep** is quiet, deep (with difficult)
- **Intellect** is middle (high, low),
- **Memory** is good (excellent, infringe)
- **Speech** is in middle (quickly, slowly) temp, right (with many mistakes), rhythmical or not.
- Craniocerebral and peripheric nerves state (it is described in the case of appropriate pathology presence).
- Reflexes cutaneous, tendon, from mucos tunic, painful points investigation.
- Pathological reflexes and symptoms.
- Mobility: paralysis and paresis.
- Unconditioned reflexes of newborns
- Meningeal syndrome.

Clinical examination of the organs and systems is carried out below:

- Skin and visible mucous membranes.
- Subcutaneous fat layer.
- Lymph nodes.
- Muscle-skeletal system. Anthropometry.
- The respiratory system.
- The cardiovascular system.
- Digestive organs.
- The urinary system.
- The endocrine system.

Topic 2.

Physical development is a dynamic process of growth and biological maturation of a child in a different period of childhood. While assessing the physical development of the child, one should know his height, weight, proportion of body parts.

The **main criteria** for the physical development of a child are: 1. Body weight; 2. Body length; 3. Head circumference; 4. Chest circumference; 5. The balance (proportionality) of these parameters.

Body weight. Body weight of full-term newborns ranges from 2600g to 4000g, and the average is 3-3.5 kg. Normally, during 3-4 days after birth, the physiological weight loss is observed in most of the children. The maximum weight loss is seen on the third day and accounts for 6-8% of total body weight at birth. Loss of more than 10% of birth weight is considered to be abnormal and is evident as the disease or some lack of children's care. The subsequent gaining of body weight can be achieved in several ways. Further increase in the weight continues after restoration. On the average a one-month increase of the mass is 600 grams.

Dynamics of body weight is characterized by greater increase in the first 6 months of life and by lesser one closer to the first year. Body weight doubles to 4.5 months' and triples to the year

To calculate the weight of the child the doctor uses a certain formula. Weight increases by 800 g per month in the first half, second half - by 400 g.

M = m + 800 x n

M = m + 800 x 6 + 400 x n, n – months of age

Example: body weight at birth was 3500 g, and now his age is 9 months, thus at this age, body weight should be equal to 3500 + 800 x 6 + 400 x 3 = 9500 g.

After a period of breastfeeding before the age of 10 years, body mass is calculated by the formula: **M = 10 kg+ 2 x n**, 10 kg - average body weight of the child 1 year, n — years of age. After 10 years, body mass increases by 4 kg per year.

An example: A child is 2 years old; body weight at 1 year was 10 kg and the body weight at 3 years should be equal to = 10 +2 x3 = 16 kg;

- A child is 13 years old, body weight should be equal to = 10.5 + 10 x 2+ 4 x 3 = 42.5 kg;

Regulation of body weight measurements are: children up 6 months are placed on the special children's electronic scales in the supine position. The kid over 6-7 months old may be on the same scale in a sitting position. Body weight of a child over the age of the year is measured in the balance of a standing position. It is necessary to weigh him in the morning before the first feeding after urination and defecation to determine the exact weight of the child.

The length of the body

The most stable index of physical development is the child'sgrowth. It determines the absolute length of the body and accordingly the increase of body size, development, maturation of organs and systems, formation of the functions at a certain period of time. The growth reflects the features of plastic processes in the human body. Retarded growth can cause different chronic diseases. The

length of the body at birth of the babies born in due term is 50-52 cm on the average. For calculation of the growth parameters the following regularities are used:

during the first quarter (quarter is 3 months) growth increases by 3 centimeters per month, during the second quarter - by 2.5 cm per month, during the third quarter - by 2 cm per month; during the fourth quarter - by 1-1.5 cm per month.

During 12 months the length of the body increases by 1.5 times. The average growth of the one-year- old baby is 75 cm. The average growth of the baby 4- year –old baby is 100 cm.

The average length of the body up to 4 years increases by 8 cm per year (in 4 years is 100 cm). To determine the child's growth under 4 years old the following formula is used:

L (under 4 years old) = 100 - 8 (4-n)

After 4 years length of the body is increased by 6 cm per year.

L (over 4 years old) = 100 + 6 (n-4)

Examples: A boy is 5 months, body length at birth is 56 cm, body length at 5 months is 56 + 3x3 + 2,5x2 = 70 cm;

Body length after infancy increases unevenly at different years of life, and also depends on the gender. More rapid gaining in body length, so-called first push growth (elongation) is observed at 4-5.5 years old for boys and 6-7 years old for girls. Then the growth rate slows down. At 11-14 years old girls and 12-17years old boys with a second push growth and then - a significant slowdown. Body length reaches its maximum at 18-20 years old.

Rules of measuring the length of the body are the following. A one- year- old baby is placed on a horizontal stadiometer covered by diaper. The baby's head is located near the immovable wall of stadiometer, so that the upper edge of the ear canal and the edge of the lower eyelid of a baby are on the same vertical line. The lower limbs are straightened and are pressed against the wooden surface. The movable plate is applied to the feet. Body length is the distance from the immovable wall to movable bar. It is indicated by the numbers along the stadiometer. The length of the older children is measured in a standing position on a standard wooden stadiometer. The child is pressed against the vertical bar back of his head, spine between the shoulder blades, the sacrum and heels.

The **head circumference** (HC) is different in different babies and might depend on many factors. Certain of those factors are completely harmless, while others are pathological. For example, if one of the parents has a head that is pretty small or pretty big compared to the rest of the body, it is quite normal to expect that the baby might develop the same head-to-body proportion. However, if the head is too big it might be a sign of hydrocephalus (a condition in which the liquor in the head cannot drain further and is accumulated in the cranial cavity (around the brain beneath the bones of the skull). This liquor increases the pressure inside the head and affects the development of the brain.

The head of a newborn makes up almost one third of total size compared with the adult proportion approximately 1:7.

Normally, head circumference at birth is larger than chest circumference. The head circumference of a full term newborn is about 2-3cm bigger than the chest circumference which is on average 30.5-33cm. By the age of four months, the head circumference equals the chest circumference, and later the chest circumference is larger than head circumference except the case of malnutrition.

Head circumference is measured by centimeter tape. It is applied on the site behind the maximum protrusion of the occipital protuberance, on supraorbital arches anteriorly.

Indices of head circumference growth are the following:

I half-year – increase this index occurs by 2 cm per month; **II half** - by 0.5 cm per month; **up to 5 years** old- 1 cm per year, **up to 15 years old** - by 0.6 cm per year.

HC (under 5 years old) = 50 - 1 (5-n)

HC (over 5 years old) = 50 + 0,6 (n-6)

Another important parameter used for the assessment of the harmonicity of physical development is the **circumference of the thorax**. This value closely correlates with the functional parameters of respiratory and cardiovascular systems. The following formulae are used to estimate the indices of the chest circumference raises:

a) The chest circumference at the age of 6 months is taken as 45 cm. At the first 6 months of age this parameter raises by 2 cm per month, in the second half of the year - by 0.5 cm per month.

CC (I half) = 45 - 2 (6-n)

CC (II half) = 45 + 0,5 (n - 6)

b) For the children at the age from one to ten years, the following formula may be used: **CC= 63 - 1.5 (10 - n)**

For the children at the age over ten years: **CC= 63 + 3 (n - 10),** where n – age in years, 1.5 or 3 cm – means annual increment value, 63 cm – mean circumference of the thorax at the age of ten year.

Chest circumference within infancy is measured in the supine position, during the second year of life - in the standing position. Measuring tape is placed back at an angle of blades, anteriorly - on nipples.

The criteria for evaluation the **neuropsychological development** are:

Ⅰ. Motor skills; (motor function / movement. Ⅱ.Static; Ⅲ.Reflex activity (1 signal system); Ⅳ.Speech (2 signal system); Ⅴ.The higher nervous activity.

Ⅰ. Motor skills (movement ') - it is a purposeful, manipulative activities of the child. Healthy **newborn** is characterized by the physiological muscle hypertonicity and against the background of this flexion posture. The arms are bent at all joints, and pressed to the chest. Brushes are bent into a fist, thumbs brought to his hand. The legs are also bent at all joints and slightly at the hips, to feet prevails REAR flexion. Even during sleep the muscles are not relaxed. Movements of a newborn are limited, chaotic, irregular (disorderly), trembling (trempling). Tremor and physiological muscle hypertonus disappear gradually after the first month of life.**At 2-3 weeks**there are the following

skills: coordinated movements of the eye muscle, when the child fixes his look on a bright object; rotation of the head followed by a toy indicates the development of the cervical muscles. **At 4 months** of life a manual work hand is developed: the child brings about his upper extremities to his eyes and has a good look at them. The movements are getting purposeful: the kid takes a toy by hands (in the second half of the year he can take a bottle of milk and drink it). At the age of **4-5 months** the coordination of movement of the back muscles is developed, which is manifested by overturning from back to stomach. **At 5-6 months** – a baby can turn over from stomach to back so he turns completely over. At the end of the **first year** of life a child begins to walk, resulted from coordinated aimed movement of all muscles in the proper direction.

Ⅱ. Static is a fixation and holding of specific parts of the body in the desired position. Signs of the static are the following: 1) the head holding - appears on the second or third month of life; 2) the ability to sit - develops in 6-7 months. In addition, at the 6th month of age the baby begins to crawl (born creep, crawl), on the 7th - creeps well. 3) The ability to stand - at 9-10 months. 4) the ability to walk - to the end of the first year of life.

Ⅲ. Reflex activity - it is an adequate response of the child to irritating environmental factors and their own needs. The main reflexes are the following: 1) Dominant food. 2) By the end of the first month a child examines his mother's face carefully. 3) In the second month a smile is formed. 4) In the third month - a joyous movement of the extremities at the sight of his mother appears. 5) joyful exultation on his face (exultant air), smile, movement of hands, feet appear when a mum or dad approach him. 6) The auditory and visual concentration.

Ⅳ. Speech - a child at 4-6 weeks starts hallooing. Pronunciation of the first sounds is called hum (buzz). At 6 months, the child pronounces certain syllables (ba-ba-ba, ma-ma-ma, etc.), without understanding their meaning called babbling baby-talk, (babble, prattle). By the end of the first year of life the baby says 8 - 12 words, the meaning of which he understands (let on, Dad, Mom, etc.). They include sound imitators (am-am - eat, au-au - dog, tick-tock - the clock, etc.). At 2 years old the vocabulary comes up to 300, there are brief suggestions.

Ⅴ. Higher nervous activity. This criterion is developed on the basis of formation of the nervous system, the formation of all the previous criteria, education and child development. It is a sign of maturity and mental ability and human intelligence. The final conclusion on the state of the higher nervous activity can be made at the age of 5-6 years.

Topic 3

Breastfeeding is the normal way to provide infants with the nutrients they need for healthy growth and development. Exclusive breastfeeding is recommended up to 6 months of age, with continued breastfeeding along with appropriate complementary foods up to two years of age or beyond.

Why breastfeeding is important. **1.**Early breast milk is a liquid gold – colostrum is the thick yellow first breast milk that mother make during pregnancy and just after birth. This milk is very rich in nutrients and antibodies to protect a baby. Colostrum changes into mature milk. It is a thinner type of milk than colostrum, but it provides all nutrients and antibodies the baby needs. 2. Breast milk is easier to digest than formula. The proteins in formula are made from cow's milk and it takes time for babies' stomachs to adjust to digesting them. 3. Breastfeeding protects a baby from nemerous illnesses. Stomach viruses, lower respiratory illnesses, ear infections, and meningitis occur less

often in breastfed babies and are less severe. Human milk is protective and immunomodulatory. The anti-infective properties in colostrum and breast milk have both soluble and cellular components. The soluble components include immunoglobulins (SIgA, IgM, IgG), lysozymes, lactoferin, the bifidus factor. 4. Breastfeeding can protect baby from developing allergies. Babies who are fed a formula based on cow's milk or soy tend to have more allergic reactions than breastfed babies. 5. Breastfeeding may protect a child from obesity;may lower baby's risk of SIDS- sudden infant death syndrome;canreduce mother's stress level and her risk of postpartum depression;may reduce mother's risk to develop some types of cancer.

Nutritional composition of breast milk

Fats. The fat content of human milk is 4-4.5gm/100ml, more in hind milk, the fatty acid composition is about 42% saturated, and 57% unsaturated and rich in long chain polyunsaturated fatty acids which are important in brain development and myelination. Human milk contains a non-specific bile salt stimulated lipase and contributes to the infant's fat digestion.In breast milk, there is an enzyme called lipase. Lipase breaks down fat so that the fat is in small globules. This allows better digestion and absorption in baby's stomach.

Carbohydrates. The next big component of breast milk is carbohydrates (37%). The major carbohydrate in the human milk is lactose, about 7gm/lOOml. Lactose is a disaccharide (galactose and glucoselinked together). It is metabolized into glucose and galactose, a constituent of galactolipids needed for the development of CNS.It facilitates calcium and iron absorption and promotes intestinal colonization with Lactobacillus bifidus – lactose is converted to lactic acid by the Lactobacillus (a naturally occurring gut bacterium).In human milk there arealso sucrose, fructose and maltose, which stimulate the growth of L. bifidus and is called bifidus –factor. L. bifidus makes Baby's stomach acidic and maintains an acid milieu in the gastrointestinal tract, inhibiting the growth of pathogenic bacteria, fungi and parasites that are not supposed to be in Baby's stomach.

Proteins. Mature human milk contains protein in the range of 1.3 to 2.07gm/100ml. Mature milk has many different kinds of proteins but the two major players are whey and casein. The whey casein ratio is nearly 80:20. Whey is a smooth, liquid-like protein whereas casein is a coarse protein that tends to curdle. The lactalbumin results in formation of softer gastric curd, reduces gastric emptying time and that's why whey is easier to digest and absorb well in baby's stomach. Because of this, babies fed on breast milk will have runny poops, and get hungry more often. Cow's milk has more casein and less whey. Casein is harder to digest & absorb because it clumps in Baby's stomach. There are many other important proteins: antibodies, lactoferrin, lipase, amylase, lysozyme, and other enzymes (helpful in digestion and create a healthy environment in baby's intestines). Human milk has higher levels of free amino acids, cystine and taurine too.

Vitamin. The vitamin concentrations in human milk are almost always adequate for infants' need. The mineral concentration is lower in human milk than the cow's one and is thus better adapted (digested). Calcium is better absorbed because of high calcium: phosphorus ratio (2:1). The bioavailability of iron is high.

Routine (Mode) of infant feeding. After the normal delivery a newborn is firstly applied to the breast in the delivery room. Within 2-3 months, the child is usually fed in 3 hours, the baby is fed **seven times a day**: at 6 a.m, 9, 12 p.m, 3p.m, 6p,m, 9 p.m and 12 p.m. The period within night and

morning feeding is 6 hours. When the baby suckles as often and long as he wants, it is considered to be the most efficient in the first months of life. This mode of feeding is free. The number of feedings may be from 8 to 12 times during the day. In the third month of life the baby starts to get breast milk in 3.5 hours, i.e. **six times a day**: 6.00, 9.30, 13.00, 16.30,20.00 and 23.30. Night-time - 6.5 hours. Beginning from the age of 5-6 months, when solid foods is to be introduce by the time a child is one year old, feeding interval is changed to 4houers, so the baby should eat **five times daily**: 6.00.10.00,14.00,18.00 and 22.00. Night-time - 8:00.

The calculation of the necessary amount of food eaten by the child.

To calculate approximatly the necessary amount of breast milk for a child at the age of 7-10 days, we can use several ways.

The formula of Tur's: **V=n** x **70** (**80**), where **n** is the age of the child in days.

Example: if the weight of a child at birth is 3200 g, and the child is 4 days,
V = 4x70 = 280 ml, having divided this volume into the number of feedings weobtain 280 ml: 7 = 40 ml - the amount of milk necessary for every single feeding.

The amount of milk necessary for one feeding on the average is **V= 10 x n.**

Example: The child is 2 days, during every single feeding he should drink
10x2 = 20 ml of milk.

The daily amount of food we can calculate by the **volumetric method**.

10 days - 2 months - 1/5 of body's mass ; 2 months – 4 months - 1/6 of body's mass;4–6 months - 1/7 of body's mass; 6–9 months - 1/8 of body's mass. The daily quantity of milk for the first year of life must not be more than 1000 ml

For example: A 5-month-old baby, whose weight of the body is 6650 gm,should eat (drink) 6650 : 7 = 950 ml (necessary amount of food within a day should be 6650 : 7 = 950 ml). The amount of food during every single feeding will be - 950 ml: 5 = 190 ml.

Complementary feeding is the process starting when breastmilk alone (itself) or infant formula alone is no longer sufficient to meet the nutritional requirements of an infant. All infants should keep on receiving breast milk for at least the first year and preferably for the second, otherwise, more nutritious foods should be added by the time the infant reaches six months of age.
Complementary foods isa gradual replacement of milk cooked food.
The need /necessity/ for add feeding is justified as follows: 1. the amount of mother's milk is reduced gradually; 2. human milk provides a child with ingredients only for 5 months; 3. for the proper functioning of the gastrointestinal tract fibers are necessary, which arenot present in breast milk; 4. chewing (mastication) during feeding – an important factor for the proper development of speech apparatus;

Guiding principles for appropriate complementary feeding: 1. Go on frequent, breastfeeding or on demand until a baby two years old or more; **2.** Complementary feeding can be started only when a child is healthy**; 3.** first cooked food is given before breast-feeding, then the baby is fed with breast milk. **4.** start with small portions - 15-20 ml and increase gradually: *In the days of feeding the input increases to 20-30 ml*. **5.** One breast is replaced by cooked food for

2 weeks**; 6.** Only one feeding in the diet is replaced by cooked food, the other 4 times the baby gets breast milk**; 7.** cooked food should be homogenous.

First complementary food (Solid foods) - is given at 6 months old; first solid food is given in the form of vegetable puree. At first, potato is used, in a week other vegetables are added - carrots, zucchini, cabbage.At the age of 6 months oil can be added, at 6-7 months – butter. When a baby is 7 months oldbread is given in the amount 5 g to 10 g.

Second complementary food is given at 7 months oldand introduced in the form of milk porridge. The most rational are the following cereals: rice, buckwheat (born buckwheat), corn (born maize, corn).At the age of7 months the range of vegetable foods is expanded turning into dinner, i.e. soup: the child receives a low-fat meat bouillon (60-70 ml), minced meat (chicken, pork, beef). Begin with small portions - 5 g, single dose minced meat gradually increases to 20-30 gr. The total amount of food is up to 200 ml - 120 ml vegetable puree, 50 ml of meat bouillon and 30 g of meat. At the age of 7-8 months the**third III Complementary food**is introduced - low-fat cottage cheese (30 g and 50 g by the end of 1 year), and yogurt.At the age of 10 months of life a baby can be given a second time on the day of porridge, but from the other types of cereals.

Example of menu (6, 5 months):

6.00	10.00	14.00	18.00	22.00
breast milk — 200 ml	vegetable puree -200 ml	breast milk — 200 ml	milk rice porridge -200 ml	breast milk -200 ml

Fruit, berry and vegetable juices are given to a child at the age of 5-6 months. Usually one starts with apple juice, made from non-acidic yellow-green sorts of apples. Juice is introduced very carefully, slowly with increasing volume. Fruit puree. The first fruit is apple puree, then a variety (assortment) is expanded, using the combined fruit and fruit and berry puree of different kinds of fruit and berries.

Unfortunately, there are some limitations as for the breastfeeding. The main indication for mixed and artificial feeding is mother’s hypogalactition (agalactia). Other indications for artificial feeding are the following **contraindications to breastfeeding**.

From **Mother's side there are:**1) malignant neoplasms and chemotherapy; 2) acute mental illness;3)dangerous infections (tetanus, typhoid); 4) the open form of tuberculosis;
5) syphilis infection affecting mother after the 6-7th month of pregnancy; 6) chronic diseases at decompensation stage; 7) viral hepatitis, salmonella, dysentery, however expressed sterilized milk can be used; 8) measles, chicken pox. In this case it is possible to fed a child with breast milk after the introduction of gamma globulin; 9) in case of SARS, bronchitis, pneumonia, you need stop breastfeeding for a while mother’s temperature and toxicity are reduced.

Child's side. During the neonatal period, there are some serious pathological conditions, which are contraindications for breastfeeding, but breast milk is not prohibited. In these cases, children are fed with expressed milk or milk donors**:** severe disorders of cerebral circulation, hemolytic disease of a newborn in the first 7-10 days of life, profound prematurity with the lack
of swallowing and sucking ref-lexes**;** severe respiratory distress. These children are fed with

expressed breast milk, and in case of hemolytic disease - milk donors. There are some congenital disorders: phenylketonuria, galactosemia, cystic fibrosis

Artificial feeding is a type of feeding, when a child doesn't get breast milk in the first half of the year, but it is fed only with formula milk. If the child the first months of life receives, at least 20% of the daily diet of breast milk, this type is also called artificial feeding.

In case of artificial feeding a baby instead of breast milk gets specially prepared mixes, which are divided into 2 groups: 1.adapted formulas(Nutrilon", "Nan", "Alprem" (Switzerland), "Prehipp" and "hippie-1" (Austria),Tutelli ("Valio"), Bona" (Finland),"Heinz" (England),"Humana"); 2. Non-adapted -made of fresh or dried milk of animals without any special processing. Infant formulas are also divided on: 1. fresh (пресные) mixture and acidophilus (sour milk кисло-молочные) mixture; 2. standard is adapted to preparations based on cow's milk, and specialized which are made for special categories of children: premature babies, children - allergies, children born with low birth weight, children suffering from food intolerance, vomiting, constipation etc.

Topic 4

The World Health Organization defines normal term for delivery ranging from 37 weeks and 42 weeks. At birth, a baby is classified as one of the following: Premature [preterm] (less than 37 weeks gestation), Full term (37 to 42 weeks gestation), Post term (born after 42 weeks gestation). A Newborn child is a child aged from birth to 28 days.

Primary treatment of a newborn is carried out immediately after birth in the delivery room. **Stage 1**–To remove mucus from the nasal passages and the mouth of the fetus at birth using electric pumps to prevent aspiration. Put the child on changing table on a warm dry sterile diaper under a radiant heat. **Stage 2** - 1 drop of 30% solution of sulfacetamide should be dropped in the eyes to prevent gonoblenorei. **Stage 3**- Two Kocher clamps are applied to the umbilical cord - one at a distance of 10-15 cm from the umbilical ring, the other one - to 2 cm outside of it. Plot of umbilical cord located between the clamps, is treated with 5% iodine solution and 96% alcohol and intersected with sterile scissors. The cord and umbilical ring are treated with 96% alcohol. At a distance of 0.3-0.5 cm from the umbilical ring clamp Kocher's is applied, then it is replaced by bracket of Rogovin in 1-2 min. Umbilical cord is intersected with sterile scissors above 1.5-2 cm brackets. Slice of the umbilical cord and its remainder is treated with a 5% solution of potassium permanganate, gauze bandage is applied on the umbilical cord remainder or leave it open. **Stage 4** - A baby's skin is wiped with a swab moistened in sterile oil to prevent of piodermiya. After primary treatment of the newborn following steps are carried out.

Assessmentof a newborn includes: **1. Apgar scoring** is a quick test performed on 1 and 5 minutes after birth. The baby is checked for heart and respiratory rates, muscle tone, reflexes, and color. Each area can have a score of zero, one, or two, with ten points as the maximum. A total score of ten means a baby is in the best possible condition. Apgar scores of three or less often mean a baby needs immediate attention and care.

Sign	**Score = 0**	**Score = 1**	**Score = 2**
Heart Rate	Absent	Below 100 per minute	Above 100 per minute

Respiratory Effort	Absent	Weak, irregular, or gasping	Good, crying
Muscle Tone	Flaccid	Some flexion of arms and legs	Well flexed, or active movements of extremities
Reflex/Irritability	No response	Grimace or weak cry	Good cry
Color	Blue all over (cyanotic), or pale	Body is pink, hands and feet are blue	Pink all over

2. Birth weight and measurements: The average weight for term babies (born between 37 and 41 weeks of gestation) is about 3.2 kg. Other measurements are also taken of each baby: head circumference, abdominal circumference, length. **3. Physical examination** includes the assessment of the following: vital signs (temperature, pulse - normal 120 to 160 beats per minute, breathing rate - normal 30 to 60 breaths per minute, general appearance - physical activity, tone, posture, and level of consciousness, skin, head, neck and clavicles, fontanels (the open "soft spots" between the bones of the baby's skull), face, mouth - palate, tongue, throat, genitals and anus - for open passage of urine and stool, arms and legs - movement and development. **4. Gestational assessment.** An examination called The Dubowitz/Ballard Examination for Gestational Age is often used. The new scale Ballard uses six morphological and 6 neuromuscular signs, by whichgestational agecan be estimated, counting the number of points.

Physical maturity. Assessment areas include the following signs: 1. skin textures (sticky, smooth, peeling); 2. lanugo (the soft downy hair on a baby's body) - is absent in immature babies, then appears with maturity, and then disappears again with postmaturity; 3. plantar creases - these creases on the soles of the feet range from their absense to covering the entire foot, depending on the maturity. 4. breast - the thickness and size of breast tissue and areola (the darkened ring around each nipple) are assessed. 5. eyes and ears - eyes fused or open and amount of cartilage and stiffness of the ear tissue. 6. malegenitals - presence of testes and appearance of scrotum, from smooth to wrinkled, female genitals - appearance and size of the clitoris and the labia.

Neuromuscular maturity. Six stages to evaluate of the baby's neuromuscular system are performed. These include: 1. posture - how does the baby hold his/her arms and legs. 2. square window - how far the baby's hands can be flexed toward the wrist. 3. arm recoil - how far the baby's arms "spring back" to a flexed position. 4. popliteal angle - how far the baby's knees extend. 5. scarf sign - how far the elbows can be moved across the baby's chest. 6. heel to ear - how close the baby's feet can be moved to the ears. Scores from neuromuscular and physical domains are added to obtain total score. When the physical assessment score and the neuromuscular score are added together, the gestational age can be estimated.

5. Functional maturity. A newborn is considered to be **functionally mature** when his organsfunction properly to maintain the livelihoods of the body in the extrauterine environment. In this case, signs of functional maturity are as follows: 1.Sufficient spontaneous motor activity (periodic limb movements, active response to loud sound, the

glare, hunger); 2. Physiological flexor hypertonus; 3. Expressed by innate unconditioned reflexes; 4. Active sucking; 5. A loud cry; 6. Adequate thermoregulation (keeping a constant body's temperature when adequate ambient temperature).

PHYSICAL CHARACTERISTICS of a mature newborn.

Weight and Length. The average baby weighs between 2500 gr and 4500gr. The average total body length is 46–54 cm.

Head. Shape: A newborn's head is very large in proportion to the body, and the cranium is enormous relative to his or her face. While the adult human skull is about 1/8 of the total body length, the newborn's is about 1/4. The head circumference is 33 – 35 cm. **Soft spots:** An infant has two obvious soft spots or fontanelles. One is on the top of the head and the other is near the back of the head. The larger one, in front of the head closes by 6 – 18 months. The smaller one is closed. **Hair**: Infants may be born with full heads of hair; others, particularly white infants, may have very fine hair or may even be bald.

Skin The skin is thin and dry. Immediately after birth, a newborn's skin is often grayish to dusky blue in color. As soon as the newborn begins to breathe, usually within a minute or two, the skin's color reaches its normal tone.

Lanugo: baby's body is covered with fine downy hair. This hair is most noticeable on the back, shoulders, and ear lobes. It will fall out in time.

Vernix: Newborns are wet, covered by streaks of blood, and coated with a white, cheese-like substance known as vernixcaseosa , which is known to act as an antibacterial barrier. It is not necessary to scrub it off. **Fingernails** grow to the edge of the fingers. **Newborns' digestive tracts** are filled with a greenish-black, sticky material called meconium. This material is excreted in the first few days. **Heart beats**: Usually the heart rate is 140 – 160 beats per minute. **Respiratory rate**: It is faster than adults, usually 40 – 60 breaths / minute. **Genital**. In full-term boys' testicles descended into the scrotum, labia majora cover the small ones in girls.

Transient states or **temporary conditions.** The states, which characterize the processes of adaptation to extrauterine life, are called transient states.

1. In the first days of life (4-5 days), we have so-called **physiological weight loss**. Infants may lose up to five - ten percent of their birth weight during the first week of life, mainly due to loss of extra fluid.

2. **Hormonal crisis** is caused by hormones a baby gets from the mother during pregnancy: **1) swollen breasts** are normal for both boys and girls during the first days after birth; **2) Milia.** These are tiny white spots often seen on the nose and chin. They are caused by obstruction of oil or sebaceous glands**; 3) Genitals-Swelling & Vaginal Discharge:** The genitals of both boys and girls are usually large and swollen due to hormones passing from the mother through the placenta. Male infants have an unusually large scrotum. Girls may also have a white, mucoid, and sometimes blood-tinged vaginal discharge.

3. **Transitory changes of skin: 1)** simple erythema – skin response manifested as reddening which appears after primary lubrication removal and first bath. It is connected with the capillaries widening, disappears to the end of the first week of life.

2) Toxic erythema is erythematous, slightly tight spots, often with dark grayish papules or vesicles in the center containing a clear serous fluid and often located in groups on the extensor surfaces of the limbs around the joints, on the scalp, chest, buttocks, sometimes on the stomach and face.

4. **Transient hyperbilirubinemia**. The basis origin of physiological jaundice is particularly bilirubin metabolism in the newborn. Clinically transient hyperbilirubinemia is seen as icteric skin for 2-3 days of life that disappears by 7-10 day life, among premature children it disappears much later. General condition (state) of such infants has no changes, gepatolienalny syndrome is absent. Umbilical bilirubin level makes 17,1 – 51 mkmol/l, bililirubin maximal level doesn't exceed 204 mkmol/l in mature and 170 mkmol/l in immature newborns, indirect fraction of bilirubin becomes predominant, the urine is light-yellow, feces are yellow.

5. **Transitory peculiarities of kidneys functioning** caused by the increased catabolism, lead to increased purine metabolism with the formation of uric acid, which is deposited as crystals in the renal tubules, resulting in yellow-brownurine. Oliguria in the first 3 days of life and proteinuria (albuminuria) also are detected.

6. **Transient impairment of thermoregulation**: Transient hypothermia - in the first 30 minutes temperature is reduced by 0,1-0,3 ° C per minute and reaches about 35,5-35,8 ° C; Transient hyperthermia - occurs on 3-5 days of age, the body temperature can rise up to 38,5-39,5 ° C and above.

7. **Transitory dysbacteriosis.** Defecation disorders are associated with the transition to the new baby type enteral nutrition. In the first 1 - 2 days for all infants produce original feces (meconium) - thick viscous mass of dark green color. For 3 - 4 days of life transition stool becomes nonuniform in consistency and color (lumpy, mucus, green and yellow). By the end of the first week feces are usually in yellow slurry.

Tests Topic 2

1. The main indices of physical development
   A. Body weight, Body length (growth), Head circumference
   B. body weight, respiratory rate, vital capacity,
   C. body weight, head circumference, heart rate, body length
2. Monthly weight gain in the first half
   A. by 400 g per month
   B. by 600 g per month
   C. by 800 g per month
   D. by 200 g per month
3. The monthly increase in body length in the 1st quarter
   A. by 1 centimeter in quarter
   B. by 2 centimeters in quarter
   C. by 3 centimeters in quarter
   D. by 4 centimeters in quarter

4. The annual increase in body length after 4 years old

A. by 6 centimeter
B. by 8 centimeters
C. by 3 centimeters
D. by 4 centimeters

Task. An 8-months-old girl, was born with 54 cm body length, 35 cm head circumference. Calculate the appropriate normative anthropometric indices.

Topic 3

Tests

1. Which feeding must be the first in the child's ration?

A. Kefir.
B. Milk porridge.
C. Vegetable puree.
D. Clear soup.

2. What are the differences between proteins of breast milk and of cow milk?

A. There are more globulins in breast milk.
B. The protein quantity is the same for the cow's and breast milk.
C. There are more albumins in breast milk.
D. There is more casein in breast milk.

3. A 3- months-old baby is breast-fed. His body weight is 5400g. Define the daily food volume for this infant:

A. 900ml.
B. 950ml.
C. 930ml.
D. 850ml.
E. 870ml.

4.A 4- months-old baby is being artificially fed with adapted mixture "Humana". What is the correlation between serous proteins and casein in adapted substitutes of breast milk?

A.80:20
B.70:30
C.60:40

D.40:60

E.30:70

Task № 1.An infant was born deeply premature; of the 4th pregnancy, mother had gestoxicosis of its 2 half. A 37-years-old woman caught influenza at the 12th week of gestation.She had precipitated labor. After new-born infant's inspection the neonatologist forbade to put it to the breast. Point out the absolute contra-indication for this child to be put to the breast:

A.Deep dysmaturity.

B.Gestosis of the 2 half of gestation.

C.Mother's age is 37 years.

D.Influenza during gestation.

E. precipitated labor.

Topic 4 Tests

1. When is baby checked by Apgar score?

A. on 1_{st} minute and 5 minutes after birth
B. on 1_{st} minute and 10 minutes after birth
C. on 5 minute and 10 minutes after birth

2. What signs doesn't involved into Apgar?

A. heart and respiratory rates
B. Body weight; Body length
C. muscle tone
D. reflexes, and color

3. Baby is classified as Premature

A. less than 37 weeks of gestation
B. less than 38 weeks of gestation
C. less than 39 weeks of gestation

4. What is bililirubin maximal level in mature in case of transient hyperbilirubinemia?

A. it doesn't exceed 170 mkmol/l
B. it doesn't exceed 204 mkmol/l
C. it doesn't exceed 120 mkmol/l

Task. Examination of the newborn infant by Apgar scale has found: RR – 80, cyanosis, small retraction during respiration; weak cry; some flexion of arms and legs. What is the score by Apgar scores?

1. 5
2. 3
3. 4
4. 7

List of references

Pediatrics [Text] : textbook / O. Tiazhka [et al.] ; ed. O. Tiazhka ; Ministry of Public Health of Ukraine, National O. O. Bohomolets Medical University. - Vinnytsia : Nova knyha, 2011.

1. Nelson Textbook of Pediatrics, 2-Volume Set, 20th Edition
2. TEXTBOOK OF PROPEDEVTIC PEDIATRICS For students of II – III years**.** Kyiv 2006**.** Autors: O. Vinnitzka, T.Lutaj, A.Antoshkina, Ju. Piatnitzki, M. Vasiukova, N. Gorobetz, L.Martinova, T. Mellina, L.Slipachuk, O.Stroj, P.Tovmash
3. Pediatrics. Guidance aid for students of V year of study. Edited by Professor O. Tiazhka

MINISTRY OF PUBLIC HEALTH OF UKRAINE
O.BOHOMOLETSNATIONALMEDICALUNIVERSITY

PEDIATRIC DEPARTMENT N5

"Approved"
on the methodical conference
Department of Pediatrics № 5
protocol №2 from 07.09.2016

GUIDELINES
FOR STUDENTS

Academic discipline	Pediatrics
Study subject	Rickets. The most common children respiratory diseases. The most common children gastro-intestinal diseases
Course	4
Faculty	Dental

Kyiv-2016

Relevance of the topic.

TOPIC 1. Rickets is a disease of growing bone that is unique to children and adolescents; it is a disorder caused by a deficiency of vitamin D, calcium, or phosphate and therefore by a failure of osteoid to calcify in a growing person. Failure of osteoid to calcify in adults is called osteomalacia.Spasmophilia and hypervitaminosis D are severe diseases that strike young children. If not to treat these conditions properly lethal outcome is possiblein the absence of adequate medical interventions.**:**

TOPIC 2. Anatomical and physiological features of a children respiratory system cause specific pulmonary diseases that rank the first placeamong childrenmorbidity. Pneumonia results in high morbidity and mortality. According to WHO (2001) one-third of children who do not survive up to 5 years die of acute respiratory infections (ARI) and associated pneumonia.

TOPIC 3. Children gastro-intestinal diseases occur because of their high prevalence, features of the clinical course, a high risk of early manifestation and disability are a serious medical and social problem.

Questions for self– checking

1. Etiological factors of various forms of bronchitis
2. The etiology of pneumonia depending on the age of the child.
3. Classification of bronchitis and pneumonia.
4. The major clinical syndromes of bronchitis and pneumonia in children.
5. Etiotropic and pathogenetic therapy of respiratory diseases.
6. Pneumonia complications and their treatment
7. The main etiological factors leading to the development of rickets in children.
8. The properties of vitamin D and the pathogenesis of rickets.
9. Classification of rickets and clinical characteristics of mild, moderate, severe stages of rickets.
10. Treatment, rehabilitation and prevention of rickets.
11. Clinical classification of hypervitaminosis D in children.
12. The treatment of hypervitaminosis D in children.
13. The main clinical manifestations of spasmophilia.
14. Emergency care given incase of spasmophilia.
15. Clinical and diagnostic manifestations of chronic gastritis, gastroduodenitis.
16. Modern views on the etiology and pathogenesis of gastric and duodenal ulcer disease.
17. Clinical and laboratory- instrumental signs of ulcer disease.
18. Differential diagnosis of chronic gastritis, gastroduodenitis and ulcer disease.
19. Treatment of children with chronic gastritis.
20. Etiopathogenetic versions fordiseases of the biliary tract.
21. Clinical- laboratory and instrumental diagnostic criteria of cholecystitis.
22. Differential diagnosis of cholecystitis and dyskinesia.
23. Treatment features of hypertensive form of biliary duct dyskinesia.
24. Treatment features of hypotensive form of biliary duct dyskinesia.

Topic 1. Rickets, spasmophilia, hypervitaminosis D

Rickets can be caused by 1) Dietary deficiency of vitamin D, calcium or phosphorus, **2)** Little walking on the fresh air, because Vitamin D-3 (cholecalciferol) is formed in the skin from a derivative of cholesterol under the stimulus of ultraviolet-B light. 3) Prematurity **4)** Deficientmetabolites of vitaminD

Vitamin D-deficiency rickets classification.

I. Period of disease – initial, manifestations, reconvalescence,permanent changes

II. Severity of the disease - mild; moderate (Intermediate), severe

III. Course of the disease- acute, subacute, relapsing (recurrent).

The initial period - The first symptoms usually appear on the 2-3rd month of life. The child's behavior changes: in the form of anxiety, fearfulness, irritability, tremors under external stimuli (loud noise, a sudden flash of light), sleep becomes disturbing. Sweating is growing, especially in the scalp and face. Sweat has a sour odor and irritates the skin, causing itching. The child rubs his head on the pillow, resulting in the appearance of patches of alopecia on the back of his head. We can see appearance of the soft skull bones, delayed closing of the soft spot at the top of baby's head (the anterior fontanelle), muscle.

The **manifestation period** is often at the end of the first six months of life and is characterized by more significant disorders of the nervous system and musculoskeletal system.

The processes of osteomalacia predominate in the acute period of disease. There are craniotabes (areas of thinning and softening of bones of the skull), compliance and deformity of the chest with a depression in the lower third of the sternum ("breast shoemaker) or protrusion ("chicken" or "carinate" chest), O--shaped (genu varum), rarely X-shaped(genu valgum)) bending of long bones, narrowed rachitic flat pelvis, Harrison groove (the weakened ribs pulled by muscles produce flaring over the diaphragm, which is known as Harrison groove)

The processes of osteoid hyperplasia predominate in the subacute stage of illness.Hyperplasia is manifested by the following symptoms: the formation of hypertrophic frontal and parietal protuberances, changes in the skull resulting in a distinctive "square headed" appearance; in the chest, knobby deformities results in the so-called rachitic rosary along the costochondral junctions; widening of wrist to form the so-called " rachitic **brasletok**, it is due to metaphysial cartilage hyperplasia; thickening of the inter-phalangeal joints of the fingers to form the so-called "strings of pearls."These deformities persist into adult life if they are not treated.

The following tests may help diagnose rickets: 1) The serum calciumconcentration reduced(normal 2,5-2,7 mmol / l); 2) The concentration of serum phosphorus reduced (normal 1.3-2.3 mmol / l); 3) Serum alkaline phosphatase may be high; 4)Arterial blood gases may reveal metabolic acidosis. 5) Determination of Ca, P in Urinalysis per day. Qualitative reaction to determineamount of calcium in the urine which is Sulkovychtest. In healthy children, the content of Ca in urine is - "+ +", while the manifestation period of rickets - negative Sulkovych test is typical. 6) Bone x-ray may show loss of calcium in bones or changes in the shape or structure of the bones. **Bone x-rays for diagnostic purposes is not currently being done.**

Prevention of rickets

Antenatal prevention

1. Antenatal non-specific prevention of rickets includes compliance with regime of the pregnant woman – to sleep a lot during day and at night, walking on the fresh air, balanced diet , physical exercises,

2. Antenatal specific prevention - All pregnant women in the period of 28-32-weeks gestation should be prescribe of vitamin D daily for 500 ME per day for 6-8 weeks, excluding the summer months. Contraindications: maternal age over 30 years, as well as a maternal illness.

Postnatal prophylaxis of rickets include

1. Postnatal non-specific prevention - breast feeding, keeping the regime of the day (being on fresh air, air bath in summer); tempering,massage and gymnastics for 30 – 40 min a day, walking on the fresh air.

2. Postnatal specific prevention – 1) 2 courses of UVI (autumn – winter) for 10 – 15 sessions (after UVI - vitamin D is not prescribed for 2 months). 2) Vitamin D is prescribed to full-term children from 4 weeks to 1 year (400 – 500 IU) and to pre-term children from 10 -14 days to 1,5 years (500 -1000 IU).

Treatment of rickets should be complex, long-term and focused on the elimination of its cause. Great importance is the non-specific treatment, which includes the rationalfeeding, strict regime, regular walking on open air (with sufficient inso-lation), medical gymnastics and massage, tempering, treatment of opportunistic diseases.Specific treatment of rickets includes the administration of vitamin D, calcium and phosphorus. Vitamin D can be given in supplement from 1000 to 4,000 IU per day for six to 12 weeks, depending on the severity of the disease. The treatment is controlled by Sulkovichtests. The study is conducted before the prescription of therapeutic doses of vitamin D, and then every 7-10 days of treatment. If the result Sulkovich test is as "+++" or "++++" a therapeutic dose is canceled. In such cases, the *transition* is carried out taking a prophylactic dose of vitamin D.

Hypervitaminosis D. Hypervitaminosis D (D-vitamin's intoxication) is a disease caused by a direct toxic effect of the drug of vitamin D on the cell membrane, and its high content in the blood and urine which causes deposition of calcium salts in the walls of blood vessels, especially the kidneys and the heart. It often leads to chronic pyelonephritis, chronic renal failure, even disability.

Cause of the condition: An overdose of vitamin D; The combination of vitamin D and UV; idiopathic hypercalcemia.

Intoxication with vitamin D is manifested by the following symptoms: poor appetite, thirsty, vomiting, constipation (possible unstable and loose stools), irritability, poor sleep, fatigue, artragia, dry, grayish-pale skin, muscular hypotonia, a gradual increase in malnutrition, premature closure of the largefontanelle, weight loss and dehydration, changes in the cardiovascular and urinary systems.

Laboratory criteria of hypervitaminosisD :hypercalcemiya,hypophosphatemia; positive Sulkovich test (++++) is detected; X-rays of bones demonstrate increase of the number of cores ossification for the childage, increase calcium deposits in the bones and areas of growth.

Treatment - 1. Diet and arrest of vitamin D, UV, drugs Ca intake;2. Enterosorbents; 3. Intravenous fluids: glucose, albumin, reopoliglyukin, forced diuresis.4. Vitamin A, E, C, B1, B6. Retinol is the antagonist of ergocalciferol. 5. Short-term hormone therapy; 6. Symptomatic therapy.

Spazmophilia(tetany) is a disease pathogenetically associated with rickets, characterized by a tendency of a child during the first 6-18 months to convulsions and spastic conditions, caused by abnormal disturbance of calcium-phosphorus metabolism, which takes place in rickets.

Clinical picture. Hidden (latent) and the apparent /manifested/spazmophilia can be defined. Latent /Hidden/ spazmophilia is characterized by the appearance in the child sleep anxiety, motor restlessness, undue shyness, tachycardia, enhanced sweating, hyperesthesia and various disorders of the gastrointestinal tract. On examination of a child a number of characteristic features as symptoms Chvostek, Trousseau, Lust and Erbisrevealed. Manifested forms are laryngospasm (glottidospasm, laryngismus), carpopedal spasm, eclampsia. Laryngospasm - a spasm of the glottis, occurs suddenly when a child is crying or is frightened. It manifests *sonorous or hoarse* breath while crying and screaming and breathing arrest for a few seconds; frightened facial expression, cyanosis, cold sweat,sometimes loss of consciousness are observed.The attack ends with a deep sonorous breath, breathing gradually becomes normal and the baby falls asleep. Carpopedal spasm is a muscle spasm of hands and feet, taking the typical position (wrist position is "hands obstetrician," stop - in sharp plantar flexion). Often there is a spasm of the circular muscles of the mouth, causing the lips take the form of "fish mouth".Eclampsiaisclonic-tonic convulsions occurring with loss of consciousness. They occur after short-term tonic convulsions. In mild cases, the attack is manifested by sudden blanching of the face, stupor, twitching of facial muscles.Severe attack also begins with twitching of facial muscles, further seizures spread to the neck, limbs, covering all the major muscle groups, including the respiratory muscles. Breathing becomes intermittent, ***sobbing;*** cyanosis occurs

Emergency care of spasmophilia.

Withlaryngospasm you should undress the child, provide access to fresh air and create dominant focus of excitation in the brain by stimulatingthe nasal mucosa (blow and tickle the nose, give ammonia smell), the skin (prick, clapping and dousing cold water on the face), the vestibular apparatus ("shaking" of the child), change body position.

General clonic-tonic seizures require the following measures: 1) introduction of anticonvulsant drugs: chloral hydrate in enema. 0,5% solution of seduxen 0,5 mg/kg IM or IV, 25% MgSO4 0,2 mg/kg IM, Na oxibutirate 50 – 120 mg/kgIM or IV.

2) Pathogenetic treatment is prescribing calcium to increase the content of its ionized levels - after the emergency determination of calcium in the blood 10% calcium chloride or gluconate is injectedintravenously. Antirahitic treatment begins in 2 - 3 days after administration of calcium preparations.

TOPIC 2: The most common children respiratory diseases.

Acute bronchitis is anacute inflammation of the bronchial mucosa with no signs of damage to lung tissue.

Etiology. Infectious agents are represented by viral pathogens (influenza, para-influenza, adenovirus, respiratory syncytial, ECHO-and Coxackie virus) and bacterial pathogens (Chlamidia pneumonia, Streptococcus pneumoniae, Haemophilusinfluenzae, and Moraxella catarrhalis).

Pathogenesis. Viruses multiply and damage the epithelium, causing dystrophy, impaired relations between the individual cells, their rejection. Serous exudate with macrophages, white blood cells and epithelial cells appear in the lumen of the bronchi. Therefore, the barrier properties of the bronchi walls are oppressed so conditions for the development of the inflammatory process of bacterial etiology become favorable.

Clinical presentation. The symptoms of acute bronchitis usually begin on the 3 to 4 days after an upper respiratory infection, such as a cold or influenza (flu).

The symptoms of bronchitis may be combined into two groups. The first group consists of infectious toxicitysigns: fever,tiredness, deterioration of general well-being of the child, headache, dyspepsia (in infants). The second group consists of organ-specific symptoms.

1. The symptoms of acute bronchitis are preceded by the signs of acute respiratory viral infection: fever with chills, myalgia, headache, weakness, a tiredness and rhinitis, pharyngitis (coughing, soreness in the throat), sometimes laryngitis (hoarseness), tracheitis (soreness and pain behind the breastbone, dry painful cough) and conjunctivitis (with adenoviral infection).
2. The organ specific symptoms include cough, which is the main symptom of acute bronchitis. At first it may be dry (it does not produce any mucus) and after a few days may bring up mucus from the lungs (productive cough); a sensation of tightness, burning or dull pain in the chest under the breastbone.
3. On examination: signs of intoxication and respiratory failure are absent.
4. On palpation and percussion - changes in the lungs are usually absent
5. Auscultation: hard breathing is detected, rales are usually listened on both sides and in different regions of the lungs, rales are changed to coughing. At the beginning of the disease rales are dry and then they become wet.
6. Peripheral blood analysis shows no changes or represents a viral or bacterial infection.
7. On the chest X-ray there isan increasedlung pattern, a shadow root of the lung is more intense.

Acute obstructive bronchitis

Etiology – it is caused by respiratory syncytial virus, parainfluenza virus type 3 viruses, adeno-and rhinoviruses, herpes, chlamydia, mycoplasma

Pathogenesis. Inflammatory changes affect the bronchi of small caliber(size). Edema, bronchospasm and hypersecretioncause airway obstruction and impaired alveolar ventilation, resulting in respiratory failure. In obstructive bronchitis edema and swelling of the mucous membrane predominate.

Acute obstructive bronchitis is typical for children of the first three years of life. The **clinical picture** is characterized by acute onset and consists of syndrome of infectious toxicosis and signs of bronchial obstruction:

1. Dry, paroxysmal cough, by the end of a week becomes wet
2. Prolonged whistling breath, which can be heard at a distance
3. On examination: expiratory dyspnea, inflated chest, supporting muscles involved in breathing (intercostal retractions ; participation supporting in the breathing process).
4. On percussion there is vesiculotympanitic {bandbox, wooden} resonance (sound of empty box).
5. On auscultation - prolonged exhalation, dry wheezing throughout both lungs, and later moist rales(medium and large bubbling) are detected. Wheezing is a high-pitched whistling sound during breathing. It occurs when air flows through the narrowed breathing tubes.
6. Peripheral blood analysis may detect the moderate leukocytosis, ESR is usually normal.
7. Chest X-ray shows that there are no signs of pneumonia. If it is, then the X-ray determines clear or lucent lung fields,low position of the cupula of diaphragm,narrowing the median shade.

Pneumonia is an acute infectious inflammation of the lungs (alveoli), accompanied by respiratory disorders and confirmed by changes in chest X-ray examination.

Causes.The most common cause of bacterial pneumonia are: Streptococcus pneumonia, Haemophilusinfluenzae type b (Hib), Escherichia coli (Birth to 20 days), Chlamydia pneumonia, Mycoplasma pneumonia; the most common viral cause areAdenovirus, Influenza virus, Parainfluenza virus 1, 2, 3, Respiratory syncytial virusres, Cytomegalovirus,Herpes simplex virus.

Pathogenesis. Pneumonia is characterized by inflammation of the alveoli and terminal airspaces in response to invasion by an infectious agent introduced into the lungs through hematogenous spread or inhalation. The inflammatory cascade triggers the leakage of plasma and the loss of surfactant, resulting in air loss and induration.

Classification of acute pneumonia:

1. **The form** - the focal, segmental, lobar, interstitial;
2. **The course** of pneumonia – acute (2-6 weeks), protracted;
3. **The severity -** uncomplicated; with the complications: a) pulmonary: pleurisy, pulmonary decomposition, pneumathorax; b) extrapulmonary: infectious - toxic shock syndrome, the cardiovascular collapse and others.

Clinical picture

1. Catarrhal signs of the upper respiratory tract. Upper respiratory symptoms of a cold, such as a runny nose, usually precede the symptoms of pneumonia.After having symptoms of a mild upper respiratory tract infection, such as a runny nose and mild cough, children who develop pneumonia may have a sudden worsening and develop other symptoms.
2. Intoxication syndrome - headache, weakness, .sleep disturbance, loss of appetite, pale skin.
3. Hyperthermia (over 38) for 3-5 days.

4. The cough has usually dry obtrusive character at the onset of the disease, it becomes productive, softer, productive and deep on the 5-7 day
5. Signs of respiratory distress – dyspnea, increased respiratory rate (tachypnea), cyanosis, swelling of the nose, grunting, nasal flaring and retractions at subcostal, intercostal, or suprasternal sites (spaces).
6. Palpation - increased the voice vibration
7. Percussion - shortening of percussion tones over the affected section of the lung. Percussion may reveal important data. Occasionally, a child presents with a high fever and cough but without auscultation findings suggestive of pneumonia. In such cases, percussion may help identify a thickening area.
8. Auscultation: the presence of crackles or rales which are moist, sonorous, and *small vesicles*, asymmetry of breath sounds in infants, such as focal wheezing or decreased breath sounds in one lung field or local bronchial breathing.
9. The clinical picture of interstitial pneumonia caused by viruses consists of intoxication syndrome, severe respiratory and cardiovascular failure.
10. Blood test shows increased amountsof leukocytes with a shift to the left of the formula (inflammation), toxic granulation of neutrophils, ESR acceleration.
11. X-ray picture –increased pulmonary patterns, enlarged lung roots, focal inflammatory infiltration shadow.

Treatment

- Milk and vegetable diet, a diet enriched with vitamins.Water schedule /lavishdrinking (tea, broth hips, alkaline mineral water, hot milk).

- Etiotropic therapy: 1) In case of bronchitis, antiviral therapy is prescribed, antibiotics are used by indications. Ativiraldrugs:Laferon, viferon; groprinozin, amixin, rimantadine, arbidol; 2) Antibiotics are used into or intramuscularly, as cephalosporins (cefazolin,), macrolides (rovamycin, roxithromycin (rylide), clarytromycin (clacyd), acytromicin), semisynthetic penicillins (amoxicillin). In case of serious and complicated course of pneumonia the amino acids are prescribed (amicacin, tobramycin). Antibiotics are usually prescribed for 7-14 and more days.

- Antipyretics in age dosage in case of body temperature elevation above 38,5-39,0 ° C. The drug of choice isparacetamol.

- Antitussives (sinekod, libexin) are used only in case of dry intrusive cough. Hypersecretionof mucus is a contraindication to antitussives.

- Expectorants (drugs of thermopsis, essential oils, terpinhydrate, sodium and potassium iodide) and mucolytic drugs (acetylcysteine,bromhexine, ambroxol)

- bronchodilators are used if there are clinical signs of bronchial obstruction in the form of inhalations inside - ß-adrenomimetics (salbutamol,salmeterol (serevent), anticholinergics (Atrovent) and methylxanthines (theophylline medications).

TOPIC 3. Chronic gastritis (gastroduodenitis) (CGD) is a chronic relapsing focal or diffuse inflammation of the mucous membrane of the stomach (and duodenum) with secretory, motor and

evacuation disorders.

Duodenal ulcer (DU) is a chronic disease with acyclic course, characterized by an ulcer in the duodenal bulb.

The etiology. In the development of CGDand DU the following factors are important: alimentary factors (irregular nutrition; fast feeding; dry eating, rough food, smoked food, spices; too hot food), immunological, infectious factor (Helicobacter pylori (Hp),hereditary factors(caused genetically hyperchlorhydria, pepsinogen-1 in blood, increased gastrin level after meal,low resistance of mucous coat to damaging factors).

Pathogenesis. The occurrence of CGD and DU occurs when an imbalance between the factors of aggression (overproduction of hydrochloric acid and pepsin, infection with Helicobacter pylori, bile acids in duodenal reflux) and mucosal protective factors (adequate production of mucus by surface cells and the secretion of bicarbonate ions by the antrum and duodenum, adequate motor activity antrumand duodenum, sufficient synthesis of prostaglandins, trophic function of the mucous membrane and its regeneration).

Classification of chronic gastritis

I. By etiology: infectious (bacterial, viral, fungal), chemical, autoimmune, allergic;
II. By endoscopic picture: a) superficial, b) hypertrophic, c) atrophic, d) erosive;
III. By histological picture: a) superficial, b)atrophic, c) atrophic-hyperplastic;
IV. By secretory function of the stomach: 1) hyperacidic, 2) normacidic, 3)hypoacidic.
V. The phases of the disease distinguished: 1. Exacerbation; 2. Remission.

According to the Sydney classification of gastritis following types are distinguished:

1. Gastritis type B (antral gastritis) - bacterialgastritis is caused by Helicobacter pylori (90% of total) and characterized by changes in the antrum; immune disorders are absent, the level of serum gastrin is normal or even reduced.
2. Gastritis type A - Endogenous, autoimmune gastritis (1-3% of all cases of gastritis) are due to the production of autoantibodies to the parietal cells of the stomach; the body of the stomach is primarily affected, primary atrophic changes and reduction of gastric secretionare typical, serum gastrin content and increased blood circulating antibodies to parietal cells are present
3. Gastritis type C (refluxgastritis)

Clinical manifestations. The clinical picture consists of two main syndromes - pain and dyspeptic.

In case of increased (or normal) hydrochloric acid (usually **gastritis type B**) pain isintense and prolonged, associated with eating. Early pain occurs during meal or 10-20 minutes after eating andis typical of gastric fundus, and late pain (that appears 1-1,5 hours after a meal) is typicalforantralgastritis. In case of reduced secretion of hydrochloric acid (usually **gastritis type A**) pain is poorly expressed, characterized by long-standing pains in the epigastrium. After meal, a feeling of heaviness /fullness/ in the upper abdomen appears;

In the case of **duodenal ulcer** pain is more persistent which is predominantly of nocturnal origin early morning hours.Pain is localized in epigastric and piloroduodenal area, sometimes radiating to the left upper quadrant.

Dyspeptic symptoms are severe heartburn, nausea, vomiting,constipation and appetite disorders. There are no any specific clinical manifestations of Hp infection, it is sometimes asymptomatic.

Diagnosis should be defined on the basis of clinical and anamnestic data and additional laboratory and diagnostic findings (gastroscopy, intragastric pH examining,). To detect

Helicobacter Pylori (HP) in the stomach mucosa different methods are used - Microbiological method means incubating HP colonies in special selective environment, Morphological method of HP identifying in biopsy materials of the stomach mucosa, Biochemical method (examining biopsy materials of ofthe stomach mucosa for ureasepresense), Immunological diagnostic of HP (identifying immune bodies to HP).

Treatment.

1. The regime. Diet;
2. Antacid therapy (Almagel, Maalox, Phosphalugel);
3. Antisecretory therapy. There are three groups of drugs: 1. M-anticholinergic-gastrotsepin, 2.H2-histamine blockers - ranitidine, famotidine, nazotidin, 3. Proton pump inhibitors (omeprazole);
4. Citoprotectors - Venter, De-Nol, Cytotec, sucralfate;
5. H. pylori therapy. Eradication therapeutic complex is administered in case of chronic gastroduodenitis and peptic ulcer, associated with Hp infection. It is composed of citoprotectors (de-nol, bismofalk), antibiotics (amoxicillin), antimicrobials (metronidazole).
6. In case of motor-evacuation disfunctionMotilium is used and to correct pathological casts of duodenal contents into the stomach adsorbents (smecta, enterosgel, wheat bran) should be prescribed.
7. Physiotherapy (laser therapy, inductothermy).

Biliary tract dysfunction

Biliary tract dysfunction is any motility disorder of the gallbladder and sphincter which are manifested by bile flow impairment into the duodenum and accompanied by pain in the right upper quadrant.

Etiopathogenesis. The pathogenesis of primary dysfunction is a disorder of neurohumoral regulation and condition of the **vegetative nervous system**that leads to motility disorders of the gall bladder and sphincter.Secondary ones accompany diseases of the stomach and duodenum.

Classification of biliary tract dysfunction.

Dysfunction of the biliary tract: a) hypotonic (hypokinetic); b) hypertension, (hyperkinetic).

Clinic. The main clinical syndromes are:

1. **Pain syndrome.** In hypertensive form of dysfunction pain is sharp, paroxysmal, stabbing, cutting, short-term (about 5-15 minutes), located in the right upper quadrant and occurs due to negative emotions, anxiety, physical exertion. In hypotonic biliary tract dysfunction following symptoms are detected: discomfort or a feeling of heaviness in the right upper quadrant, the pain is prolonged, dull, occasionally it becomes more intense due tofatty food consumption (1-1.5 hours), after exercise (jumping, running).
2. **Dyspeptic syndrome** - loss of appetite, bitter taste in the mouth, nausea, tendency to constipation in hypertensive form, to diarrhea in hyperkinetic one.

Diagnostic:

1. Ultrasonography of the liver and biliary tract;
2. Level of bilirubin, cholesterol and bile acids in biochemical studies of bile.
3. **Cholecystostsintigrafiya**

Chronic cholecystitis (CC) is a chronic inflammation of the gallbladder and bile ducts, characterized by pain, dyspeptic, intoxication syndrome and minor liver dysfunction.

Etiopathogenesis. The causetive agents of the inflammatory process are often represented as: Escherichia coli, Proteus, Staphylococcus, and others. CC can result from viral hepatitis, as well as

after enteroviral disease. Microorganisms can get into the bile ducts in three ways: ascending (from the intestines), hematogenous, lymphatic (from liver, pancreas, gastrointestinal tract)
Under physiological conditions, bile has a strong bactericidal activity against many pathogens. Ifbileoutflow has been impaired and cholestasis develops due to dysfunction or abnormalities of the biliary tract and the inflammatory process occurs.
Clinical manifestations.
Three major clinicalsyndromes are distinguished:
1. Pain syndrome - dull, aching pain or acute pain episodes in the right upper quadrant, worsened by physical exertion, often occurs at night. Pain characterdepends on the type of biliary dysfunction.
2. Dyspeptic syndrome - loss of appetite, nausea, vomiting that occur rarely, and constipation, bitter taste in the mouth and belching.
3. Intoxication syndrome.
Typical symptoms of cholecystitis are

- Hepato-cystic symptoms that are detected by clinical examination of child;
- liverenlargement;
- An ultrasound examination shows extended or decreased gallbladder with thickened wallof 3 mm or more, extended bile ducts
- Blood test detects slight leukocytosis, hypoproteinemia, dysproteinemia, thymol test positive, an increased cholesterol, alkaline phosphatase

Treatment.
1. Regime and diet.
2. choleretics - drugs that stimulate bile formation: allohol, holenzim, holagon, as well as chemical derivatives (Nicodin, oxafenamid,) and herbal preparations - flavin, holosas, febihol.
3. cholekinetics - drugs that stimulate bile secretion and improve the tonus of the gall bladder (cholecystokinin, pituitrin, egg yolks) and relaxation of sphincters (atropine sulphate, platifillin hydrogen tartrate, an extract of belladonna, no-spa).
4. Antibiotics are prescribed in children in case of other foci of infection.
5. Lipoic acid, hofitol, potassium orotate, holesol, hepatophalc, essentiale, Karsil, gepabene, vitamins: B1, B6 are used to promote further recovery of liver function

Tests

Topic 1.

1. Which of the following typical for the initial period of rickets?
A. O-and H-shaped deformations of the limbs.
B. Increased body temperature.
C. Increased sweating, anxiety.
D. Resistant hypocalcemia, hypophosphatemia.

2. What is the vitamin D daily therapeutic dose?
A. 2000-5000 IU.
B. 10 000-12 000 IU.
C. 9000-10 000 IU.
D. 7000-8000 IU.
E. 500-1000 IU.

3. Which of the following is the III-rd stage of the rickets' illness?
A. Agitation, the disturbance of the sleep.
B. Increased sweating.
C. Conditional reflexes descent.
D. Tachycardia, functional noises.

4. Which one of the following findings is typical for subacute clinical course of rickets?

A. Craniotabes.
B. The prevalence of the bonessoftness.
C. The prevalence of the osteoid tissues hyperplasia.
D. Presence of hypocalcemia.

5. What is the level of the blood serum calcium in hypervitaminosis D?
A. Hypercalcemia (calcium levels above 2.89 mmole/l).
B. Within the physiological fluctuations.
C. Hypocalcemia (calcium levels below 2.3 mmole/l).

.

Tasks

1. A 9-months -oldboy was delivered by ambulance (his mother complained of cramps) with loss of consciousness and body temperature rising to 37,2° C. He felt ill 30 minutes ago, after crying convulsions of the face muscles appeared and quickly spread to the limbs, cyanosis developed. Anamnesis: the child was born after first physiological pregnancy and first normal birth. Household living conditions are poor. The examination revealed pale skin, expressed frontal and parietal tubers, no teeth, inferior aperture of thorax was dilated, costal rosary palpated. The heart and lung have any changes. The abdomen is soft, the liver protrudes from the costal margin to 3 cm. Kerning's andBrudzinsky's reflexes are negative. Which one is a correct diagnosis?
A. Spasmophilia.
B. Epilepsy.
C. Meningitis.
D. Hyperthermia convulsions.
E. Congenital heart disease.

Topic 2. Tests

1. What causative agent causes home pneumonia most often?

A. Pneumococcus.
B. Streptococcus.
C. Proteus.
D. Hemophilic bacillus.
E. Staphylococcus.

2. Which of the following agents causes the development of interstitial pneumonia?

A. Staphylococcus.
B. Pneumococcus.
C. Blue pus bacillus.

D. Klebsiella.
E. Mycoplasma.

3. What is most important criterion for differential diagnosis of acute bronchitis and acute pneumonia?

A. Symmetric physical data.
B. Asymmetric physical data.
C. Hyperthermia more than 3 days.
D. Vocal tremorchange.
E. The mixed character of dyspnea.

4. What is the main clinical diagnostic syndrome of acute pneumonia?

A. Dyspnea.
B. *Weakened* or bronchial breath sounds.
C. Local crepitation.
D. Numerous bubbling rales.
E. Dullness of percussional tone.

Tasks

1. An 8-years- old childcomplains of headaches, fever, febrile body temperature, dyspnea, paroxysmal coughing with small amount of sputum. He has sick for a week. Several classmates are sick with pneumonia. Mixed character dyspnea, dullness of percussional tone paravertebrally, rough respiration, moderatediffuse dry rales are observed. Enhanced lung marking with microfocal diffuse infiltration is detected by an X- ray diagnostic. Make a diagnosis of the disease.

A. Focal pneumonia.
B. Interstitial pneumonia.
C. Obstructive bronchitis.
D. Simple bronchitis.
E. Miliary tuberculosis.

Topic 3. Tests.

1. What causative agent plays the leading role in ulcer disease development?
A. Streptococcus.
B. Klebsiella.
C. Helicobacter.
D. Staphylococcus.
E. Escherichia.

2. What preparation is appropriate to use for H. pylori eradication?
A. Benzylpenicillin sodium salt.
B. Gentamycin.
C. Sucralphate.
D. Biseptol.
E. Clarythromycin.

3. What antisecretory preparation is mostused commonly in pediatric practice?
A. Ranitidin.
B. Famotidin.

C. Omeprazol.
D. Gastrozepin.
E. Cimetidin.

4. Withetiopathogenetical form of chronic gastritis is complete recovery probable?
A. Gastritis A - autoimmune.
B. Gastritis B - helicobacter- associated.
C. Gastritis C - reactive, chemical, due to duodenogastric reflux.

5. What gastroenterological pathology is prevalent among children?
A. Isolated gastritis.
B. Isolated duodenitis.
C.Gastroduodenitis.
D. Gastric ulcer disease.
E. Duodenal ulcer disease.

Task 1. A 12 –year- old boy complains of poor appetite, heartburn, paraumbilicaldullpain that often occurs in the morning on an empty stomach and 2- 3 hours after meals, but it subsides immediately after the meal. Objectively: there are pale skin, soft abdomen but painful while deep palpation in the epigastrium and piloroduodenal region. What diagnosis is the most likable?

A. Gastric ulcer disease.
B. Mesenteric lymphadenitis.
C. Chronic cholecystitis.
D. Chronic gastroduodenitis.

List of references

4. Pediatrics [Text] : textbook / O. Tiazhka [et al.] ; ed. O. Tiazhka ; Ministry of Public Health of Ukraine, National O. O. Bohomolets Medical University. - Vinnytsia : Nova knyha, 2011.
5. Nelson Textbook of Pediatrics, 2-Volume Set, 20th Edition
6. TEXTBOOK OF PROPEDEVTIC PEDIATRICS For students of II – III years. Kyiv 2006. Autors: O. Vinnitzka, T.Lutaj, A.Antoshkina, Ju. Piatnitzki, M. Vasiukova, N. Gorobetz, L.Martinova, T. Mellina, L.Slipachuk, O.Stroj, P.Tovmash
7. Pediatrics. Guidance aid for students of V year of study. Edited by Professor O. Tiazhka

MINISTRY OF PUBLIC HEALTH OF UKRAINE
O.BOHOMOLETSNATIONALMEDICALUNIVERSITY

PEDIATRIC DEPARTMENT N5

"Approved"
on the methodical conference
Department of Pediatrics № 5
protocol №2 from 07.09.2016

GUIDELINES
FOR STUDENTS

Academic discipline	Pediatrics
Study subject	The most common children cardiovascular diseases. The most common children urinary diseases. Hemorrhagic diathesis in children
Course	4
Faculty	Dental

Kyiv-2016

Relevance of the topic.

Topic 1.Acute rheumatic fever is still one of the most important problems of modern clinical pediatrics. Recently the course of this disease in children has become more favorable but it has had a tendency to recurrence, progression and formation of acquired heart lesions. The problem of rheumatic fever is very relevant as it leads to disability of patients in adulthood.

Topic 2. Glomerulonephritis ranges the 3rd among children kidney diseases. The complexity of the pathogenesis, clinical manifestations, the need for long-term treatment, a tendency to chronic inflammation, the possibility of chronic renal failure at young working age determine the relevance of this problem.Among the diseases of the urinary system inflammatory lesions of kidneys and urinary ways are most common. Most of all hospitalizations in the nephrological department are inflammatory diseases, among which pyelonephritis takes the leading place. This disease may become chronic if not to diagnose it in time and provide proper treatment.

Topic 3. According to WHO, in Europe the frequency of hemophilia A is 1:10 000, Hemophilia B - 1:40 000 male births, and other hereditary coagulopathy totally - 5-6:1 000000, idiopathic thrombocytopenic purpura - 2-2,5:100 000, hemorrhagic vasculitis - 2,5:10 000.The growth of this disease over the last 10-15 years in infants and young children, is mainly due to idiopathic thrombocytopenic purpura, and hemorrhagic vasculitis associated with an increasingtendency to allergyamong children and environmental disadvantage. Diagnosis of hemorrhagic diathesis in childhood is important to prevent bleeding, which can lead to severe complications and disabilities.

Questions for self– checking

1. Etiology ofglomerulonephritisand pyelonephritis
2. Clinical features of pyelonephritis in children according to the age
3. The main differential- diagnostic criteria of pyelo- and glomerulonephritis.
4. Antibiotics use for pyelonephritis treatment.
5. Chemical preparations used totreat pyelonephritis
6. Laboratory research methods applied for pyelonephritis patients
7. Clinical course features of glomerulonephritis in children, the frequency of different syndromes, according to the age.
8. Diagnostic criteria of glomerulonephritis with nephrotic and isolated urinary syndromes.
9. Basic principles of patient's treatment with glomerulonephritis, due to observed syndromes. Indications for corticosteroids and cytostatic prescription.
10. Etiology and pathogenesis of acute rheumatic fever.
11. Diagnostic criteria of acute rheumatic fever in children.
12. Characteristics of different variants of rheumatic carditis in children.
13. Clinical features of rheumatic arthritis in children, differential diagnosis.
14. Nervous system lesions in children with acute rheumatic fever.
15. The treatment principles of acute rheumatic fever in children.
16. Primary and secondary prevention of rheumatism in children.
17. Reciprocal between rheumatism in children and health of adults.
18. Classification of congenital heart anomalies.

19. Clinical features and treatment principles of congenital heart anomalies.
20. The concept of hemorrhagic diathesis. Classification.
21. Hemophilia A and B. The etiology, pathogenesis, clinical features, diagnosis.
22. Replacement therapy in acute care patients with hemophilia.
23. Hemorrhagic vasculitis in children. Etiology, pathogenesis, clinical forms, diagnosis, differential diagnosis, treatment and prognosis.
24. Thrombocytopenic purpura in children. The etiology, pathogenesis, clinical features, diagnosis, differential diagnosis, treatment and prognosis.
25. Emergency treatment for bleeding

Topic 1. Acute rheumatic fever (ARF) is an autoimmune disease that may occur after a group A streptococcal throat infection that causes inflammatory lesions in the connective tissue, especially that of the heart, joints, blood vessels, and subcutaneous tissue.

Causes. ARF is the result of interaction of the organism with beta-hemolytic streptococcus group A. B-hemolytic group A streptococcus also causes human diseases such as scarlet fever, pharyngitis, tonsillitis, and then rheumatic process may develop. However, streptococci are not the direct, causative agent of rheumatic fever, because rheumatic fever develops in only 0.3 - 3% of children who have had quinsy, pharingitis or scarlet fever. Therefore it is believed that genetic factors andheridatory factors are essential in developing the disease, especially in the course of every specific patient.

Pathogenesis. Toxic and immunological theory of ARFs generally accepted.

1. The toxic mechanism - associated with the direct influence of exo-and endotoxins of streptococci on the cells and tissues

2. The mechanism of cross-reacting responses - streptococcus antigens cross-react with antigens of the myocardium.

3. Immune mechanism - the formation of anti-streptococcal antibodies, formation of immune complexes and their deposition on the basal membrane of capillaries, arteries, and synovial membranes, causing their damage.

4. Autoimmune mechanism - the formation of autoantibodies that react with myocardial fibril and cause their necrosis and increase the expression of myocarditis.

At present, according to WHO recommendations, diagnostic criteria developed by Johns and revised by ACA in 1992 are used for acute rheumatic fever. The **major Johns' criteria** for diagnosis include arthritis in several joints (polyarthritis), heart inflammation (carditis), subcutaneous nodules (Aschoff bodies), Sydenham's chorea, skin rash (Annular erythema).The **minor criteria** include joint pain (arthralgia), fever; indices of acute phase: ESR(erythrocyte sedimentation rate), elevated C-reactive protein; EKG changes (prolonged PR interval),andother laboratory findings (increased titer of antistreptococcal antibodies: AST-O, elevated streptococcal antigen test).

Clinical patterns (Physical). ARF strikes children at the age ofabove 3 years, oftenat school age. The first signs of ARF can be evident in 2-3 weeks after quinsy or pharyngitis manifesting as fever, intoxication symptoms, articular syndrome, carditis, etc.

Polyarthritis is characterized by acute onset, swelling, redness, warmth, and joint tenderness, absence of deformities and any functional alterations in the affected joints. Arthritis of ARF is usually symmetrical and involves major joints, such as the knees, ankles, elbows, and wrists. Arthritis is classically described as migratory.

Carditis. It is usually pancarditis involving the pericardium, the myocardium, and the endocardium. Myocarditis is the most common manifestation of rheumatic heart disease. The following symptoms are observed – worsening of the child's general condition, heart pain, weakness, fatigue, pale skin, the shadows under his eyes, cyanosis of the lips or nasolabial triangle, weak pulse, tachycardia, bradycardia; Percussion shows - the dilation of heart borders to the left; auscultation reveals that 1 tone is muted, short systolic murmur; ECG - lengthening of atrioventricular conductivity, the change of the terminal ventricular complex can be seen. Endocarditis (the defeat of valvular heart).The affection of mitral valveis characterized by the appearance of systolic murmur "blowing" tone, of the moderate loudness, it is auscultated on the top of heart, well gets in the left axillary region, can be accompanied by the reduction of the first tonesonority.The affection of aortic valve is characterized by diastolic murmur which is better heard in the Botckin's point on the inhalation, very short, then it is auscultated as effuse diastolic murmur along the left side, maximum is from the left in the third- fourth intercostal space. Pericarditis indicates, usually pancarditis as all layers of the heart are affected. The appearance of pericarditis is clinically expressed by a sudden worsening of the patient's state, complaints about a sharp pain in the heart area, dry intrusive cough, forced position in bed - sitting leaning forward (orthopnea), cyanosis, murmur of pericardium rubbing (in case of dry fibrinous pericarditis).

Subcutaneous nodules. Theytend to occur several weeks after illness onset, which are usually painless, and are found primarily over the bony surfaces or prominences and in tendon sheaths. The common sites include the elbows, knees, wrists, ankles, over the Achilles tendon, the back of the scalp, and spinous process of the vertebrae.They usually persist for 1-2 weeks.

Erythema marginatum. It is manifested as pale pink annular rash, which does not elevate over the skin surface and disappear upon pressure. Patients or parents may report a nonpruritic, painless, serpiginous, erythematous eruption. The skin of the trunk and the limbs is frequently involved.

Sydenham chorea.This is a neurological disorder characterized by emotional lability, personality change, muscular hypotonia (weakness), and uncoordinated, involuntary, purposeless movements, hyperkineses, frequently bilateral; hyper- or hyporeflexia; various vegetative disorders.

Medical Care.

1. **Regimen** – bed rest for 2-3 weeks.
2. **Diet** – with reduced amount of salt and liquids, food rich in potassium is prescribed (baked potato, raisins, dried apricots, and prunes).
3. **Etiotropic therapy** – Penicillin, Amoxicillin, macrolids (Azithromycin, Claritromycin), cephalosporins. In case of allergy to Penicillin, Erythromycin is recommended.
4. **Pathogenetic therapy.**

- Non-steroid anti-inflammatory drugs (NSAID) effects are mediated through inhibition of prostaglandin synthetase preventing the synthesis of prostaglandins possessing pro-inflammatory activities. Acetyl salicylic acid, Indometacin (Metindol), Voltaren, Brufen (Ibuprofen), Maproxen, Mefenamic acid are used.

- Steroid anti-inflammatory drugs are also effective but should probably be reserved for patients whom salicylates fail to respond. Prednisolon and Metipred are often used.

5. **Symptomatic treatment** comprises the drugs intended for the normalization of vascular permeability and metabolic process in the heart; antihistamine drugs. In case of insufficient blood circulation, the cardiac glycosides, diuretics, peripheral vasodilators and other drugs are used**.**

Congenital anomalies of the heart and blood vessels arise during the first 10 wk of embryonic development and are present at birth. The incidence is 1/120 live births; estimated risk is 2 to 3% in children with an affected 1st-degree relative.

Etiology.

- chromosomal abnormality (eg, trisomy 13, 18, or 21; Turner's syndrome) -about 5% of patients
- genetic syndrome (eg, Holt-Oram, Noonan's, Williams, 22q11 deletion)
- maternal illnesses (eg, diabetes mellitus, SLE, rubella)
- environmental exposure (eg, to thalidomide, isotretinoin, lithium or
- alcohol or a combination of some factors

Pathophysiology. Congenital heart anomalies are classified as

1. Cyanotic: Tetralogy of Fallot; Transposition of the major arteries; Tricuspid atresia; Pulmonary atresia; Hypoplastic left heart syndrome; Persistent truncusarteriosus; Total anomalous pulmonary venous return
2. Acyanotic: A) left-to-right shunts: Ventricular septal defect; Atrial septal defect; Patent ductusarteriosus; Atrioventricularseptal defect and B) obstructive lesions: Pulmonary stenosis; Aortic stenosis; Aortic coarctation

Physiologic consequences of congenital heart anomalies vary greatly, ranging from asymptomatic heart murmur or abnormal pulses to severe cyanosis and heart failure (HF).

Symptoms and Signs

Manifestations of various heart anomalies are limited to several common ones: 1.Murmurs; 2. Cyanosis; 3. HF. Less commonly, chest pain, diminished or nonpalpable pulses, circulatory shock, and arrhythmias are present.

Murmurs.Most left-to-right shunts and obstructive lesions cause systolic murmurs. Increased flow across the pulmonary or aortic valve causes a midsystolic (ejection systolic) murmur. Regurgitant flow through an atrioventricular valve or flow across a VSD causes a holosystolic (pansystolic) murmur, possibly obscuring heart sounds as its intensity increases.

PDA causes a continuous murmur that is uninterrupted by the 2nd heart sound (S2) because blood flows through the ductus during systole and diastole.

Cyanosis: This manifestation is characterized by bluish discoloration of mucous membranes or nail beds, clubbing of nail beds, or pulse oximetry < 93 to 95%.

Heart failure: in infants, symptoms or signs of HF include: Tachycardia; Tachypnea; Dyspnea while feeding; Diaphoresis; Restlessness; Irritability.

Dyspnea while feeding causes inadequate intake and poor growth, which may be worsened by increased metabolic demands and frequent respiratory tract infections. Hepatomegaly is common. In contrast to older children, most infants do not have distended neck veins and dependent edema, although they occasionally have edema in the periorbital area.

Other manifestations: In neonates, circulatory shock may be the first manifestation of certain anomalies. Neonates look extremely ill and have cold extremities, weak pulse, low BP, and reduced response to stimuli.

Chest pain may be manifested by unexplained irritability in infants with a coronary artery anomaly. In older children and adolescents, chest pain due to a cardiac etiology is usually associated with exertion and may be caused by severe aortic stenosis, pulmonary stenosis, or Eisenmenger's syndrome.

Diagnosis

- Pulse oximetry, ECG, and chest x-ray
- Echocardiography
- Sometimes cardiac MRI or CT angiography, cardiac catheterization with angiocardiography

Diagnosis is suggested by the presence of heart murmurs, abnormal pulses, cyanosis, or HF.

Treatment

- Medical treatment of HF (eg, with O_2, diuretics, ACE inhibitors, digoxin, and salt restriction)
- Surgical repair of anomalies amenable to correction

After medical stabilization of acute HF symptoms or cyanosis, most children require surgical or transcatheter repair; the exceptions are certain VSDs that are likely to become smaller or close with time. Transcatheter procedures include balloon atrial septostomy for palliation of severely cyanotic neonates with transposition of the great arteries, balloon dilation of severe aortic or pulmonary valve stenosis, and transcatheter closure of cardiac shunts (most often atrial septal defect and PDA).

TOPIC 2: The most common children urinary diseases Acute post-streptococcal glomerulonephritis is an immunocomplex disease with predominantly glomerular lesions occurring in 10-14 days after streptococcal infection (sore throat, impetigo, scarlet fever, pyoderma, etc.) characterized by nephritic syndrome.

The **causes** of acute glomerulonephritis .Acutepoststreptococcal glomerulonephritis (APSGN) is caused by nephritogenic strains of group A beta-hemolytic streptococci. Glomerulonephritis results from viral infection, drug treatment (mercury drugs, antibiotics, sulfonamides), infusion of protein substances, consumption of large quantity of honey, after vaccination, biting insects, snakes.

The disease develops in 10-14 days after nasopharyngeal infection (sore throat), or 3 weeks after skin infections (impetigo, pyoderma).

The **pathogenesis** of acute glomerulonephritis. Streptococcal antigens are deposited in the glomeruli during acute streptococcal infection. After 10-14 days, the immune response begins, during which antistreptococcal antibodies bind to antigen and circulating immune complexes are formed and deposited in the renal glomeruli.

Clinical manifestations of acute glomerulonephritis can be divided into two main groups: the extrarenal (intoxication, hypertensive, edematous, hematological syndrome) and renal (urinary syndrome, low back pain)

.**Extrarenal features:**

1) Intoxication syndrome (malaise, weakness, nausea, pallor, low-grade fever) is caused by acidosis and accumulation of toxic metabolic products in the blood.
2) Edema syndrome. Its appearance is due to increased hydrodynamic pressure, decreased colloid-osmotic blood pressure, increased capillary permeability, increased reabsorption of water and the reduction of urinary sodium excretion. Visible edemas appear with in body weight gain by 10%. Edemas are located mainly on the face, legs, lumbar region, which are more expressed in the morning.
3) Hypertension syndrome includes high blood pressure, headaches, tachycardia, extrasystoles, muffled heart sounds, shortness of breath, damage on the eye fundus. Hypertension is caused by an increase in blood volume because glomerular filtration is reduced and fluid retains in the bloodstream. The greatest elevation of blood pressure (usually up to 130/90 mm) is observed in the first days of the disease,thenit becomes lower.
4) Hematological syndrome - a mild normochromic anemia, mild leukocytosis, eosinophilia, accelerated ESR (30-40 mm / h).

Renal features include:

1) Urinary syndrome. It is characterized by the following features: oliguria - decrease urine output by 50-80%, the relative density of urine is high (1030 and above); albuminuria - constant symptom in glomerulonephritis, proteinuria is 1-2 grams per day; hematuria (urinary excretion of red blood cells) - is in 100% of cases, with macrohematuria urine becomes brownish-red or brown, sometimes leucocyturia - usually is moderate; cylindruria- due to hyaline casts.
2) Changes in the biochemical blood parameters - hypoproteinemia (protein <60 g / l) disproteinemia(decrease of albumin increasing the alpha-2 and gammaglobulin), hyperlipidemia and hypercholesterolemia (> 7 mmol / l), hyperasotemia (> 8.32 mmol / L), kreatinemiya (> 0.105 mmol / l), increased titer of ASL-0, circulating immune complexes, fibrinogen (> 6 g / l).

Taking into the account glomerulonephritis course the following syndromesare distinguished:

- nephrotic - is presented by severe edema (face, waist, external genitals, lower limbs, anasarca), proteinuria> 2.5 g / day, hypoproteinemia (<25 g / L), hypercholesterolemia, hyperlipidemia;

- isolated urinary - hematuria, cylinduria, proteinuria to 1.5 g / l, leucocyturia;

- nephritic - edema, hematuria, hypertension.

Treatment

Basic therapy: 1. Bed rest. Diet - restriction of salt, protein, food rich in potassium and dosed fluid intake.2. Antibiotic therapy (group of penicillin, cephalosporins, macrolide) is prescribedfor 1.5-2 months.3. Antihistamines (suprastin, tavegil, fenkarol); 4.Vitamin therapy. Vitamins C, B, A, and E are administered in therapeutic doses.

Symptomatic therapy includes hypotensive medicines, diuretics.

5. Diuretics are prescribed to patients with edema, hypertensive syndrome and oliguria (furosemide, hydrochlorothiazide, triamterene).

6. Antihypertensive therapy is administrated in case of severe and resistant hypertension, use of reserpine, raunatin, captopril; dibazol.

Pathogenetic therapy is prescribed on the base of disease form**:**

7. Immunodepressants: glucocorticoids (prednisone) are prescribed in case of nephritic syndrome,nephrotic syndrome with hematuria and hypertension, acute glomerulonephritis with acute renal failure; cytostatics (hlorbutin, azathioprine) are prescribed for hormone - resistant progressive disease;

8. Anticoagulant (heparin) and antiplatelet (Curantylum, dipyridamole) therapy. The indications for the use of heparin are edema, hypercoagulation, reduced renal function and in case of nephritic syndrome.

9. Nonsteroidal anti-inflammatory drugs (aspirin, indomethacin). If thereis acute glomerulonephritis with isolated urinary syndrome, nephritic syndrome and tendency for long course it can be prescribed.

10. When hematuria is persisted for more than 2 months, the appointment of quinoline drugs (delagil, Plaquenil) is recommended.

Pyelonephritis is a non-specific infectious-inflammatory kidney disease, mainly affecting the *renal pelvis system*, tubules and interstitium, which is characterized by signs of infectious disease and impaired renal function by tubulointersticial type.

Etiology. In the etiology of pyelonephritis the Escherichia coli (54.2%); klebsiela (5-20%), Proteus (4.5%), Staphylococcus aureus (4-15%), Streptococcus (10.5%) and virusesare important causative agents.

Pathogenesis.There are the following ways of microorganismspenetration: ascending, hematogenous or lymphogenous (rarely).Two stages are distinguished in the pathogenesis of pyelonephritis: 1) non-specific (inflammatory) - the destruction of the renal parenchyma occurs under the influence of lysosomal enzymes and neutrophil superoxide radicals due to the activation of the complement system; 2) specific (immunological) the formation of immune complexes and their deposition on the basal membrane tubules.

Classification of pyelonephritis.

By the form: 1) Primary - develops in the absence of abnormalities of the urinary system (ie, the disease develops in a healthy person). 2) Secondary - occurs due to organic and functional disorders or metabolic nephropathy (oxaluria, uraturia, tubulopathy et al.).

By the course:1)Acute - is characterized by the active stage the disease and recurrant development of symptoms with complete clinical and laboratory remission for 6 months; 2) Chronic - is characterized by the duration longer than 6 months from the beginning or in the presence of at least 2 relapses during this period

By activity process: 1) active stage, 2)partial remission - no clinical signs, but urinary syndrome iskept, 3) complete clinical- laboratory remission - no both clinical and laboratory findings.

For renal function: 1) without renal failure, 2) with impairment of kidney function, 3) chronic renal failure.

Clinical picture:

1. Intoxication syndrome - fever, chills, headache, malaise, anorexia, pallor of the skin
2. Pain syndrome. Pain in the abdomen and lumbar region which is aggravated by physical effort
3. Urinary syndrome: leucocyturia of neutrophilic type (more than 50% neutrophils),bacteriuria (more than 100 thousand microbial cells in 1 ml of urine), proteinuria (less than 1 g / l protein).
4. Dysuria syndrome (with involvement of the lower urinary tractin the pathological process) - frequent urge to urination, pain or sensation of burning (particularly at the end of urination) and may urinary incontinence
5. In the analysis of blood: increased erythrocyte sedimentation rate (more than 15 mm / hr.), Leucocytosis (more than 9×10^9 / L) with shift to theleft.

Treatment

1. Diet. Use of vegetable andprotein food without salt and fluid restrictions and exception fat, fried, spicy food, extractives are prescribed.

2. Etiotropic empirical therapy - non-nephrotoxic antibiotics:

- "Protected" penicillins -amoxicillin + clavulanic acid (eg, amoxiclav, Augmentin), ampicillin + sulbactam (unazin).
- II generation of cephalosporins (cefuroxime).
- III generation of cephalosporins (cefotaxime).

3. Uroantiseptics after a course of antibiotics

Topic 3. Hemorrhagic diathesis is an inherited predisposition to any abnormalities characterized by profuse bleeding. According to pathogenesis, it is classified into 1. coagulopathy, 2. vasopathy and 3.platelet disorder (thrombocyte-penia and thrombocytopathy). Each type is subdivided into congenital and acquired.

Haemophilia is a group of hereditary genetic disorders that impair body's ability to control blood clotting or coagulation, used to stop bleeding when a blood vessel is broken.

Causes. Haemophilia is a recessive sex-linked, X chromosome disorder involving a lack of functional clotting Factor VIII (in hemophilia A/classic hemophilia, and represents 80% of haemophilia cases),FactorIX (in hemophilia B/Christmas disease), XI (in hemophilia C/Rosenthal's syndrome is an autosomal genetic disorder (i.e. not X-linked)) or XII (in hemophilia D).

Signs and symptoms

1. Hematoma type of bleeding, which is common for hemophilia, is characterized by painful, intense hemorrhages and prolonged bleeding
2. Hemorrhages in the subcutaneous tissue, muscles, major joints, in the peritoneum and the retroperitoneal space are observed in hemophilia.
3. A typical symptom of hemophilia is hemorrhages into joints (hemarthrosis), which is very painful, and often accompanied by high fever. Large joints are most often affected - knees, elbows, ankles, at least - shoulder, hip and small joints of the hands and feet. This is most common with severe haemophiliacs and can occur spontaneously (without evident trauma). If it is not treated promptly, joint bleeds can lead to permanent joint damage and articular shape alteration.
4. Hemorrhages into soft tissues such as muscles and subcutaneous tissues hematomas are less severe but can lead to damage and so they require treatment.
5. Hemorrhages in the bone also occur, resulting in aseptic necrosis, decalcification of the bones.
6. Hemophilia is characterized by prolonged bleeding from the mucous membranes of the nose, gums, mouth, rarely gastrointestinal tract, kidneys. Any medical procedure, especially intramuscular injection, tooth extraction and tonsillectomy can lead to prolonged bleeding. Hemorrhage in the brain and menings, causing death or serious impairment of the CNS are alsolikable to develop.

Laboratory diagnostic criteria

1. Prolonged coagulation time more than 10 minute
2. Prolonged recalcification reaction more than 200 seconds
3. Reduced consumption of prothrombin.
4. Deficiency of the antihemophilic factors VIII or IX
5. Thromboplastin generation test

Henoch-Schönleinpurpura (HSP) is an inflammatory disorder characterized by a generalized **vasculitis** involving the small vessels of the skin, GI tract, kidneys, joints, and, rarely, the lungs and CNS. It is the most common type ofvasculitis in children. It is a disease of the systemic vasculitisgroup.

Etiology. The etiology of Henoch-Schönleinpurpura is unclear. It is thought to be multifactorial with genetic, environmental, and antigenic components. The current understanding of the etiology of HSP suggests the involvement of toxins, viruses, idiopathic causes, and drugs.This disease is associated with infectious factor (viral or bacterial)**,**vaccination, drug intolerance, food allergies, trauma, cooling**,** insect sting**,** allergic predisposition of the organism, which in the presence of foci of chronic infection (chronic tonsillitis, dental caries, Tbc, etc.) leads to a child'sreactivity decrease. These factors may precede the development of disease.

The beginning of the disease develops 1-4 weeks after a sore throat, viral respiratory infections, scarlet fever or other infectious disease. The factors are more predisposing than etiological.

Pathogenesis. HSPis thought to be an immunoglobulin A (IgA)–mediated autoimmune phenomenon. The leading factor in the mechanism of the disease is the influence a provoking factor. An unknown antigenic stimulant has been postulated to cause a rise in IgA. The clinical manifestations of HSP are the result of antigen-antibody complexes depositing throughout the body, which cause hallmarks of HSP - typical rash, migratory polyarthritis, renal and GI involvement.

Age.HSP affects primarily children. Adults are rarely affected.Approximately 75% of cases occur in children aged 2-11 years.

The **clinical picture** is characterized by various combinations of typical syndromes: hemorrhagic skin, joint, abdominal and kidney, rarely - pathology of other organs.The prodrome is associated with the following: headache, anorexia, fever. After the prodrome, various combinations of typical syndromes develop.

Skin syndrome. Purpura of the skin is the most prominent physical finding in HSP and is characterized by the following:

- It begins with a symmetrical erythematous macular rash on the lower extremities that quickly evolves into purpura. Rash may initially be confined to malleolar skin but usually extends to the dorsal surface of the legs, the buttocks, and the ulnar side of the arms. Within 12-24 hours, the macules evolve into purpuric lesions that are dusky red and 0.5-2 cmin diameter. The lesions may coalesce into larger plaques - ecchymoses. Thus, a typical hemorrhagic rash is a pathognomonic, obligatory symptom BSHG. In severe cases rash is complicated by central necrosis and covered with crusts.
- Rash is localized on the extensor surfaces of the lower extremities, around the joints, on the buttocks, on the lower trunk and perineum, rarelyonthe upper extremity, head and trunk.Rash does not disappear at pressing and leaves behind a long-continued pigmentation.Eruptions usually last 3 weeks.
- In some cases, angioneuroticedema occur in patients (usually on the face, hands, feet).

Joints syndrome. Involvement of the joints is the second characteristic symptom, observed in 2/3 of patients. It usually appears simultaneously with hemorrhagic rash on the 1st week of illness, or at a later date. The large joints(knees, ankles), and (less commonly) wrists are involved. **Periarticular** swelling (edema) of the joints with migrating joint painorsoreness is developed.Resistant deformation of the joints, warmth, erythema are not typical for HSP.

Abdominal syndrome caused by lesions of the gastrointestinal tract occurs in approximately two thirds of the total number of patients. Abdominal syndrome is manifested by spastic abdominal pain, nausea, vomiting, diarrhea with gross or occult blood. In addition to abdominal pain, GI findings can include the following: hemorrhagic or erosive duodenitis, intussusception, bowel infarction with or without perforation. Hemo- positive stool is the primary finding on GI examination.

Renalsyndrome. Renal involvement is present in 30-50% of patients and may persist as long as 6 months after the onset of rash.Renal involvement manifests a ranging from mild hematuria or proteinuria to oliguria and renal failure.

Idiopathic thrombocytopenic purpura (ITP) is defined as isolated thrombocytopenia with normal bone marrow and the absence of other causes of thrombocytopenia. ITP is a decrease in the number of circulating platelets in the absence of toxic exposure or a disease associated with a low platelet count.

Etiopathogenesis. Thrombocytopenia is developed due to platelet destruction by immune mechanisms. Antibodies to their own platelets may appear in 1-3 weeks after viral or bacterial infections, vaccinations, medication for their individual intolerance, hypothermia or insolation, after surgery, injury.Antigens (e.g., viruses, drugs, including vaccines) are deposited on the platelets and induce an immune response. Antiplatelet antibodies are predominantly IgG. Reactions "Ag-AT" occur on the platelet surface. Lifespan platelet is reduced to a few hours, instead of 7-10 days in normal. Premature death of platelets occurs in the spleen. Bleeding when ITP is caused by a decline in platelet count and secondary vascular damage due to angiotrophic platelet function impairment.

According to **clinical manifestations** two variants of thrombocytopenic purpura aredistinguish: a "dry" - the patient has only skin hemorrhagic syndrome, "wet»-haemorrhages in combination with bleeding. Pathognomonic symptoms of ITP are **haemorrhages** into the skin, mucous membranes and **bleeding.**

Typical signs of children illness are:

1. Abrupt onset (childhood ITP). Gradual onset (adult ITP)
2. Skin and subcutaneous hemorrhage, hemorrhage in the mucous membranes and natural cavity (chest, abdomen, joints). These hemorrhages is characterized by the following symptoms:

- spontaneity of appearance, mainly at night
- Polymorphism - nonpalpablepetechiae, ecchymosis, hemorrhagic bullae on mucous membranes, purpura
- Polychrome - hemorrhages on the skin of children of different colors - from red to bluish-green and yellow, a symptom of "leopard skin"
- asymmetric and chaotic arrangement without "favorite" locations.

3. Retinal hemorrhages. Evidence of intracranial hemorrhage, with possible neurologic symptoms
4. Bleeding, which appear as arbitrary, and after surgery (tooth extraction, tonsillectomy, etc.). The most frequent are bleeding from the nose (Epistaxis) and the mucous membrane of the mouth: gums, tongue, etc.(Gingival ble-eding), the gastro-intestinal (melena, etc.), hematuria (blood in urine),meno-and metrorrhagia (bleeding during menstruation).Spontaneous bleeding when platelet count is less than 20,000/mm ^{3}.
5. Nonpalpable spleen.

We can distinguish 1. acute**course**(lasting up to 6 months) and 2. Chronic(lasting more than 6 months) forms of the disease.

Laboratory parameters:

- thrombocytopenia,
- increased bleeding time,
- reducing blood clot retraction.

Treatment

1. Bed rest during the acute period of illness, diet - a high-energy, easily digestible, enriched with protein and potassium nutrition
2. High-dose parenteral glucocorticoids - prednisolone at a dose of 1-2 mg / kg for 2-4 weeks with subsequent dose reduction
3. Immunosuppressants are prescribed in the absence of glucocorticoid therapy effect - azathioprine, cyclophosphamide, vincristine
4. Antirhesus immunoglobulin - contributes to increase of the number of platelets
5. Interferon-alpha-2 reduces the production of antiplatelet antibodies
6. Platelet transfusion is indicated to control severe hemorrhage
7. Splenectomy is indicated for patients with life-threatening bleeding whom medical therapy fails to help.
8. Symptomatic treatment of hemorrhagic syndrome in children includes the application of hemostatic sponges, aminocaproic acid, adrokson, Dicynone.

Tests Topic 1.

1.What causative agent is rheumatism etiologicallyassociated with?

A. Blue pus bacillus.
B. Group A beta- hemolytic streptococcus.
C. Staphylococcus aureus.
D. Influenza virus A2.
E. Epidermal streptococcus.

2. What diseases are preceded rheumatism most often?

A. Quinsy.
B. Scarlet fever.
C. Erysipelas.
D. Influenza.
E. Quinsy, skarlatina, erysipelas.

3. Which group drugs are used most often for rheumatism treatment?

1. Macrolids.
2. Penicillins.
3. Cephalosporins.
4. Aminoglycosides.
5. Fluoroquinolones.

4. For which CHD skin cyanosis is the most characteristic feature?

A. coarctation of aorta
B. aortic stenosis
C. fibroelastosis of endocardium
D. Fallot's tetralogy

5. Clinical manifestations of CHD include all listed below except for:
A. cyanosis of skin
B. paleness of skin
C. hyperemia of skin
D. shortness of the breath

Task 1. A 3 –year-old boy suffer from constant cyanosis of the skin, physical developmentretardition. Shortness of breath on exertion, strain the fingers in the form of time windows, drum sticks are observed. Periodically, shortness of breath and cyanosis get increased. Percussion border of cardiac dullness shifted to the right, the II tone auscultation of the pulmonary artery is weakened, rough systolic murmur in the III-VI intercostal space to the left of the sternum is marked. X-ray shows increase the right heart enlargment. What is the diagnosis?

A. Mitral stenosis.
B. Mitral insufficiency.
C. Aortic stenosis.
D. Aortic failure.
E. Tetralogy of Fallot .

Topic 2. Tests

1. What causative agent is the most common etiological factor of pyelonephritis in children?
A. Escherichia coli.
B. Proteus.
C. Enterococcus.
D. Staphylococcus.
E. Clamydia.

2. What is the main way of kidney infection?
A. Hematogenic
B. Ascending.
C. Lymphogenic.

3. What complications of acute glomerulonephritis occur in childrenrarely?
A. Acute renal failure.
B. Angiospastic encephalopathy.
C. Heart failure.

4. What drugs are used for glomerulonephriticpathogenetic therapy?
A. Glucocorticoids.
B. Antihistamines.
C. Diuretics.

D. Hypotensive drugs.
E. Vitamins

5. What glomerulonephritis form is hypoproteinemia characteristic of?
A. acute glomerulonephritis with nephrotic syndrome.
B. acute glomerulonephritis with isolated urinary syndrome.
C. acute glomerulonephritis with nephritic syndrome

Task 1. An 8-year-old girl fell seriously sick: her body's temperature is 38,7° C, pain in the lumbar region, painfulurination. Examination data: intoxication signs, positive Pasternatsky's symptom on both sides, more marked on the left. What disease is more likely to develop?
A. Acute glomerulonephritis.
B. Influenza.
C. Acute cystitis.
D. Acute pyelonephritis.

Topic 3.Tests

1. Hemorrhagic syndrome in hemorrhagic vasculitis is characterized by:
A. Nasal bleeding
B. Symmetry hemorrhagic rash
C. The presence of hemarthrosis
D. The presence of hematoma

2. What blood indicators are changed in hemophilia?
A. The duration of bleeding
B. Blood clotting time
C. The adhesion of platelets
D. Prothrombin index

3. What factors are indicators of the severity of hemorrhagic vasculitis?

A. Reduction of blood clotting time

B. Increased alpha 1-acid glycoprotein

C. Reduction of alpha-2-gammaglobulin

D. Reducing the time of the plasma recalcification

4. Which indicators typical of thrombocytopenic purpuraare changed?

A. The duration of bleeding

B. Blood clotting time

C. The plasma recalcification

D. prothrombin time

Task 1.A 10-years-old child has prolonged massive bleeding for 6 hours after tooth extraction. Prior to that, the boy had experienced nasal bleedings and extended subcutaneous hematoma at blows.

A. Hemophilia,
B. Idiopathicthrombocytopenicpurpura
C. Hemorrhagicvasculitis

List of references

8. Pediatrics [Text] : textbook / O. Tiazhka [et al.] ; ed. O. Tiazhka ; Ministry of Public Health of Ukraine, National O. O. Bohomolets Medical University. - Vinnytsia : Nova knyha, 2011.
9. Nelson Textbook of Pediatrics, 2-Volume Set, 20th Edition
10. TEXTBOOK OF PROPEDEVTIC PEDIATRICS For students of II – III years**.** Kyiv 2006**.** Autors: O. Vinnitzka, T.Lutaj, A.Antoshkina, Ju. Piatnitzki, M. Vasiukova, N. Gorobetz, L.Martinova, T. Mellina, L.Slipachuk, O.Stroj, P.Tovmash
11. Pediatrics. Guidance aid for students of V year of study. Edited by Professor O. Tiazhka

MINISTRY OF PUBLIC HEALTH OF UKRAINE
O.BOHOMOLETSNATIONALMEDICALUNIVERSITY

PEDIATRIC DEPARTMENT N5

"Approved"
on the methodical conference
Department of Pediatrics № 5
protocol №2 from 07.09.2016

GUIDELINES
FOR STUDENTS

Academic discipline	Pediatrics
Study subject	Infectious diseases in children. Vaccination scheme.
Course	4
Faculty	Dental

Kyiv-2016

Relevance of the topic. Over the last decade, several epidemics including among children in Ukraine has passed,. The relevance of this topic is that infectious diseases are the leading positions in the structure of infant morbidity and mortality.

Study objective. To be able to diagnose a major clinical syndromes of childhood infections; to know the vaccination schedule.

Questions for self– checking

1. Rubella. Epidemiological features.
2. Scarlet fever. The modern view of scarlet fever as one of the forms streptococcal infection.
3. Etiology. Pathogenesis.
4. Features anti-toxic and anti-bacterial immunity.
5. Measles. Etiology. Epidemiological features. Clinical manifestations in the different periods of the disease. Treatment
6. The differential diagnosis of measles and rubella.
7. The prophylactic immunization calendar.

Rubella

Causes. Rubella and congenital rubella syndrome are caused by rubella virus

Postnatal rubella. Virus is transmitted from person to person via the aerosolized particles from the respiratory tract. The incubation is usually 14-21 days after exposure to a person with rubella. Prodromal symptoms are unusual in young children but are common in adolescents and adults. The following signs and symptoms usually appear 1-5 days before the onset of rash: **1. General symptoms** - Eye pain; Conjunctivitis; Sore throat; Headache; General body aches; Low-grade fever (it is usually not higher than 38.5°C); Chills; Anorexia; Nausea; 2. Tender **lymphadenopathy** (particularly posterior auricular and suboccipital lymph nodes); 3.Forchheimer**Rash -** The exanthem of rubella consists of a discrete rose-pink maculopapular rash ranging from 1-4 mm. The synonym "3-day measles" derives from the typical course of rubella exanthem that starts initially on the face and neck and spreads centrifugally to the trunk and extremities within 24 hours. It then begins to fade on the face on the second day and disappears throughout the body by the end of the third day. **Mouth -** The Forchheimer sign may still be present on the soft palate.

Congenital Rubella Syndrome. The classic triad presentation of congenital rubella syndrome consists of the following: **1.** Sensorineural hearing loss is the most common manifestation of congenital rubella syndrome; **2**. Ocular abnormalities including cataract, infantile glaucoma, and pigmentary retinopathy occur in approximately 43% of children with congenital rubella syndrome; **3.** Congenital heart disease including patent ductusarteriosus (PDA) and pulmonary artery stenosis is present in 50% of infants infected in the first 2 months' gestation. Other findings in congenital rubella syndrome include the following: CNS abnormalities, Hepato-splenomegaly, Jaundice, Hepatitis, Skin manifestations, including blueberry muffin spots that represent dermal erythropoiesis and dermatoglyphic abnormalities, Endocrine and Hematologic disorders

Medical Care

Postnatal rubella. Treatment is supportive. No specific antiviral agent for rubella is currently available. Antihistamines may be useful with uncomplicated rubella. For complicated cases,

treatment is as follows: For severe arthritis affecting weight-bearing joints, encourage rest. Nonsteroidal anti-inflammatory drugs (NSAIDs) may be helpful, but corticosteroids are not indicated.

Isolation of hospitalized patients. Droplet precautions and standard precautions are recommended for 7 days after the onset of rash in patients with postnatally acquired rubella infections.

Measles, also known as rubeola, is one of the most contagious infectious diseases, Measles is marked by prodromal fever, cough, coryza, conjunctivitis, and pathognomonic enanthem (ie, Koplik spots), followed by an erythematous maculopapular rash on the third to seventh day. Infection confers life-long immunity.

Maternal antibodies play a significant role in protection against infection in infants younger than 1 year and may interfere with live-attenuated measles vaccination.

Etiology. The cause of measles is the measles virus, a single-stranded, negative-sense enveloped RNA virus of the genus *Morbillivirus* within the family Paramyxoviridae. Humans are the natural hosts of the virus; no animal reservoirs are known to exist. This highly contagious virus is spread by coughing and sneezing via close personal contact or direct contact with secretions.

History

The **incubation period** from exposure to onset of measles symptoms ranges from 7 to 14 days (average, 10-12 days). Patients are contagious from 1-2 days before the onset of symptoms to 4 days after the onset of rash

This **prodromal phase** is marked by malaise, fever, anorexia, and the classic triad of conjunctivitis (see the image below), cough, and coryza. Other possible associated symptoms include photophobia, periorbital edema, conjunctivitis and myalgias.

The characteristic enanthem generally appears 2-4 days after the onset of the prodrome and lasts 3-5 days. Small spots (Koplik spots) can be seen inside the cheeks during this early stage (see the image below).

The exanthem usually appears 1-2 days after the appearance of Koplik spots; mild pruritus may be associated. On average, the rash develops about 14 days after exposure, starting on the face and upper neck (see the image below) and spreading to the extremities.

Complications. Common infectious complications include otitis media, interstitial pneumonitis, bronchopneumonia, croup, encephalomyelitis, diarrhea, sinusitis, stomatitis, subclinical hepatitis, lymphadenitis, and keratitis, which can lead to blindness.

Treatment

Drugs for specific treatment for measles have not been developed.

Symptomatic treatments include expectorants, mucolytics, antiinflammatory sprays facilitate airways.

Pediatric Scarlet Fever

Causes. Scarlet fever results from an erythrogenic toxin produced by group A streptococci

History. Scarlet fever is characterized by the following findings: **1**.Headache; Vomiting; Abdominal pain; Fever; Flushed face with perioral pallor is observed; **2**. Sore throat. Tonsils are edematous, erythematous, and covered with a yellow, gray, or white exudate. Petechiae are noted on the soft palate. **3**. Tender anterior cervical lymphadenopathy may be present. **4.** Rash appears 1-2 days after onset of illness, first on the neck and then extending to the trunk and extremities. Exanthem texture is usually of coarse sandpaper, and the erythema blanches with pressure. A few days following generalization of the rash, it becomes more intense along skin folds and produces lines of confluent petechiae known as the Pastia sign. These lines are caused by increased capillary fragility. The rash begins to fade 3-4 days after onset, and the desquamation phase begins. This phase begins with flakes peeling from the face. Peeling from the palms and around the fingers occurs about a week later and can last up to a month.**5**.Appearance of the tongue consists of the following: During the first 2 days of the disease, the tongue has a white coat through which the red and edematous papillae project. This is referred to as a white strawberry tongue. After 2 days, the tongue also desquamates, resulting in a red tongue with prominent papillae called the red strawberry tongue.

Laboratory Studies

The following studies are indicated in scarlet fever:

- Throat culture or rapid streptococcal test
- Anti-deoxyribonuclease B and antistreptolysin-O titers (antibodies to streptococcal extracellular products)

Treatment.

Treat patients who have scarlet fever with a standard 10-day course of oral penicillin or erythromycin. Antipyretic drugs, vitamins are also used.

Theprophylacticimmunizationcalendar

(The Order of the Ministry of Public Health N. 276 from 31.10.2000 "About the procedure of prophylactic immunization taking in Ukraine").

The list of infections	*The term of vaccination start*	***The term of revaccination***					***The notes***
		I	II	III	IV	**V**	
Against tuberculosis	3 – 5 day of life	7 years	14 years				Vaccination and revaccination is taking disposably. Children with a negative Mantoux test are subjected to revaccination in 7 and 14 years
Against poliomyelitis	3 mon., 3 times	18 mon.	3 years	6 years	14 years		Vaccinations are taking 3 times with an interval for 1

							month among each time
Against pertussis, diphtheria and tetanus (APDT)	3 mon., 3 times	18 mon. – APDT					Immunization is taking by APDT-vaccine 3 times with an interval for 1 month among each vaccination in the dose of 0.5 ml. APDT-vaccine is used till 3 years 11 months and 29 days
Against diphtheria and tetanus			6 years - ADT	11 years – ADT-m	14 years – ADT-m	18 years – ADT-m	The second vaccination is taken by ADT-anatoxin in the dose of 0.5 ml. The third and all other revaccinations are taken by ADTM-anatoxin
Against rubeola	12 – 15 mon.	6 years					Vaccination and revaccination is taking disposably.
Against parotiditis	12 – 15 mon.	15 years – boys					
Against rubella	12 – 15 mon.	15 years – girls					
Against hepatits B	0 (12 hours of life), 3 mon., 5 mon.						Vaccine against hepatitis is prescribed to newborns in the dose of 0.5 ml

Tests

1. What is a BCG?

A. Live attenuated vaccine of mycobacteria tuberculosis' bull strain.
B. Killed vaccine of mycobacteria tuberculosis' human strain.
C. Live vaccine of mycobacteria tuberculosis' human strain.

2. What established term of vaccination against tuberculosis is?

A. 1 year
B. 3 – 5 days
C. 5 years
D. 15 – 17 days

3. What established term of vaccination against diphtheria, pertussis, tetanus, poliomyelitis is in Ukraine?

A. 1 month
B. 3 month
C. 6 month
D. 4 month

4. What is II revaccination against diphtheria and tetanus taken by?

A. By APDT-vaccine
B. By ADT-anatoxin
C. By ADT-M anatoxin

Task 1. Girl 5 years old acutely got sick: a rise in body temperature to 38 ° C, cough, runny nose. In the next three days the temperature was kept, catarrhal phenomena have intensified. There were cough, swelling of the eyelids, photophobia. Maculopapular rash on the face behind the ears has appeared on 4 day and then a rash in the next 2 days has spread over the trunk and limbs. Then the temperature dropped, the condition improved. What is the diagnosis?

A. Rubella
B. Rubeola
C. Pediatric Scarlet Fever
D. Allergic rash

List of references

12. Pediatrics [Text] : textbook / O. Tiazhka [et al.] ; ed. O. Tiazhka ; Ministry of Public Health of Ukraine, National O. O. Bohomolets Medical University. - Vinnytsia : Nova knyha, 2011.
13. Nelson Textbook of Pediatrics, 2-Volume Set, 20th Edition
14. TEXTBOOK OF PROPEDEVTIC PEDIATRICS For students of II – III years**.** Kyiv 2006**.** Autors: O. Vinnitzka, T.Lutaj, A.Antoshkina, Ju. Piatnitzki, M. Vasiukova, N. Gorobetz, L.Martinova, T. Mellina, L.Slipachuk, O.Stroj, P.Tovmash
15. Pediatrics. Guidance aid for students of V year of study. Edited by Professor O. Tiazhka