HEALTH MINISTRY OF UKRAINE

ODESSA NATIONAL MEDICAL UNIVERSITY

Department of Propaedeutics of Pediatrics

I APPROVE

Vice-rector for scientific and pedagogical work

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" ___ " _____ 2022

METHODICAL RECOMMENDATIONS FOR LECTURES

Course 3 Faculty international

Academic discipline "Propaedeutics of Pediatrics"

The program was discussed

at the Department of Propaedeutics of Pediatrics board

Protocol № 1 from 30.08.2022

The Head of the Department _____ Olena STARETS

Developers: O. Starets, professor, MD, PhD; O. Fedorenko, assoc.prof., MD, PhD; T. Khimenko, assist., MD, PhD, M. Dubkovska, assist., MD, PhD; S. Chernysh, assist., MD.

Lecture № 1

Topic: “Pediatrics as a science about the healthy and sick child, its place in general medicine system. The basic historical stages of pediatrics in Ukraine. Principles of the organization and methods of the treatment-and-prophylactic help to children in Ukraine. The children age's periods and their characteristic and features”

The actuality of the topic: professional training of the doctor should be based on sources of the organization of medical aid to children in Ukraine. Each student should know about pediatrics development in Ukraine. Development stages are many-sided - from rural hospital to well-organized specialized hospitals.

The history of development of pediatrics in Ukraine influences on the formation of an active position of the doctor and motivation of studying of a theme.

Aims of the lecture

1.1. Practical (training) aims:

- to acquaint students with concept "Pediatrics" and its place of preparation of the future doctor. To acquaint students with history of development and coryphaeuses of the Ukrainian pediatrics, scientists-pediatrists of Ukraine, Odessa, the scientists-pediatrists, working on chair, their scientific achievements.
- To acquaint with the basic methods of the organization of treatment and prophylactic establishments.
- To acquaint students with the periods of children's age, laws of physical, psychological development of the child. Value of exogenous and internal causes in a case rate of children, feature of age pathology.

1.2. Educational aims:

- To show value of history of pediatrics in formation of a public position, feeling of patriotism, internationalism in the future doctors. On concrete examples to show that child care is the most important state problem.
- To bring up in students a modern clinical view in section of medicine and "Pediatrics" problems.

Plan and structure of the lecture :

№№	**The main steps of the lesson**	**The aims in the levels of learning of the material**	**Materials of methodical software**	**Time (in minutes or %) of a hole lesson**
1	**2**	**3**	**4**	**5**
I.	*The preparatory level*	1	The presentation, Auditory	5%
1.	The defining the purpose			

2.	Formation of the positive motivation			
II.	*The basic level.*	*II*.		85-90%
3.	The presentation of the lecture by plan 1. Pediatrics as a science about the healthy and sick child, its place in general medicine system. 2. The basic historical stages of pediatrics in Ukraine. 3. Principles of the organization and methods of the treatment-and-prophylactic help to children in Ukraine. 4. The children age's periods and their characteristic and features.	*II*. *II*. *1*. *II*.	Illustrations	
III.	Final level.		Literature, questions, tasks.	5%
4.	The conclusion of the lecture.			
5.	Answering questions.			
6.	The task for self-control			

The content of the lecture

Pediatrics (from Greek words, “pais” – child and “iatreia” – curation) studies the laws of the child’s growth, reasons and mechanism of disease formation, types of their determination, curation and prophylaxis. Thus, it studies the growth period, the most important of the life’s lustres. Being a pediatrician needs a great deal of humanism and a sense of responsibility.

The subject of pediatrics is a very important branch of science. All around the world three children are born in this world every moment. They are our future. We are all responsible of their health and well-being.

Any occurring inequity injures children primarily. Due to international women democratic federation and the other progressive world foundations the day of November 20 has been adopted as child rights declaration day, which strictly determines the basic rights of the children of the world.

Teaching of pediatrics in the medical faculty commences in the second year of studying. In fact, that is the first chair that gives a sort of practical skill to the students. Each period of maturation has it own morphological, physiological send psychological properties. That why the knowledge of clinical anatomy and physiology is of great importance for evaluation of organism/systems injuries and during different periods of nutrition.

Semiotics of different systems injuries is being studied here too detailed studying of diseases and their nosology is the topic of basic pediatrics and well examine them in a general way. Diagnosis and symptoms are being studied in two enlarged generalized aspects that are based on age norm and examination method. Its aim is pathology consultation. On the other side is the diagnosis of symptoms i.e. path physiological binding of disease symptoms and thus discovering functional decomposition of certain physiological system.

Well study the basic nursing and medical manipulation skills, thus practicing the entire skills do a nurse.

Latter pediatric teaching will be performed at other chairs, like children's infection, children's surgery etc…

Your further skills and success is determined by your studying order here at the university. The knowledge here becomes the basis for further improvement, which is limitless. That includes examining scientific literature, pediatric medical issues, manuals and monographies. Advanced pediatric improvement depends on your participation at the regional council for pediatrics. Such councils exist all over Ukraine. Being its member is pride and an honor for any doctor.

Pediatrics is not a simple field of science but a name for a doctor's activity in the Health Protection System. Pediatricians make real achievements of harmonic development of medical science and provides proper development of children, diagnosing, duration and prophylaxis. their working places are children's institutions (kindergartens, schools, orphanages, sport camps, polyclinics, a and children's hospitals- general and specialized, pediatric first aid stations, infant centers of obstetric and maternity homes, different consultation foundations and children's sanatoriums.

Studying in a Ukrainian Medical University, the student will gain the knowledge of the Ukrainian pediatric science history. It began its existence in the 19th century. The founder of the first Ukrainian pediatric hospital was Vladimir Arkadyevich Frankovsky. It was founded in Kharkov, on September 22, 1878 and had 20 beds.

V.E. Chernov the head of an independent pediatric chair was one of the pioneers of Ukrainian pediatrics. His scientific activities were connected to many cities- Kiev, Kharkov, and Dnepropetrovsk. He was the inventor of the method "protein determination by niter input in organism". He also voted for the adoption of the Mercies therapy in the treatment of 'esophagus narrowed by scars', which he himself experienced. He used the laboratory methods of experimentation in practice and greatly improved the theoretical tutorial pediatric

terms, used in the Kiev University, thus adopting the pediatric teaching on clinical observations.

As we can see, he was the one to create the promises for Ukrainian pediatric school development

The first pediatric chair was founded in Kharkov medical university on 22 January 1892.its founder was M.D Ponomaryov, the private docent of the chair for Feminine, Obstetrics and Infantile diseases. He was he first to adopt the clinical experiment, thus basing pediatrics on scientific grounds.

V.F. Yakubovitch was the first head of children's diseases clinic and the chair in our city. He dedicated his researches to children's infection diagnosis, clinical work organizations, and children's diseases studying course details. His monograph "Manual on children's diseases diagnosis and the ways of their examination with children" was useful for both students and doctors.

Professor O.M Chohol, a member of the scientific academy had participated actively in the Ukrainian pediatric development. The scientific exploration she carried out in the Kiev medical university, were dedicated to etiology, clinics and treatment of acute gastro-intestinal diseases with green aged children, problems of children's rheumatism and cardiovascular pathology, curation and prophylactics of pneumonia, meningitis, tuberculosis, post meningitis rehabilitation, curation at sanatoriums. She and her colleagues began working on the formulas for nutrition mixtures for infants.

Professor V.O. Belousov was the perfect mentor and doctor, who has been at the head of the chair since 1944 till 1965, he published a textbook "Children's diseases" and many other manuals and, monographs. As his scientist ancestors, he endeavored to solve the problems that ere practically acute. Thus he explored the problem of meningeal tuberculosis treatment, diagnosis and treatment of rheumatism and cardio-vascular insufficiency.

In 1995 the scientific community of Kharkov celebrated the 100th anniversary of his birth and honored his memory, of that of a outstanding professor, a member of the scientific academy and a celebrated scientist of the Ukraine

F.D. Rumyantsev continued the work of the Kiev pediatrics i.e. their study of the children's infectious pathology, thus he studied the diagnosis and treatment of scarlet fever, diphtheria, measles etc... His pedagogic activity in Kiev Medical University lasted for 30 years and was successful enough.

L.L. Finkelstein participated in Ukrainian pediatric science creation too. His piercing mind of an analytician enabled him to be an innovator scientist. His restless fancy endeavored in efforts to find the new trends of science.

Arakdiy Ivanovich Scorsky devoted 46 years of his labors to Odessa pediatric clinic and since 1928 to 1954 was its irreplaceable head, this way having turned it into an outstanding medical foundation, equipped with clinical, biochemical and bacteriological laboratories, X-rays unit, ECG, physiotherapeutic services, an ambulatory and a library. He created children's balneological treatment centre for children suffering from rheumatism, rheumatoid arthritis, poliomyelitis, and other diseases. His scientific researches were dedicated to problems of diphtheria, typhus, Siberian ulcer, tetanus, meningitis, tuberculosis, chronic nutrition and digestive impairments in newborns. Some of his researches were dedicated to theoretical pediatrics. Hid monographs concerning tuberculosis, infections and health resorts are considered classics. Since 1928 to the end of his life, he was the head of Odessa pediatric

community and Odessa regional pediatric service.20 candidate and doctor researches were carried out under his observation and inspiration.

His industrious nature, enduring character, his irreproachable skills as a diagnoser, and his strong will to help people in trouble made him their hero. Thus, he deserves all the love and gratitude of the people. More than 50 years have passed since his death, but still people call the clinic, where he worked as the "Skrotsky Clinic". His library, which contains numerous tomes, was dedicated to the clinic and his summer cottage was given to the local orphanage-all according to his will.

Ivan Michaylovich Rudneff became the head of the pediatric chair of Kiev Medical Institute in 1963. Before that, he had been the head of Lvov Medical Institute Pediatric Chair was founded by him. He and his colleagues were researching the problem of rheumatism. He paid a lot of attention to the diagnosis of rheumatisms and the other cardiopathies in children.

Latter he studied the problem of children allergic diseases. He was the first in Ukraine to work out a system for bronchial asthma in children suffering from hypersensitivity to bacterial antigens. The method proved to be effective in cases of chronic and severe diseases.

He is the author of the following monographs:

– "Diagnosis and treatment of rheumatism in children" – 1964

– "Practical cardiology" – 1969

– "Treatment of children by glucocorticoids" – 1969

He inspired the idea and was one of the chief editors of the textbook: children's diseases. The second edition of the textbook received the state awards, but the author received it post-mortally. He fathered to 74 scientific research works, he was known for the novation of problems and preciseness of solutions.

P.M. Gudzenko became the heads of the chair in 1965.for 25 years he would also be the chief pediatrician of Ukraine. He was the chief editor of "Pediatric, obstetrics and gynecology" magazine for many years. His personal scientific ambitions were amid the problems of the digestive tract diseases, staphylococcal infection and immune system. His researches on diagnosis of notorious diseases are of a great value to the scientific world. He originated the system of the peculiarities of staphylococcal infection at different age groups and their treatment with staphylococcal anti-toxin. The dosages of the preparations were greatly reduced according to his advice. The innovation turned to be of great positive effect .His scientific research considered problems of meningitis tuberculosis too. He also worked out the problem of children's feeding at different age groups. He baby milk mix he worked out was used for the infants feeding for many years successfully.

He was the author of 153 scientific researches and the chief editor of "Children's Diseases", thus his work received a Ukrainian state award in 1979

His basic monographies are:

– "Children's primary Pyelonephritis"

– "Tuberculosis in children" -1969

– "Pediatric receipt manual"

There is a certain system of foundations aimed a to protect the health of Ukrainian children .Heir components are women consultations, maternity hospitals, children's polyclinics and hospitals ,kindergartens, crèches, sanatoria's, relaxation camps, etc.. The mentioned structures are tightly bound with each other and form a net, providing the full performance of curative and prophylactic actions.

Pregnant women attend special consultations, countryside hospitals and obstetric nurses of special foundations.

At the first half term of the pregnancy, the women should attend the doctor at least once per month; once or twice in the second term and once a week before delivery of the child. The woman is given an appointment in the maternity hospital a short time before the delivery. The maternity hospital consists of many departments, like for the normal infants, for physiological birth giving and for women with fever. The acceptance block consists of a hall, a filtering foundation, an observation room, and sanitary control room.

The newborn is kept in a special department, where a specially trained doctor keeps a watch over them. Before being placed into specific department, each new born under goes an anthropological examination and the data collected. The newborn spends two hours on a heat table. To prevent the infectious disease the filling of infant department room should be cyclic.

To heal the ill newborns the special departments of neonatal path physiology are created now. The time when he newborn leaves his department depends on the state of his health and the state of his mother. Healthy infants spend 6-7 days in it.

When the infant leaves the maternity hospital, his data is transferred to the district children polyclinic, where the chief nurse accepts it and writes it down in the registration ledger. Since that moment, the child is under observation of the district pediatrician. If some hazardous factor does exist, then the doctor should visit the new born the next day of his arrival at home. Later the doctor visits the infant three times during the first month (he nurses perform their rounds not least than 6 times the first month). At large during the fist life year of the healthy child the doctor visits him not least than 13 times. The same number for the nurse is 14.

The foundation hat controls the notorious problems is children's polyclinic. Its work is based on territorial placement. Since the time the child leaves maternity hospital and until the day, his documents are being transferred to the teenage heath service; his case belongs to the same pediatrician. Each district of pediatric service should consist of not more that 800 children and have 1 doctor post and 1.5 posts for nurses.

Each 8 pediatric districts are united into one department headed by a chief doctor. His duties are as follows:

– prophylactic work
– anti-epidemic work
– treatment activity

Prophylactics occupies 80 % of a district doctor daily duties and consist dispensary children observation, preparing them to pre-scholar foundations and schools admittance, anti-epidemic affairs organizing and educative work with parents.

Anti-epidemic work consists of the following:

– calculation of children that ought to undergo vacation
– planning of vaccination
– home visits of infectious patients
– observation of recovering patient

The work of a district pediatrician consists of polyclinic work and daily rounds.

The doctor at the polyclinic should examine 5 patients in each hour(thus 3 hours account for 15 children)he should dedicate 1 hour to daily rounds and for 1.5 visit once per week is dedicate to the prophylactic work with healthy children.

Each new-born has his personal case in polyclinics registry. Any visit to the doctor claims a corresponding mark in the case history. Case history is the main medical document of the patient. It is being kept in the registry.

A doctor and a nurse carry out polyclinic observation. Daily round work means visiting the ill child on the day, his appointment originates from his parents, treating him, watching his recovery or admitting him to the hospital if needed.

Moreover, district doctors visit healthy children everyday, visit the ill children even when not being called by the parents. At the weekends and holidays, ill children are visited by the pediatrician on duty.

During the visit, the doctor observes the child in the presence of the parents and relatives. All the data he collects, his suppositions and advices ate written down in the case history.

Children's hospitals treat children until the age of 18. The hospital consists of:

1. Acceptance department and children department of various specializations, including wards for ill neonates.
2. Department of treatment, prophylactics and laboratories.
3. Department of pathological anatomy.

Other structural components: drugstore, kitchen, medical statistics room, library, storeroom, etc.

The most advanced structure of acceptance in departments is Meltzer's boxes.

When the child comes to the hospital, he should have an assignment from his district doctor with a determined diagnosis and a note assuring that he is not infectious. Diagnosis is to be approved at the admitting department, the child is prescribed a proper treatment course and diet, and the sanitary actions are to be performed there too.

After the child has come to the department, the proper room is to be prepared for him. Each bed should be not less that 6 square meters of area. All departments should have large and small beds rooms. That enables the doctor to group the patients according to their age, sex and the disease form. Rooms are to be filled in an empathic way.

Sometimes children are to be isolated before the type of his disease is determined. Newcomer children are not to being put into the same room with the ones(recoverig), who are going to a children polyclinic that had sent the child to a hospital.

Before the child leaves the hospital, his data is being put into an epicrisis, where treatment is detailed and advises for further medical actions are given. An epicrisis is being given to a children polyclinic that had sent the child to a hospital.

There is a widely developed system of children sanatoria:

1. For rheumatic children.
2. For children with diseases of respiratory organs (not tuberculosis).
3. For children with kidney diseases.
4. For children with skin diseases.
5. For the children with diseases of Psychoneurotic type.
6. For children with diseases of gastro-intestinal tract.
7. For injured by tuberculosis of bones and joints and other forms of tuberculosis.
8. For children with impairments of skeleton and muscles.

Special sanatoria with an educative course exist for ill children who need education due to their age.

The most widely met health support foundations are camps for schoolchildren. Moreover, special orphanages exist. Orphans, forlorn children and some other are being kept there.

Quality of medical control is determined by certain indices. One of them is infantile mortality i.e. dead children who did not reach the age of one year, out of total number of children, being born alive this year. That index is to be cultivated for a thousand children born alive. Infantile mortality index contain 3 more indices that indicate the mortality rate of the different age groups of children:

1. Early neonatal mortality rate – is the ration of children aged 0-6days who die to thousand born alive per year
2. Late neonatal mortality rate- is the ration of children aged 7-28days who die to thousand born alive per year
3. Post-neonatal mortality rate- is the ration of children aged 29days - one year who die to thousand born alive per year

The important index reflecting the medical control of pregnant and newborn is the internationally adopted prenatal mortality index. It is a ratio of born dead and dead at the first 6 days of life to the thousand of all born (dead and alive).

Mortality rate of children had reduced at about a dozen times during the years of Soviet power. The main causes of it are as follows: the most important is newborn's disease, than go respiratory organs diseases, congenital impairments, etc. the main death reasons of children elder than 1 year are traumatism, more seldom- oncology and infectious diseases.

One more important index of medical aid efficiency is sick rate.

Sick rate is the number of once again occurred diseases or other cases if one and the same group that occur per year. Due to the efforts of government and the prophylactic bound of medical activity sick rate decreases every year.

Recently decades have a strong feature for disease character change. Allergic rate increases greatly, the same happens with gastro-intestinal diseases, diseases of the urinary system and congenital diseases.

The important index that characterizes diseases of a certain type and medical aid efficiency is lethality rate that is the amount of deaths to 100 of certain disease cases. Different pathologies lethality rate decreases greatly in recent years. Most infectious diseases occurring with children aged over one year have no lethal outcomes.

The most important task now is to decrease the lethality rate in children in the first year of life and especially in newborn. This should become the basic bound of medicine, especially obstetrics and pediatrics. The means that can be taken prophylactic performance rate increase, especially of children in the risk group, improvement of treatment and diagnosis levels in polyclinics and hospitals and improvement of bonds between these structures.

Rehabilitative treatment is of great importance now. Great attention should be paid to disease prevention at schools and pre-scholar foundations, improvement of dispensary system for healthy and sick children. The mentioned means are the basement for health improvement of a new generation.

2. Children are different from grown ups by many features in morphology and organs and systems functioning.

In practice while dealing both with healthy and sick children you have to pay attention to these peculiarities. Peculiarities of child anatomy and physiology determine the specialties of children's pathology depending on child's age. Thus first we have to pay attention to the periods the green age is divided into and characterize them.

There are two periods:

1. Intrauterine
2. Extrauterine period.

The intrauterine period lasts for 270 days; 9 months since fertilization or 280 days since the last menstruation first day.

There are two stages of this period:

1. The phase of embryonic development (before II – III month)
2. The phase of placental development (after II – III month up to birth).

There are next periods in intrauterine development:

1. Germinal period. It begins since the ovum fertilization and lasts till the moment of fertilized ovum penetration into the uterine mucous coat. The whole period lasts for a week.
2. Implantation period. It lasts for 40 hours approximately, or 2 days.
3. Embrional period lasts for 5-6 weeks. Its basic feature is the creation and organogenesis almost of all inner organs of the future baby.
4. Neofetal or embriofetal period lasts for 2 weeks. That is the time of placenta formation .That is the process of great importance because placental blood circulation determines further embryo's development.
5. Fetal period lasts from 9 weeks till the birth. It consists of two periods- early and late.

Early fetal period (9th week-28th week) is characterized by the intensive growth and differentiation of embryo's tissues.

Late fetal period begins with the 28th week of pregnancy and lasts till the perifetal liquid outflow time i.e. birth giving time start. Late fetal period is followed by the intranatal one that lasts since the regular birth giving efforts commence till the moment of umbilical cord fixation (2-4 hours - 15-18 hours).

Early stages of fetus formation have their special periods of increased sensitivity to alterative agents, so called critical periods.

The stage before the implantation is called the first embryogenesis critical period, i.e. the ovum is especially sensitive to different menacious factors and actions. That period is characterized by the law of "all or nothing" .If the dose of altering agent is great the ovocyte perishes - the process is called the abortion. If the altering factor influences the organism during the other development periods, deformities may occur. The organs injured by them are organs of sight, central neural system and others.

Different deformities may appear at the 9-10th weeks of pregnancy. Their representatives are hypotrophies and different enzyme systems insufficiency.

Nowadays teratogenic factors are being divided into three groups:

- exogenic
- genetic
- exogenic plus genetic

Exogenic factors consist of:

- physical
- chemical (including remedies)
- biologic

Literature mentions infectious diseases of pregnant influencing fetuses. Such diseases are the ones induced by toga viruses, virus of grippe (30% of deformities), Botkin's disease, brucellosis.

Toxicosis in the second half of pregnancy, hypertensive diseases, hyperthyreosis and diabetes have a bad influence on the fetus.

Remedies and toxic agents penetrate placenta and influence the fetus. These agents are opium, morphine, chloralgidrate, chininum, luminalum and others. The problems of their influence are to be paid special attention to.

A, B, C, D vitamins run through the placenta to the fetus quickly too. The same goes with penicillin, streptomycin, PASA, phtivazide and insulin. The exceeding contains of A and D vitamins may influence the fetus badly.

Literature indicates the negative influence of “bad customs” such as smoking and drunkardship of mother. That is why propaganda and sanitary enlightening is of great importance now.

Exogenic factors include ionizing radiation too. Genetic factors are mutant genes that cause development deformities: family cases of upper lip split, Down's disease and deformities incompatible with life.

After the umbilical cord is cut the **extrauterine period** begins. It starts with a neonatal period. There are early and late neonatal periods.

Early neonatal period lasts since the moment of umbilical cord cut till the 7th day of life.

Late neonatal period lasts since the 8th life day till the 28th one. The length of the periods is determined by the newborns adaptation to the out-womb conditions time.

This period is characterized by some significant morphologic, functional and biochemical changes. Lung respiration begins, small and large blood circulation cycles start functioning, umbilical vessels get emptied, independent, though insufficient thermoregulation forms. This period is characterized by organs and systems imperfectness.

The baby seems to be helpless- his head hangs, arms and legs move chaotically; extremities are in a hypertonic state. Children are born with some unconditional reflexes (sucking, throwing, trunk etc.).They stay asleep all the time but the feeding. Water metabolism, neural and endocrine regulations are insufficient that's why plasma osmotic pressure is unstable and the babies dehydrate easily. Thus the systems of organism are in a state of unstable equilibrium.

At this period semi-physiologic and semi-pathologic states may occur − erythema of newborns, physiologic jaundice, physiologic mastitis, transitory fever, body weight loss, sexual crisis, albuminuria.

Diseases of neonatal period have their own peculiarities. That is the periods when such diseases as congenital lues, tuberculosis, malaria, viral hepatitis, toxoplasmosis, congenital deformities (organs development impairments), birth traumas, impairments of cerebral blood circulation, hemolytic disease, results of intra-uterine asphyxia, septic diseases(mostly of coccous nature) show up.

The period of infancy begins from the 29th day of life and finishes at 12 months. It is characterized by the rapid physical and psychical development. Main adaptive processes are over; the mechanism of breast feeding is over too. Baby’s length increase by 25 cm, his head circumference increase by 12 cm and the chest by 13-15 cm. Babies body proportions change greatly thus approaching the ones of a grown up(extremities length increases greatly, head and body length increase doesn't reach the same grade). Static functions develop: the baby manages to keep his head up by 2 months, at 7 months he sits down by himself. Decidual teeth start appearing since 5-6 months, their number reaches 8 by 1 year.

Psychic development of the child of notorious age is rapid. His sight fixes to bright objects at 3 months, he smiles by the end of 2 month, and mumbles emotionally since 3-4 months, recognizes his parents, since 6 months he manages to utter the syllables like "ba",

"ma", "pa". By the end of the first year he utters single words and understands the words of prohibition.

Unbalanced nutrition with certain components deficiency may cause diseases like anemia, rachitis or hypotrophy. Relative immaturity of gastro-intestinal tract function is the premise of gastro-intestinal diseases.

Anatomy-physiologic peculiarities of infant's respiratory organs make premises for bronchiolitis and pneumonia's with severe occurrence.

The first 3-4 months of life are characterized by the resistance to numerous infectious diseases (measles, scarlet fever, diphtheria, smallpox, etc.) due to the passive immunity that was obtained transplacentaly.

At the same time the baby is sensitive to alien micro-organisms and especially to intestinal flora. He is inclined to inflammation process generalization and general response to any influence. Thus in case of some diseases (pneumonia, flu, dysentery) convulsions, meningeal signs and toxicosis may occur.

Pre-pre-school or toddler period (1 year-3 years, cache period). This period is characterized by physical development speed decrease and bigger grade of physiologic systems maturity. Muscular mass increases rapidly. Decidual teeth have to appear all by the end of the second year.

The baby is agile and curious, he examines his surroundings and his main development form is a game, thus he obtains some complicated skills. His vocabulary volume increases. One should organize the regimen properly at this period not to overload the kid with impressions and protect him from negative emotions. The motor activity of the kid is great thus the hazard of accidental injuries increases; aspiration of alien particles and accidental poisonings are possible.

Due to contraction rate increase and immunity system immaturity, acute respiratory diseases, acute children's infectious diseases occur very often at this period. Allergic states may form at this period.

Pre-school period (3-7 years).

This period is characterized by physiologic growth increase, body weight increase and extremities growth speed inhibition. Decidual teeth fall away and permanent ones appear. Intellect develops intensively, labor activity becomes more complicated. The kid speaks fluently; his precise cardinal movements (writing and drawing) improve.

The most characteristic diseases of this period are infectious ones, the diseases of respiratory organs and diatheses.

The peculiarities of diseases are the decrease of process generalizing and toxic reactions.

Early school period (7-11 years). Decidual teeth are replaced by permanent ones, sexual dimorphism of physical development start to show up (growth and stature).Movements, co-ordination and memory improve, and the kids get interested in various new subjects.

The characteristic impairments are eyesight and bearing defects, caries, infectious diseases, possible rheumatism and other cardiac diseases, allergic diseases. Children may become more obese.

Elder school age (12 - 18 years) is characterized by the significant reorganization of endocrine system. That is a period of rapid sexual maturation for girls and its initiating period for males.

Feminine secondary sexual signs develop sooner than the masculine ones (some 1-1.5 years of exceed). That is a hard period of psychological development. Such features as will,

consciousness, morale form. The treating of parents and friends undergoes reformation too. This period is characterized by extreme opinions and extreme actions.

The diseases of the period are the same as that of the adults. Functional disorders of cardio-vascular, nervous system occur. They are caused by bodies and separate organ's non-proportional growth and the vegetative-endocrine system unstableness. Impairments of physical and sexual development may occur. Vascular tonus impairments may become severe. Gastro-intestinal tract diseases are widely spread (gastritis, duodenitis and ulcer disease).

Each kid has his individual speed of biologic development that may be something different from the ones of his contemporaries. To evaluate the biologic age of a kid one should take into consideration the indexes that reflect the biologic maturation process rate. The ones for the infants are appearance and vanishing of newborn's reflexes and further formation of motor customs, growth of decidual teeth. The pre-school period kids maturity rate is to be evaluated according to permanent teeth appearing, school age – development of secondary sexual signs. The special examinations determine the biologic age according to X-ray examination of ossification zones.

A kid is not a small adult, his growth and formation obeys different laws that direct him in a proper bound. His growth and development undergo constant quantity and quality changes. Of course in some respect his development depends on genetic material he had inherited from his parents.

The conditions created by the adult surroundings of the kid are important too. lt is useful to manipulate with scientific data at each period of age to make it contemporary and maximal.

The development of kids at the first months of life is much alike: mumbling, smiling at 2 months, muttering at 6-8 months, words uttering at 1 year But the further development of kids is different: thus the vocabulary volume of 3 years kids is 300-1200. Why so?

Psychologists think that intellectual abilities form at the youngest age (1-15 years), the sensitivity to the outer world is maximal till 4 years, and later that ability grows less.

Psychologists say that a kid sees and hears more than he'd wished too. Information flow causes the need for further information. But the growing kid has his own limits of mastering. The excess of this limit causes overload and neuroses.

A.S.Makarenko said "love needs dosage both as chinine and food. No one’s able to devour 6 kilos of bread at once and be proud of his nice snack. Thus love needs its dosage and measure too.

Children are incredibly sensitive and guess the real feelings of their surroundings. That is why communication with kids should be extremely sincere.

L. Tolstoy wrote in his "Anna Karenina" – "Even the wisest may be cheated by pretence, but the most obtuse kid reckons the utmost hidden pretence and turns away".

Materials to activate the students during lecture:

Questions :

1. Pediatrics as a science about the healthy and sick child, its place in general medicine system.
2. The basic historical stages of pediatrics in Ukraine.
3. Principles of the organization and methods of the treatment-and-prophylactic help to children in Ukraine.

4. The children age's periods and their characteristic and features.

Materials of methodical software of the lecture:

- Auditory
- Software
- Illustrations

The materials for self study of students. (Literature for the student.)

a) For the topic of the current lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 13 –24; 68 – 74.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

b) For the topic of the next lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 88 – 106.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

Literature used by lector during preparation the lecture:

1. Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – 808 pp.
2. Partha,s Fundamentals of Pediatrics. Ajanta offset &Packagings Ltd., New Delhi.-2013.- 782 pp.
3. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.
4. Vicky R. Bowden, Cindy S. Greenberg. Pediatric nursing procedures. - Lippincott Williams & Wilkins. - 2011. - 822 pp.
5. Pediatric Nursing Procedures. Vicky R. Bowden, Cindy S. Greenberg. - Wolters Kluwer Health.- 2015 -728pp.
6. Nelson. Essentials of Pediatrics. Sixth Edition. Canada: 2011. - 831p.
7. Robert M. Kliegman. Nelson Textbook of Pediatrics 19th ed. – W.B. Saunders Company, 2011. – 2680 pp.
8. Pocket Book of Hospital Care for Children. – World Health Organization, 2013. – 438 pp.
9. Nelson Textbook of pediatrics / R. M. Kliegman, B. F. Stanton, J. W. St Geme III, N. F. Schor ; ed. by R. E. Behrman. – 20th ed. – Canada : ELSEVIR, 2016. – 5315 p.

Lecture № 3

Topic: «Natural feeding of newborns. Advantages of natural feeding of newborns. Value of

breastfeeding for health of the child and mather. Quantitative and qualitative structure of parent milk. An immune biological role of parent milk. Methods of calculation of daily volume of meal and a diet. Rules and techniques of feeding by parent milk. Additional feeding and correction of a meals. Difficulties in breastfeeding. Prevention of gypogalactia and mastitis» – 2 hours.

The actuality of the topic: Knowledge of Natural feeding of newborns, Advantages of natural feeding of newborns, Value of breastfeeding for health of the child and mather, Quantitative and qualitative structure of parent milk, An immune biological role of parent milk, Methods of calculation of daily volume of meal and a diet, Rules and techniques of feeding by parent milk. Additional feeding and correction of a meals, Difficulties in breastfeeding, Prevention of gypogalactia and mastitis is very important for a doctor. How to properly organize a child's diet since the first days of life – a guarantee of its health and harmonious development (the psychophysical). Exactly so physician to any professions must know bases an dietetics of children of the early age.

Aims of the lecture

2.1. Practical (training) aims:

- Acquaint the student with advantage breastfeeding. To teach them correctly assign your child eats first year of life receiving breast milk, with a gradual introduction of the cor~~rec~~ti~~on~~ and complementary feeding.

2.2. Educational aims: Tribute local pediatricians to develop dietetics infants (O.M. Khokhol, V.D. Ott and others) note that the industrial production of synthetic nutrient mixtures are not currently plays the unique composition and properties of human milk, so much depends from the doctor tp convince her mother to save lactation.

Plan and structure of the lecture :

№№	**The main steps of the lesson**	**The aims in the levels of learning of the material**	**Materials of methodical software**	**Time (in minutes or %) of a hole lesson**
1	**2**	**3**	**4**	**5**
I.	*The preparatory level*	1	The presentation, Auditory	5%
1.	The defining the purpose			
2.	Formation of the positive motivation			
II.	*The basic level.*	*II.*		85-90%
3.	The presentation of the			

	lecture by plan 1. Benefits pf breastfeeding 2. Quantitative and qualitative composition of breast milk 3. Methods of calculating power 4. Terms and Techniques feeding breast milk 5. Complementary feeding and correction 6. The need of the child in proteins, fats, carbohydrates 7. Mode and power feeding women. 8. Difficulties breastfeeding Prevention hypogalactia and mastitis.	*II*. *II*. *II*.	Illustrations Literature, questions, tasks.	
III.	Final level.			5%
4.	The conclusion of the lecture.			
5.	Answering questions.			
6.	The task for self-control			

The content of the lecture

The text of the lecture:

Breast-feeeding of infants. Composition of human breast milk. Quantity of feeding. Number of feeding daily. Supplementary feeding. Food requirements infants. Other foods for infants.

Breast milk as the natural food for full-term infants during the first months of life.

Human milk is the most appropriate of all available milks for the human infant because it is uniquely adapted to his or her needs.

It is always readily available at the proper temperature and needs no time for preparation.

The milk is fresh and free of contaminating bacteria, which reduces the chances of gastrointestinal disturbances.

Allergy and intolerance to cow's milk create significant disturbances and feeding difficulties that are not seen in breast-feed infant.

Human milk contains bacterial and viral antibodies. Macrophages normally present in human colostrum are able to synthesize complement, lysozyme, and lactoferrin.

Milk from the mother whose diet is sufficient and properly balanced will supply the necessary nutrients.

The psychological advantages of breast-feeding for both mother and infant.

Preparation of the retrospective mother.

The physician interested in aiding the .prospective mother to breast-feed should discuss its advantages during the midtrimester of pregnancy or whenever the mother begins planning for her baby.

Physical factors concerned to a good breast-feeding experience include establishing and maintaining a state of good health, proper balance of rest and exercise, freedom from worry, early and sufficient treatment of any infectious disease, and adequate nutrition.

Retracted nipples usually benefit from daily manual breast-pump traction during the latter weeks of pregnancy; truly inverted nipples may be helped by the use of milk cups, starting as early as the 3rd month of pregnancy.

The mother may be confidently told that she need not gain or lose weight if her diet is adequate.

Establishing and maintaining the milk supply.

The most satisfactory stimulus to the secretion of human milk is regular and complete emptying of the breasts. The early establishment of normal, vigorous nursing by letting the infant empty the breast frequently during the time when only colostrum is being formed. The infant should be allowed to nurse when hungry, whether or not there appears to be any milk. Breast-feeding should begin as soon after delivery as the condition of the mother and of the baby permits, preferably within the first hours.

Appropriate care for tender or sore nipples should be instituted before severe pain from abrasions and cracking develops. Exposing the nipples to air; applying pure lanolin; avoiding soap, alcohol, and tincture of benzoin; frequently changing disposable nursing pads lining the brassiere cups; nursing more frequently; manually expressing milk; nursing in different positions; and keeping the breast dry between feedings is recommended. When the tenderness causes the motner apprehension the milk-ejection reflex may be delayed, leading to frustration in the infant and to increasingly vigorous nursing, which further injures the nipple and areolar area. Occasionally, nipple shields may be helpful.

Psychological factors.

No factor is more important than the happiness, relaxed state of mind. The physician recognizes and appreciates mothers worries, particularly if the baby is a first-born, and by tactful reassurance and explanation can help prevent or minimize worry, thus contributing to successful breast-feeding.

Hygiene.

Once a day, the breasts should be washed. If soap is drying to the nipple and areolar area, its use should be discontinued. The nipple area should be kept dry. Care should be taken to prevent irritation and infection of the nipples caused by prolonged initial nursing, maceration from wetness of the nipple, or rubbing of by cloth.

Diet.

The diet should contain enough calories to compensate for those secreted in the milk as well as for those required to produce it. The nursing mother needs a varied diet, sufficient to maintain her weight and high in concentrated fluid vitamins, minerals.

Whenever possible, nursing mothers should not take drugs, because many preparations are harmful to the neonate and many have not been evaluate.

Technique of breast-feeding.

1. .At feeding time, the infant should be hungry, dry, neither too cold nor too warm, and held in a comfortable, semisitting position for his or her enjoyment and for ease of eructation without vomiting.
2. The mother, too, must be comfortable and completely at ease.
3. The baby is supported comfortably with the face held close to the mother's breast by one arm and hand while the other hand supports the breast so that the nipple is easily accessible to the infant's mouth and yet does not obstruct the infant's nasal breathing. The baby's lips should engage considerable areola as well as nipple.
4. Some infants will empty a breast in 10 min; others nurse more leisurely for 20 min. Most of the milk is obtained early in the feeding; 50% in the first 2 min and 80-90% in the second min.
5. At the end of the nursing period, the infant should be held erect over the mother's shoulder or on her lap with or without gently rubbing or patting the back to assist in expelling swallowed air; often this "burping" procedure is necessary one or note times during the feeding as swallowed as 5-10 min after the infant has been put into the crib.
6. The infant should empty at least one breast at each feeding; otherwise, it will not be stimulated to refill. Both breasts should be used at each feeding to encourage maximal production of milk.

Determining adequacy of milk supply.

If the infant is satisfied after each nursing period, sleeps .2-4 hr, and gains weight adequately, the milk supply is sufficient. If the infant nurses evidently or normally and completely empties both breasts but appears unsatisfied afterward, does not go to sleep or sleeps fitfully and awakens after 1-2 hr, and fails to gain weight satisfactorily, the milk supply is probably inadequate.

In general, a mother weighing her infant before and after nursing is neither necessary nor desirable in judging adequacy of milk supply. The amount of milk that an infant takes at a time is usually unimportant (the amount ingested at each feeding ranges from one to several times throughout a 24-hr period), and the results obtained are readily misinterpreted.

Before assuming that the mother produces insufficient milk, three possibilities should be excluded:

1) errors in feeding technique responsible for the infant's inadequate progress;
2) remediable maternal factors related to diet,-rest, or emotional distress;
3) physical disturbances in the infant that interfere with eating or with weight gain.

Expression of breast milk.

Although manual expression of breast milk is useful to relieve engorgement of the breasts in an emergency, the cost and availability of battery-operated and electric breast pumps usually makes this unnecessary.

Supplementary feedings.

Mother who has returned from work the breast milk production will gradually decrease so that the mother is not plagued by engorged, leaking breasts. She will usually continue to produce enough milk to supply two or three feedings a day for several months. If formula is to be given after the infant has completed a breast-feeding, the warmed bottle should be available so that it can be offered immediately after the infant has been burped. The holes in the nipples should not be so large that the infant gets this portion of food without any effort, or the infant may quickly abandon any efforts to suck adequately at the mother's breast.

Weaning.

Most infants gradually reduce the volume and frequency of their demand for breast-feedings at 6-12 month of age, and they become accustomed to increasing amounts of solid foods and liquids by bottle and cup. Weaning should be initiated by substituting formula or cow's milk by bottle or cup of part of a breast-feeding and subsequently for breast-feeding. Over several days, one of the breast-feedings is replaced and then subsequently another, and so on, until the infant is weaned completely. These changes should be made gradually.

When cessation of nursing is necessary at an earlier age, a tight breast binder may be used, and ice bags may be applied for a few days to decrease milk production. Restriction of the mother's fluid intake is also helpful. Hormones, such as small doses of estrogen for 1 -2 days, also may help decrease milk production at the termination of nursing.

Contra-indications.

For the average, healthy, full-term infant there are no disadvantages to breast-feeding, provided that the mother's milk good is ample and that her diet contains sufficient amounts of protein and vitamins.

Infrequently, allergens to which the infant, is sensitized may be conveyed in the milk. In such cases, an attempt should be made to find the specific allergen and to remove it from the mother's diet; its presence rarely is a valid reason for weaning the baby.

From the mother's standpoint, there are few contraindications to breast-feeding. These are markedly inverted nipples, fissuring or cracking of the nipples, mastitis. Septicemia, nephritis, eclampsia, profuse hemorrhage, active tuberculosis, typhoid fever, breast cancer, and malaria are contraindications to nursing, as are chronic poor nutrition, substance abuse, debility, severe neuroses, and post-partum psychoses.

Comparing of human milk and cows milk.

Both differ during the various stages of lactation and among individuals, although the differences in human milk from women with adequate diets are insignificant. Milk

during pregnancy and early after birth contains more protein, calcium, and other minerals than later during lactation. Cells are also present in colostrum and human milk.

Colostrum.

The secretion of the breasts during the latter part of pregnancy and for the 2-A days after delivery is called "colostrum". It has a deep lemon yellow color, its reaction is alkaline, and its specific gravity is 1.040-1.060, in contrast to the average specific gravity of 1.030 for mature breast milk. The total amount of colostrum secreted daily is 10-40 ml. Human or cow colostrum contains several times the protein of mature breast milk, more minerals, but less carbohydrate and fat. Human colostrum also contains some unique immunologic factors. After the first few days of lactation, colostrum is replaced by secretion of a transitional form of milk that gradually assumes the characteristics of mature breast milk by the 3rd or 4th wk.

Water.

The relative amounts of water and solids in human and cow's milks are about the same.

Calories.

The energy value of each milk may vary slightly and is approximately 0.67 kcal/ml.

Protein.

There are quantitative differences between the proteins of the two milks. Human milk contains only 1-1.5% protein compared with approximately 3.3% in cow's milk.

The increased protein of cow's milk results almost entirely from its 6-fold higher content of casein. Human milk protein consists of approximately 65% whey proteins, largely lactalbumins, and 35% casein;

Carbohydrate.

Human milk contains 6.5-7%, and cow's milk contains about 4.5% lactose. About 10% of the carbohydrate in human milk consists of polysaccharides and glycoproteins.

Fat.

The fat content of milks is about 3.5%, In human milk, fat content varies somewhat with maternal diet; during a single nursing, it is higher in the latter portion of the feeding, which may help satiate the infant at the conclusion of nursing.

The milks of different breeds of cattle vary in fat content. Most market milk in urban areas, however, is pooled, and the fat content is adjusted to a standard level, generally from 3.25-4%.

Qualitative differences exist in the fats of human milk and cow's milk. The fats of each consist principally of the triglycerides olein, palmitin, and stearin, but human milk contains twice as much of the more absorbable olein. The volatile fatty acids (butyric, capric, caproic, and caprylic) constitute only about 1.3% of human milk fat but about 9% of cow's milk fat. The small amount of linoleic acid in cow's milk is usually sufficient to prevent deficiency. The premature or debilitated infant may have steanorrhea after ingesting cow's milk fat. For such infants, it is wise to substitute a more readily assimilated vegetable fat or human milk.

Minerals.

Cow's milk contains much more of all the minerals except iron and copper than human milk; total mineral content of cow's milk is 0.7-0.75%; that of human milk is 0.15-0.25%. Cow's milk contains inadequate iron; breast-milk iron, although low, may be sufficient for the infant because it is better absorbed, and during the first 4 months some of iron stored during fetal life compensates for the milk's deficiency.

Although the need for calcium and phosphorus is great during periods of rapid growth, adequate balances are maintained on breast milk despite its low content of these minerals.

Vitamins.

The vitamin content of each milk varies with the maternal intake. Cow's milk is low in vitamins C and D. Breast milk usually contains adequate vitamin C, if the mother eats appropriate foods, and adequate vitamin D unless she is insufficiently exposed to sunlight or is darkly pigmented. Cow's milk contains more vitamin K than human milk. Both types of milk seem to contain adequate amounts of vitamin A and the B-complex vitamins for the nutritional needs of infants in the first months of life.

Bacterial content.

Although human milk is essentially uncontaminated by bacteria, pathogenic organisms in significant numbers may enter the milk from mastitis. Tubercle and typhoid bacilli and herpes, hepatitis B, rubella, mumps, HIV, and CMV may be present at times in the milk of women infected with these organisms. Cow's milk is regularly contaminated, but in most cases by bacteria that are not harmful to human. Milk, however, is a good culture medium for pathogenic bacteria, and many infections are milk borne, including streptococcal diseases; diphtheria; typhoid fever; salmonellosis; tuberculosis; and brucellosis. Furthermore, certain bacteria that may not affect older children or adults may cause diarrhea in infants. In most cities, pasteurization of all marketed whole milk is required. In addition, terminal sterilization or boiling the milk immediately before mixing the infant's formula is advisable.

Digestibility.

The stomach empties more rapidly after human milk than after whole cow's milk; however, no appreciable difference in gastrointestinal passage time exists between human milk and processed milk formulas during the first 45 days of life. The curd of cow's milk is reduced in size by boiling; it is made considerably less tough and much smaller by the heating required in evaporation, by the addition of acid or alkali, and by homogenization. In contrast, the curd of breast milk is fine and flocculent and readily broken down in the stomach. The fat of cow's milk is less readily digested than that of breast milk.

Caloric requirements.

The average caloric requirements of full-term infants is about 120 kcal/kg during the first three months of life, from three till six month – 115 kcal/kg, from six till nine – 110 and about 100 kcal/kg by 1 yr of age; individual variations are significant, and for many infants intakes of this order exceed caloric need.

Fluid requirements.

Fluid requirements are high during infancy. During the first 6 months of life, they range from 130-190 mL/kg/24 hr and may increase during hot weather. As a rule, the infant regulates his or her own fluid intake, provided adequate amounts are offered. Most of the fluid required is in the formula, but some is supplied in juice and other foods and by water between feeding.

Protein requirements.

After birth till three months protein needs are 2,2 g/kg/24 hr, from three till six months - 2.6 g/kg/24 hr and after inclusion(6 months) «solid foods» in infant diet 2,9 g/kg/24 hr.

Fat requirements.

After birth till three month fat needs are 6.5 g/kg/24 hr. from three till six months 6.0 g/kg/24 hr and till one year 5.5 g/kg/24 hr.

Carbohydrates requirements.

During infancy period carbohydrate needs are 13g/kg/24 hr.

Number of feedings daily.

The number of feeding required per day decreases throughout the first year; by 1 yr of age, most infants are satisfied with 4 meals/day. The interval between feeding differs considerably among infants but, in general, ranges from 3-5 hr during the first year, of life, averaging 4 hr for full-term, healthy infants. Small or weak infants may prefer feedings at 2- to 3-hr intervals. For the 1st month or 2, feedings are taken throughout the 24-hr period, but thereafter, as the quantity of milk consumed at each feeding increases and the infant adjusts his or her demand to the family pattern of daytime activity, the infant usually sleeps for longer period at night. As the infant develops psychologically and the loving relationship between the parent and infant evolves, demand feeding should gradually progress to a feeding regimen that accounts for the needs of both the infant and the parents.

Quantity.

There are some methods of calculation the quantity food Daily in time infancy period.

For infant after birth till 7-8 day Phinkelshtane formula can used. Quantity milk is coefficient (70 or 80) to multiply day after birth. Coefficient 80 used if birthweight more than 3200g.

Q = 70(80)-n, n – day of life

Other method for this age it is formula Zaicevoy

Q = 2% from birthweight • n (n – day of life)

The two methods (volumetric and calorie) are used since 2 weeks. Volumetric method.

Quantity milk daily calculated how part of standart weight of infant. This method is used till nine month.

1. From 2 w. to 2 month – 1/5.
2. From 2 m. to 4 month – 1/6.
3. From 4 m. to 6 month – 1/7.
4. From 6 m. to 9 month – 1/8.

Calorie method. This method is used for energy needs of infant .Breast milk contains thereabout 700 kcal/1. This method is used only till inclusion of “solid foods” in infant´s diet.

Other foods.

“Solid” foods.

The caloric contents of the various prepared baby foods differ widely. Egg yolk, cereals with added milk, meats, and puddings have greater caloric density than milk, whereas vegetables and fruits have an energy value similar to or lower than milk. Without appropriate advice, many mothers select foods with high caloric values that result in obesity. The inclusion of solid foods to the diet before 4-6 month of age does not contribute significantly to the health of the normal infant nor does it increase the likelihood of the infant sleeping through the night, providing hunger is avoided with adequate breast-feeding or formula feeding.

Any new food should be initially offered once a day in small amounts (1-2 teaspoonfuls). Any small spoon that easily fits the baby's mouth should be used. New foods are generally best accepted if fairly thin or dilute. Food is frequently pushed out by the tongue rather than back because the baby cannot yet swallow efficiently. This should be mentioned to the mother, who might otherwise interpret the "spitting out" of new foods as dislike. It is usually wise to offer the same food daily until the baby becomes adapted to it and not to introduce new foods more often than every 1-2 wk.

The feeding at which these foods are offered is not particularly important. They should be given when the baby's hunger is no longer satisfied by milk alone and when they fit into the daily schedule. There is no reason for persisting with or forcing a particular food that is definitely disliked. The family's dislikes and prejudices for particular foods are contagious and should not be displayed before the infant. The physician should avoid prescribing a definite amount of a given food left the mother interpret the suggestion too literally. Many infants are overfed by overzealous parents who mistake acceptance of food for appetite. The infant's appetite is the best index of the proper amount, and respect for the infant's wishes will avoid many problems.

Cereal.

The various precooked cereals on the market provide in a convenient form a variety of grains excellent for infants. Most contain iron and factors of the vitamin B complex. Cereal started from six month.

Fruits.

Strained or pureed cooked fruits furnish minerals and some water-soluble vitamins and usually have a mildly luxative effect. Raw ripe mashed banana is readily digested and enjoyed by most infants. Many infants who are slow in accepting new foods seem to prefer fruits. Started from 6 month.

Vegetables.

Vegetables are moderately good sources of iron and other minerals of the complex vitamins. They should be freshly cooked and strained or commercially prepared.

Vegetables are usually added to the infant's diet by about 6 month of age.

Meat, eggs, and starchy foods.

Eggs and starchy foods are usually introduced during the second 6 month of life – 7–7,5 months, although some physicians offer egg yolk at an earlier age. The yolk of the egg is used initially and is preferably hard-cooked. As with all new foods, a small amount is offered at first, with gradual increases up to a whole yolk 1-3 times a week. Egg white should be introduced with equal caution to minimize any possible allergic manifestations.

Potatoes, rice, spaghetti, bread, and similar starchy foods have principally a caloric value. As a rule, they are not included in the infant's diet until the more essential foods mentioned earlier are being taken regularly. Zwieback, toast, or graham crackers may be offered to the infant when he or she shows an interest in “gumming” on coarser foods (usually 10 months of age). It is with such foods that infants leam to chew and to feed themselves.

Meat (6,5 – 7 months of age) is an excellent source of protein as well as of iron and vitamins. Ground fresh beef or liver or the strained canned meats may be used initially by about 8 mo of age. Meats may be more readily accepted when mixed with another food.

The commercial soups and meat and vegetable mixtures are relatively high in carbohydrate and are not considered optimal sources of iron or protein. Many home-prepared soups are bulky out of proportion to their food value, and much of the vitamin content is lost by overcooking.

Desserts.

Puddings, junkets, and custard are good foods for older infants, particularly if they temporarily prefer milk in that form. If, however, such foods are given as a bribe or reward or only after other foods have been finished, poor eating habits are likely to be established. Sweet foods should be offered as casually as the rest of the meal and at any place in the meal that the child desires.

Salt intake.

To increase their palatability, particularly for the parent, excessive salt used to be added to baby foods. This practice has been discontinued. The significance of large intakes of sodium, which are in the ranges seen in populations with a high incidence of hypertension, is not clear, but the possibility that they might contribute to the development of hypertension later in life cannot be ignored.

physical development of children.

Materials to activate the students during lecture:

Questions :

1. Benefits pf breastfeeding.
2. Quantitative and qualitative composition of breast milk
3. Methods of calculating power.
4. Terms and Techniques feeding breast milk.
5. Complementary feeding and correction.
6. The need of the child in proteins, fats, carbohydrates.
7. Mode and power feeding women.
8. Difficulties breastfeeding Prevention hypogalactia and mastitis.

6.**Materials of methodical software of the lecture:**

- Auditory
- Software
- Illustrations

The materials for self study of students. (Literature for the student.)

c) For the topic of the current lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 385 – 431; 770 – 773.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

d) For the topic of the next lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 431 – 447; 770 – 773.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

Literature used by lector during preparation the lecture:

10. Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – 808 pp.

11. Partha,s Fundamentals of Pediatrics. Ajanta offset &Packagings Ltd., New Delhi.-2013.- 782 pp.
12. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.
13. Vicky R. Bowden, Cindy S. Greenberg. Pediatric nursing procedures. - Lippincott Williams & Wilkins. - 2011. - 822 pp.
14. Pediatric Nursing Procedures. Vicky R. Bowden, Cindy S. Greenberg. - Wolters Kluwer Health.- 2015 -728pp.
15. Nelson. Essentials of Pediatrics. Sixth Edition. Canada: 2011. - 831p.
16. Robert M. Kliegman. Nelson Textbook of Pediatrics 19th ed. – W.B. Saunders Company, 2011. – 2680 pp.
17. Pocket Book of Hospital Care for Children. – World Health Organization, 2013. – 438 pp.
18. Nelson Textbook of pediatrics / R. M. Kliegman, B. F. Stanton, J. W. St Geme III, N. F. Schor ; ed. by R. E. Behrman. – 20th ed. – Canada : ELSEVIR, 2016. – 5315 p.

Lecture № 4

Topic: «Anatomical and physiological features of nervous system in children. Semiotics of diseases» – 2 hours.

The actuality of the topic: Specific function of cells, organs and systems, coordinate their functions in the whole organism carried by the nervous regulation. Modern diagnostic process in diseases of somatic cells is not without Bargain study the nervous system condition. Success of the application neurological Studies in Practical activities depends on physician knowledge of nervous system and its features of in children.

Aims of the lecture

2.1. Practical (training) aims:

-Familiarizing students with the signs and methods of assessing children with nervous-mental development.

- Familiarizing students with clinical symptoms of nervous breakdown and with modern methods of its inspection;

-Familiarizing students with the methods of looking after children with a pathology of nervous system.

2.2. Educational aims: - formation in students a responsive treatment towards children, using a clinical observation of the children with abnormalities of a nervous regulation,

- formation in students a clinical and scientific thinking.

Plan and structure of the lecture :

The main steps of the lesson	**The aims in the levels of learning of the material**	**Materials of methodical software**	**Time (in minutes or %) of a hole lesson**
2	**3**	**4**	**5**
The preparatory level	1	The	5%

		presentation, Auditory	
The defining the purpose			
Formation of the positive motivation			
The basic level.	*II*.		85-90%
The presentation of the lecture by plan 1. Anatomical and physiological features of nervous system in children. 2. Nervous-mental development and its estimation**.** 3. Semiotics of major nervous system diseases.	*II*.		
Final level.	*II*.	Illustrations	5%
The conclusion of the lecture.	*II*.		
Answering questions.			
The task for self-control	*II*.	Literature, questions, tasks.	

The content of the lecture

The text of the lecture:

The nervous system reasonly is named as the main and the basic system of living organism, managing the internal world and external displays of the human ability to live. This is the nervous system that provides the person the best adaptation to constantly varying conditions of internal and external environment. It is the basic tool to know the world and its transformation. During the functioning, the nervous system of the person forms displays of feelings and movements, skill count, answers for the behavior, written and oral speech. It is known that the person's nervous system, including the child's one, consists of two basic parts such as *peripheral and central*. The **central nervous system** includes the *back and the head brain*. It, in its turn, consists of several departments: a trunk brain, including medulla or, as it

is often named the Varoliy bridge, and legs of the brain, medulla, big brain, which structure includes the formations of an intermediate brain and cortex of hemispheres.

- When we talk about a head brain of a child, it is necessary to take into account, that to a moment of birth his nervous system is not completely generated yet, it differs by significant immaturity; its various departments are not in an equal measure.

- The nervous system develops at early stages of embryogenesis - the tube is formed from ectoderm, and 3 bubbles on the third week- (forward brain bubble, average and back) then their differensation on a final brain, intermediate, average, back and medulla occurs.

- By the III month of embryogenesis there are basic departments of the centra1 nervous system: large hemispheres, ventricles, back brain.

- By the V month basic shares are differentiated , though cortex is insufficiently advanced yet.

- On the VI month cortex of head brain above bottom lying departments begins to prevail.

- A newborn weight of a head brain makes 1/8 weight of a body (at about 400 g), boys' weight is a little bit higher. The crinkles, shares of newborns are rather expressed.

- To the 9-th months of life the weight of a brain is doubled, by the end of the 1-st years it makes 1/10- 1/11 parts of the weight of a body (about 1000 g).

- The back brain is more completed in a morphological structure of newborns. It is rather longer, than adults have, it reaches up to the third lumbar vertebra. In this period the projection of segments does not correspond to vertebra. Hereinafter the growth of a back brain lags behind the growth of a backbone and as a result it reaches their conformity. Definitively the growth comes to the end in 5-6 years.

- The peripheral nervous system is not enough covered well by myelin yet. These processes occur non-uniformly. So, mielinisation of cranial-brain nerves fulfils in first 3-4 months of life and comes to the end by the 3year.

- The vegetative nervous system of newborns functions, and prevails simpatic department over parasimpatic (tachicardiais explained by it).

- A child is supplied with a set of the vital systems (breath, sucking, swallowing). They provide the ability to revive a newborn even under premature birth, as they are formed long before birth. The most sensible nervous system is during 5-7 weeks of embryogenesis. Because of this various embriopaties occur.

- Almost all reflexes of automatism can be caused from a preaturely born (sucking, swallowing, of Babkin, Moro, etc.).

- The undercortex function of prematurely born children is more expressed. It is explained by their hyporeflexion, hypotonetion of muscles, tremor of extremities and chin, easy acetose, they can have easy changeable squint, small changeable horizontal nistagm. These neurological changes can be observed till 2-3 weeks of life. The term of their disappearance depends on a degree of prematurely bornetion.

➢ A prematurely born child has high sensation of the nervous system (it is connected with increased breackness of vessels, insufficiency of the humoral factors); therefore we meet cranial-brain traumas more often.

AGE PECULIARITIES OF CEREBROSPINAL FLUID

Parameters of cerebrospinal fluid	Age of baby			
	Till 14th day	From 14th day till 3 months	4-6 months	> 6 months
Color	Xanthochromiya, bloody	Colorless	Colorless	Colorless
Transparency	Transparent	Transparent	Transparent	Transparent
Protein (g/L)	0.4 – 0.8	0.2 – 0.5	0.18 – 0.36	0.16 – 0.24
Cytosis (in 1 mkl)	3/3 – 30/3	3/3 – 25/3	3.3 – 20.3	3/3 – 10/3
Kinds of cells	Chiefly Lymphocytes, single Neutrophiles	Chiefly Lymphocytes	Lymphocytes	Lymphocytes
Reaction of Pandy	From + till ++	Till +	Rarely +	–
Sugar (mmol/l)	1.7 – 3.9	2.2 – 3.9	2.2 – 4.4	2.2 – 4.4

SEQUENCE OF NEUROLOGICAL EXAMINATION

I.QUESTIONING:

1. Passport dates
2. Complaints
3. Anamnesis morbid (case history)
4. Anamnesis vitae (life history)
5. Assessment of psycho-physiological development of the child

II. GENERAL INSPECTION:

1. Inspection of head
2. Expression of face
3. Patient's position on his bed and posture of staying

III. EXAMINATION OF THE CRANIAL NERVES

IV. PALPATION AND PERCUSSION

V. EXAMINATION OF THE MOVING AREA:

1. Volume of movements
2. Strength of movements
3. Muscle tonus
4. Coordination of movements (posture of Romberg, knee-heel probe, finger-nose probe)

VI. EXAMINATION OF REFLEXES:

1. Physiological reflexes of a newborn
2. Skin reflexes (top, middle, bottom abdominal, cremasteric)
3. Reflexes from mucous membranes (corneal, palatal, pharyngeal)

4. Tendon reflexes (from biceps, triceps, knee, Achilles
5. Pathological reflexes (Hwostec's and List's)

VII. EXAMINATION OF SENSITIVITY:

1. Pain sensitivity
2. Touch (tactile) sensitivity
3. Temperature sensitivity
4. Deep sensitivity (proprioceptive and vibration)

VIII. EXAMINATION OF MENINGEAL SIGHS:

1. Rigidity (hypertension) of occipital muscles
2. Higher, middle, lower symptoms of Brudzinsky
3. Symptom of Lessage
4. Condition of frontal fontanel

IX. PARTICULARITIES OF CEREBROSPINAL FLUID

COMPLAINTS TIPICAL FOR DAMAGE OF THE NERVOUS SYSTEM

- ❖ **Headache**
- ❖ **Giddiness**
- ❖ **Weakness**
- ❖ **Damage of sensitivity**
- ❖ **Damage of intellect**
- ❖ **Weaken of memory**
- ❖ **Sleep dysfunction**
- ❖ **Chang of speech**
- ❖ **Mood and behavior**
- ❖ **Dysfunction of consciousness with its depressions**
- ❖ **Convulsions and spasm of muscles**
- ❖ **Vomiting, nause**
- ❖ **Paresis, paralysis, muscles weakness**
- ❖ **Damages of coordination of movements**

While characterising the ner neuro-psychological development **(NP**

Criteria of estimating NPD are:

- Motility
- Statics
- Conditioned-reflexes
- Speech
- The higher nervous activity

(К) Develops of motility, statistics and speech in the healthy child during 1st year of life

Movement and skills	*Mean (average) age*	*Permissible limit*
Smile	5 weeks	3-6 weeks
First sounds (u-u-u, a-a-a, agu-agu, etc.)	5 weeks	4-6 weeks
Holding of the head	3 months	2-4 months
Purposeful activity of hands	4 months	2,5-5,5 months
The turning from supine position to the abdominal	5 months	3,5-6,5 months
Sit	6 months	4,5-8 months
Babble (separate syllables like ba-ba-ba, ma-ma-ma,	6 months	5-7 months

etc.)		
Crawl	7 months	5-9 months
Stand	9 months	6-11 months
Walk	11,5 months	9-14 months

By the **end of the first year** of life the child's vocabulary already contains **8-10** words, and he/she understands their sense ('mamma', 'baba', 'dada' etc.). By the **age of 2 years** the child's vocabulary **reaches 300 words**.

Nervous and psychological growth of a child in the 1st year of birth.

1st month. The position of the child is usual at the joints of arms and legs.

By the end of the 1st month, garises the consciousness of the child at the objects around. The child learns to follow the direction of any particular movements with the aid of a simple eye with out using the brains. The child can also comprehend any emitted voice as such.

2nd month. Increases the activity towards movement. Throwing up of the arms above the horizontal level and opening of the clutched fist's. Holding up the head at vertical position for a definite period of time takes place at the 2nd month of the child. When coaxed the child smiles, which indicates us about the siotional reaction. Reflexes at this time are noted except for the reflex of defecation.

3rd month. Increases the movement of the arms *and* eyes, mainly the upper limb joints. The child raises his or her arms more frequently at the horizontal level, and holds firmly the toy hitched it with the hand. The child smiles or at times laugh at bright objects it exhibited which shows the siotional reaction.

4th month. At this phase the child can hold his or her head firmly and rolls over to the side from where any particular sound can be heard. During this period the child is attracted to any trinkets or toy, tries to get hold of it, feels it and puts it in the mouth. If the hands of the child are tugged, he / she sits up right. The child might lie on his abdomen with the arms stretched angularly, and lifts the upper part of the body. During this period, flexor hyper tonicity is absent and the child keenly concentrates upon any moving object.

5th and 6th month. The child can sit if with one of his/her hands are held, and can turn independently upon the spine at any side. When lying on his abdomen, his/her hands are folded and often lifts the upper part of the body. The child might lie prostrate on its back and play fully moving its legs, while turning the head toward the direction of any particular emitted sound, and can distinguish known and unknown faces. Emotionally the child becomes more sensitive and at this stage the first two words are pronounced by the child for ex. – "pa", "ma", "da" etc.

7th and 8th month. Can sit independently, can turn on its stomach and back and firmly can stand on its legs. Motion's indicate definite character and the child stretches out his / her hand in the direction of his / her mother or someone known. The child at this stage learns to clinch his / her fist's and cry out "ma-ma" or "ba-ba" and tries to attract the attention of the elder's towards his own. During these months, the child can well differentiate among people his parents and grown within the sense of fright when he/she sees strangers, while expressing great joy when coming across new objects.

9th and 10th month. At this period the child can stand with the help of some sort of a support and learns to crawl. Cries out words like "papa", "mama", "baba" etc. and obeys to simple commands of the elders.

11th and 12th month. Walks with the support of one hand, and can make separate steps with his own self (independently). Sits on his own so that he could pick up its dolls or toys

to play with identifies the name of the several objects, what causes them pain or hurt. Point out the parts of the body, helps while changing clothes. Can hold the spoon or can eat by itself, loves to play with children, identifies the members of the family, pronounces several words.

EXAMINATION OF THE CRANIAL NERVES

I pare (nerve of sense of smell) – at the infants function of the first pare is checked in directly for reaction of child to pungent smell substances; at the older children – by the way to breathe in of different smelled substances and describing the smell.

II pare (optic nerve) – is checked the sharpness of sight, fields of vision, color filling and eye bottom (eye ground).

III pare (oculomotor nerve), IV pare (trochlear nerve) and VI pare (abducent nerve) – are checking at the same time. The physician ask the child to see without turning this head to subject which is location to the right, to the left, to the top, to the bottom.

V pare (trigeminal nerve) – function of movements fibers are checking to stiffness of chewing muscles, function of sensitive branches are checking by ordinary way.

VII pare (facial nerve) – for checking this nerves pare it is suggesting to patient to wrinkle forehead, to close his eyes, to show his tooth, to inflate his cheeks.

VIII pare (vestibulocochlear nerve) – its checking a distance the child can hear whispered speech.

IX pare (glossopharyngeal nerve) and X pare (vagus nerve) – function is defining to condition of soft palatine and its mobility, and nose shade of voice.

XI pare (accessory nerve) – the peripheral paralysis of shoulder, impossibility of increasing of shoulders above middle level, damage of head turning are arising with damages this pare.

XII pare (sublingual (hypoglossal) nerve) – function is defining for position of tongue in the mouth and moving them forward.

Two types of the diseases can be distinguished in the Nervous system: **functional** and **organic**. The first one appears due to functional disorders.

Defects in the activities of the Nervous system are caused by different **reasons:**

I. The main antenatal risk factors:

- Various chronic diseases of mother (anemia, arterial hypertension, chronic glomerulonephritis, heart diseases, diabetes, toxoplasmosis, rheumatic fever, etc.); infectious diseases of mother during pregnancy
- Intra-uterine infections
- Genetic defects (if the parents are mentally retarded, the probability of the birth of a child with neurological pathology increases 2 times as compared with healthy parents)
- Alcohol, smoking
- Professional aspects (hard physical work, vibration)
- Exogenous factors (high radiation, chemical substances, etc.)
- Complicated gynecologic anamnesis (the birth of the first child before 16-18 years or after 30 years, if the interval between two pregnancies is less than 2 years, threat of miscarriage, stressful conditions)
- Incompatibility of Rh-factor and ABO system
- Prolonged pregnancy, twins, hypotrophy of the newborn

II. Psychological trauma: (The psychological disorder of children may be caused by various factors. The main factors are)

- ✓ conflicts in school life (quarrelling, disagreement, opposite opinions)
- ✓ carelessness of the family (facing step father or mother)
- ✓ death of a family member or newborn brother or sister
- ✓ punishment without guilt
- ✓ watching the adult movies

In these cases we can realize that the diseases are caused not only by the character of psychological trauma but also by the state and the condition of the nervous system and its reaction. A child can face a serious trauma and may stay healthy; and other comparatively due to the insufficient stimulant strength causes Nervous disorder or disturbance (neurosis).

Stigmas

Stiamas are fine anomalies of development determined during the external examination of the child.

The most widespread stigmas are:

- Protrusion and hanging of the occipital part of the skull
- Micrognathia ('erased' chin), sometimes it is named 'the bird face' (brachygnathia)
- Macrognathia (the chin is considerably protrudes in front)
- Split chin
- Low or asymmetrically located ears
- Joint earlobes
- Deformation of earlobes
- Low growth of hair on the forehead, neck
- Epicanthus (=mongolian fold=plica semilunaris conjunctivae) - it's a fold of skin which covers the eyes
- Ptosis (falling or sinking down of the eyelid)
- Too short/too long neck, torticollis (=wryneck=stiffneck)
- A significant amount creases on the neck
- A low lying umbilicus
- Protruding umbilicus
- Polydactyly (more than 5 fingers)
- Brachydactyly (short fingers)
- Arachnodactyly (long fingers)
- Syndactyly (joint fingers)
- Similar stigmas on the feet

Down's syndrome

Down's syndrome (English doctor of the 19thcentury) (=mongolism=congenital acromicria) is one of the forms of oligophrenia on the basis of which lies the trisomy of the 21st chromosome. Etiology remains unknown.

The basic signs of the Down's syndrome are:

I. **Lag in neuro-psychological development:**

- ❖ In early neonatal period, it is already expressed by hypotension of the muscles which remains for a long time; the child can sometimes bring back the lower limbs and behind his/her occiput; quite often the disorders of coordination of movements

remain the whole life

- ❖ The parameters of statics develop on the 2nd-3rd years of life. Sometimes such children start walking only by 5 years and later
  - ❖ Delayed speech development (at 3-5 years of age the child can say I only a few words), defects of speech are frequently seen
  - ❖ Significant intellectual and mental underdevelopment, down to oligophrenia

II. Pathognomonic external signs:

- ➢ Slanting line of the eye, 'mongoloid' eyes
- ➢ Wide and flat nose bridge
- ➢ Macroglossia – the tongue quite often hangs out from the half open mouth
- ➢ Short neck
- ➢ Short feet and hands
- ➢ Brachydactyly
- ➢ On the palm a distinct cross-line is present frequently ('simian' crease)
- ➢ Deformation of feet that makes them look like sandals

III. Disorders of the internal systems:

- ✓ Underdevelopment of the brain
- ✓ Disorder in the differentiation of nervous cells and myelinization of nervous fibers
- ✓ Decrease in the sizes of internal organs and their anomalies (in 25% of cases there are congenital heart diseases)

Prognosis: the disease is not cureable.

Meningeal syndrome

The clinical symptoms arising due to the affection of meninges (inflammatory and not inflammatory genesis), is refered to as **meningeal syndrome.** It's most frequent signs are:

- **Headache** (in children of early age it is monotonous, i.e. monotonous with regard to the sound of their cry).
- **Nausea, vomiting.**
- **In** small children **- protrusion and pulsation of the frontal fontanelle** - a *very significant sign for pediatricians.*
- **General hyperesthesia**(increased sensitivity) - **a** painless **touch** of the skin of **the** child, is accompanied by his getting anxious, cryinq, shouting.
- **Rigidity (hypertension) of occipital muscles - the** doctor **cannot bend the head of the patient forward.**
- **"Meningeal position" –** the head is thrown back, legs are pressed to the abdomen, the child lies on one side
- **Kerning's symptom –** the technique corresponds to **Kerning's reflex,** however after 4 months of age is a sign of pathology.
- **Higher, middle, lower symptoms of Brudzinsky**
- **Symptom of Lessage**

Hydrocephaly - is the disease in which there is increase in brain **spaces containing cerebrospinal** fluid **and the increase in the pressure** of **cerebrospinal fluid.** Hydrocephaly are divided into:

(a) On the time of occurrence - congenital and acquired.

(b) On the course of disease - acute and chronic.

(c) On localization - external (accumulation of cerebrospinal liquid mainly in subarachnoid spaces), internal (increased quantity of liquid in ventricles of the brain) and general (external and internal). In congenital hydrocephaly at birth the sizes of the skull are normal or slight increased. Their noticible increase and other signs develop from the first week*of life.

- The head acquires spherical form.
- Cranial sutures are separate.
- The size of the anterior fontanel increases, its surface protrudes, pulsates; the fontanel remains open for a long time and closes in favorable prognosis after several years.
- The sizes of anterolateral fontanels also increase, they become protruded.
- The scalp becomes thinner.
- Scalp veins become prominent.
- Prominent overhanging forehead.
- Stuck-out ears.
- Sunken and half open eyes.

The pathognomonic sign of hydrocephaly is the 'sun set sign' - a part of the white sclera between the cornea and upper eyelid is visible. The symptom is a sign of increased intracranial pressure. It is also called the symptom 'eye protein'. The symptom can be determined when the position of the child is changed from horizontal to vertical. Periodical appearance of such white striae during the movement of the headis called Graefe's symptom (German opthalmologist of the 19th century).

Gradually the delay of neurophysical development, lag in NPD, intellectual underdevelopment appear in the child.

The prognosis for life may be bad.

Microcephaly is the **reduction of** the size of the skull. The child is **born** with such a head, the facial part of the skull is bigger than its cranial part. The fontanel and sutures between the bones are often closed. Further the facial skull prevails over the cerebral part, the head narrows upwards. Narrow and low forehead, earlobes are big and located low. Intellectual underdevelopment is usually observed.

In very rare cases microcephaly can be acquired, when it develops after the birth in the first months of life.

Cerebral palsy

Cerebral palsy (*ourterm* - children's cerebral paralysis) is defined as group of non progressive neuro-motor disorder of the brain, in the early periods of its formation. Changes in the central nervous system (underdevelopment of the structures of the brain and it's destruction) are different in location, structure and degree of severity.

The severity of disorders and frequency of cerebral palsy depend first of all on the time at which the teratogenic factors acted during the formation of brain. Most frequently (more than 50%) the paralysis arises due to exogenic and endogenic factors during the antenatal period. Often the disorders (40%) are caused as a result of birth trauma and birth asphyxia in the intra-natai-Derjoj, Serious or long term infectious diseases, cerebral trauma can cause cerebral palsy in the postnatal period (less than 10%, according to some data - no more than 3%).

Cerebral palsy is shown by the following clinical signs:

(a) One of the basic symptom is disorders in movement - inability to support a normal pose and to carry out necessary movements.

(b) Frequently there can be:

- Mental disorders.
- Underdevelopment of speech.
- Disorders of vision, hearing and sensitivity.
- Convulsive syndrome.

The founder of cerebral palsy is the English surgeon - orthopaedician of the 19th century - Little. As it was mentioned above, there are a lot of variantions of the disorders of nervous system. Different forms of cerebral palsy are defined depending on the most prevailing character of movement disorders. During the last century some classifications of cerebral palsy were given which are different in different countries.

The most common form of the cerebral palsy is the so-called spastic diplegia (= Little's illness) *(Greek di* - twice, *plege* - impact, *spasticos* - convulsive). The basic clinical display of this disease is the disorder of movement of both upper and lower limhs (i.e. tetraparesis). The disorder in the feet prevails in this case. The degree of destruction and clinical displays are different depending on the activity of the process. Besides, their external signs depend on the age of the child.

During mild forms the symptoms appear at the end of the first half-year of life when activity of the child includes muscles of legs and hips. In more severe condition symptoms appear from the first month. The basic neurological symptom is characterized by the delayed development of the nervous system, and the delay in the disappearance unconditional reflexes.

The basic clinical features are:

(a) **Hypertonus of muscles** which is observed in the neonatal period itself in severe cases. *Recollect! Normally in the first months of life the hypertonus of flexors and the physiological muscular hypertension gradually decrease.* In cerebral palsy these signs are not only present but become even more prominent. The tone prevails in the flexors of arms, extensors and adductor muscles of the leas. The continuous hypertonus of hands results in the delay of the development of manipulative activity. This may be present for a long time - the child can eat, button his shirt, draw, write, etc. much later than normally. Sometimes he/she may not be able to do it during the whole life.

(b) The dissappearance of unconditioned reflexes does not take place on time - grasping, Moro's, support reflexes, etc. One of the early displays of spastic diplegia is the presence in a child of 3 months the reflex of stepping automatism with crossed legs. However it is necessary to note, that sometimes it is impossible to determine the reflexes in a sick child because of the hypertonus of muscles,

(c) The pose of **the child** is a characteristic sign which is different at different ages. Thus, the child sits much later than that normally (later, than 6 months). The body is bent, head is inclined forward, and for balancing in this pose he/she leans on the hands - Fig. 55 C (which too is considerably delays the development of manipulative activity and often does not correspond to the normal age).

(d) The child begins to walk much later, for a long time he/she **walks** only with support (moreover a lot of patients with cerebral palsy walk on tip-toes instead of the whole foot).

(e) Approximately in 1/3 children with spastic diplegia delay of intellectual development, hearing and sight disorder are observed. In more than 2/3 of children there is defect in speech. 1/4 have epileptic attacks.

Chorea minor – one of the types of the rheumatic destruction of the CNS, may grow after tonsillitis, may be provoked by influenza, may be one of the first signs of rheumatism and may grew in the healthy child. The disease destroys different parts of a brain, mainly its sub cortical structure heart and joints are also affected. The disease grows slowly. Firstly, the mood is changed. In quiet and balanced children, irritation, and susceptibility, appears he gets tired quickly, he cannot concentrate, and his memory is affected. Then under such symptoms unconditional movements start to occur. They are met that intensive unconditional movements in the face gives a picture of grimace.

Forced, independent from the will movements occur in hand. New the writing is disturbed, the alphabets are meet equal.

Swift unconditionally the hand is thrown, the trunk is turned now articles fall on the floor, noises are produced by the vessels. Thus, patients of chorea irrespective of distribution of psyche are subject to injury. Therefore it is necessary to differentiate between chorea minor from common pranks and one should consult a doctor at the appropriate time.

Chorea minor is curable effectively if treatment is started as early as possible.

Epilepsy. The basis of its origination lies in the state of the brain i.e. in its readiness to produce tremors. It may be due to the hereditary factors or it may be caused by a trauma of the head, meningitis, encephalitis, etc.

The most common form of epilepsy is a big attack. During such a attack the patient looses consciousness, falls, has strong convulsions (phase of tonic convulsions); the clinic convulsions start during which the breath becomes facial cyanosis occurs the saliva is seen frothing from mother corners, sometimes the patient, unconsciously urinates. After the attack a phase of none periodical sleep takes place.

Another less known form of this disease is one in which there is a sudden loss of consciousness. At the beginning of these attacks the patients speech slows and breaks, he feels drowsy, his vision decreases to one point. Such state lasts for 2-3 seconds then the patient regains consciousness and restarts conversing from that point from which he left off. However during such attacks the patient looses the capability to hold things cups fall and break.

Such patients with sudden loss of consciousness also show a sudden loss of muscular tonicity and so they may fall consciousness however returns very quickly.

The patient does not succeed in understanding the cause of his fall. Most of the time he asks the people around him: "why did you push me?". This proves the shortness of the attacks. Such paradoxical states need immediate medical help serious treatment.

However there are other booms of epilepsy which are little known to medical personal but which show up in behavior like tricking grimacing and defective behavior. The child usually does some actions unconsciously ex. hits his hands on his chest, shakes his head etc. This might be stereotypical form of a particular phase.

Sometimes epileptically seizures might be shown up by the children's who run away from home. This children always cross the roads carefully. Sit in buses, buy tickets and usually normally answer the questions.

Later when the patient comes to himself he might find himself in a totally different place for away from his home. They cannot explain their presence so far away from home.

Such episodes are not linked with the situation at their homes. So a child's strange behavior, his running away from home must be taken seriously *and* the child should be shown to a doctor.

It must be mentioned that serious organic diseases of brain can also lead to strange behavior and attacks.

The rise of the temperature or unbalanced coordination of movements. Common cough and cold, leads to depression. The depressive mood of the child should be correctly read and meticulously analyzed and in difficult cases should be consulted with neuropathology doctors. Care should be aimed for the betterment of the disease. Residual pediatric encephalopathy has its own etiology and pathogenesis which leads to the destruction of the brain.

A spinal cord injury is not less important and not rarely found than cerebral injury. Path anatomical studies show up the incident of spinal cord disease in 70-80% cases (hemorrhage, ischemia and others). The maximum percentage of spinal cord injury has been observed after birth in breech presentation. In such cases, maximum stresses strike on swelling of cervical and lumbar vertebrates. The injury of the lower part of spinal cord is the result of weight on the lower part of spinal cord (in breech presentation).

The clinical symptoms can differentiate in polymorphic type.

Palsity. Shows up as hypotonic of muscles later hypotrophy and hyporeflexity.

Duchenne-Erb's palsy. This is the commest type when the 5th and the 6th cervical root are injured. It results the proximal paralysis and destroy the bronchial function. It causes the arm lie on the side with extension of elbow, pronution of the fore arm and flexion at the wrist (waiter's tip).More reflex and biceps jerles are absent on the affected side.

Dejerine- Klumpke's palsy: It is due to the affection of the lower cords of the plexus involving the 7th and the 8th cervical or even the 1st thoracic nerve. Clistal paralysis - destroy clistal parts. There is paralysis of the muscles of forearm with wrist drops and flaccid digits. The arm is flexed at the elbow, the wrist extended, the hand flaccid and the tinges flexed. When the first thoracic have is involved, there may be homolateral with small pupil due to sympathy nerve involved but the sensitivity is protected in this case.

Brachial palsy. Either the nerve roots or the trun's of brachial plexus are involved. The damage of the nerve is due to the stretching or effusion or hemorrhage inside the sheath. Tearing of the fibres is used. Arm is pronated and rotated inside the wrist. There is flexion of the forearm, the wrist and flaccid digits.

Total paralysis. involves the affection of the C5-C8 nerve. Prognosis is usually bad. Discernation - hanging down of hands. In this case mono reflex is absent. In Erb and Duchenne palsy. Hand is separated from trunks folding like

In Klumpke and Dejerine palsy - Bobkin and Robinson reflexes is lost. In reflex paresis there is the absence of head bending. Symptoms selchania in brachial joints are seen like dislocation. In connection with hypotonic symptom "shortened neck". Many give symptom Cloda-Bernar-Gornira. Head is not able to stand in erected position which shows cervical paralysis.

Clinical character of the spinal cord injury results the locomotors disorder.

When the C4 is injured the respiratory centre is affected (n. frenicus).There respiratory disorders may be the single sign of the injury. During this atelectasism may occur in a new born. In X-ray high standing of the diaphragmatic cupola, its limited movements can be seen, respiration becomes paradox.

During heavy injury of the spinal cord, the disorders of the pare organs can be noted retention of urine just after birth, obstinate constipation.

Crooked neck appears to be another symptom of the injury of the spinal cord if the vertebral arteries are injured, the head inclines to that side reflectively for the better blood supply to the brain.

The injury of the spinal cord on the level of the lumbar region is less dangerous and is not life-threatening, but may proceed with a complex of neurological disorders. The main clinical disorder is the lower paraparesis. In the first week the disorders are quickly recovered. However it should be noted that if a child is hanged by his shoulders, the legs hang like wattle.

The spasmodic syndrome in a child is encountered very often: 22 in 1000. 25% children, carrying spasmodic syndrome in an early age suffer from epilepsy. The cause of spasm may be cerebral, may be central.

Spasms, which occur just after birth arise due to the cerebral reasons, due to the disorder of liquordymaics, they may not occur further. Those, which occur at the 7th or the 8th day are bad prognostic signs. Mostly the spasms arise due to the acute, respiratory disease, meningitis, encephalitis.

In children the spasmodic syndrome may occur differently. In a new born inclined spasms are observed. After this spasmodic readiness occurs - shaking, shuddering, thermo, which points to the fact that the spasms may occur again. If after a long period of time the pause spasms occur again, then they usually won't disappear.

The spasms may occur during hyperthermia, which always does not results in epilepsy.

The most common reasons of the spasmodic syndrome are labor trauma, the disorders of the electrolytes metabolism hypocalcaemia in the premature babies.

Prophylaxis of the neural diseases. Care of the child with the neural diseases.

The golden rule of medicine says, a prevention is better than cure. It is true in the case of children with the pathology of the nervous system.

Physical growth, absence of chronicle diseases, the ability to resist infection - this is the base, without which the normal growth of a child is impossible.

The base of a child's health lies in the health of his parents. Long intoxication (specially alcoholic / diseases, for y example syphilis) affects the health of a child even before his birth. The period of pregnancy, its normal course is very important for the psychological health of the would be child. Correct, rational diet, walk in fresh air, not less than 8 hours of sleep, absence of emotional pressures and psychological trauma are the main factors.

After birth:- hygienic rules must be strictly abided to, any contacts with patients having any infectious diseases are to be avoided. Diet, nutrition, sleep and walking times must be carefully charted and maintained.

An important role in causing a child is keeping the child away from ill people. It is also necessary to distinguish and study any chronic disease of the nose and throat, digestive tract, liver, kidneys and lungs. It is also necessary to look out for worms and where needed treat them. Starting from an early age children should be treated with airing, sponging with cold water and ending with a sun bathe or bathing in natural water-bodies.

Attentive care should be given to children who have undergone cranial injury or had serious infectious diseases. The acute period may end well and the child may be on

the recovery but for a quite long time after this he remains lethargic, tired, has low memory, quickly changes tempers and is quick to cry.

Often children who worked well at school after the illness show academic deterioration. The reason of this is not just because he has fallen back due to hussed closes because of illness but because of the effect of the disease on his CNS, too.

So during this period it is necessary to let the child rest and relax. However careful attention must be given so that he eats regularly, sleeps well and goes out for walks.

The most important thing is to console the child that his falling back because of his illness and is just for a short time - that everything will be well soon. He should be treated kindly and tactfully.

In any case, apart from taking the given medications, the child should be in a peaceful surroundings and should be treated kindly and attentively at school and at home and especially at the hospital.

Materials to activate the students during lecture:

Questions :

1. Anatomical and physiological features of nervous system in children.
2. Nervous-mental development and its estimation**.**
3. Semiotics of major nervous system diseases.

Materials of methodical software of the lecture:

- Auditory
- Software
- Illustrations

The materials for self study of students. (Literature for the student.)

e) For the topic of the current lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 149 – 204; 767 – 769; 778 – 779.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

f) For the topic of the next lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 205 – 224.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

Literature used by lector during preparation the lecture:

19. Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – 808 pp.
20. Partha,s Fundamentals of Pediatrics. Ajanta offset &Packagings Ltd., New Delhi.-2013.- 782 pp.
21. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.
22. Vicky R. Bowden, Cindy S. Greenberg. Pediatric nursing procedures. - Lippincott Williams & Wilkins. - 2011. - 822 pp.
23. Pediatric Nursing Procedures. Vicky R. Bowden, Cindy S. Greenberg. - Wolters Kluwer Health.- 2015 -728pp.
24. Nelson. Essentials of Pediatrics. Sixth Edition. Canada: 2011. - 831p.
25. Robert M. Kliegman. Nelson Textbook of Pediatrics 19th ed. – W.B. Saunders Company, 2011. – 2680 pp.
26. Pocket Book of Hospital Care for Children. – World Health Organization, 2013. – 438 pp.
27. Nelson Textbook of pediatrics / R. M. Kliegman, B. F. Stanton, J. W. St Geme III, N. F. Schor ; ed. by R. E. Behrman. – 20th ed. – Canada : ELSEVIR, 2016. – 5315 p.

Lecture № 5

Topic: «Anatomical and physiological features of skin and its derivatives, subcutaneous fat in children. Semiotics of diseases» – 2 hours.

The actuality of the topic: Knowledge of Morfological and functional peculiarities of the skin and its derivatives, Peculiarities or the subcutaneous fat structure, Semiotics of its diseases, Semiotics of its main affections, Semiology of skin diseases and affections of the fatty, ability to investigate a patient is an important part of diagnostics and treatment.

Aims of the lecture

2.1. Practical (training) aims:

-Familiarizing students with the Morfological and functional peculiarities of the skin and its derivatives, subcutaneous fat in children;

- to tell the students knowledge on the main diseases of the skin and its derivatives, subcutaneous fat;

- to tell the students knowledge on the Skin , subcutaneous fat demadges.

2.2. Educational aims: - development of clinical and scientific thinking in students;

- bringing to the students the understanding of significance of the national clinical, scientific and pedagogical schools in development of the problems mentioned in the lecture;

- to teach the students deonthology and medical ethics.

formation in students a responsive treatment towards children, using a clinical observation of the children with abnormalities of a nervous regulation.

Plan and structure of the lecture :

№№	The main steps of the lesson	The aims in the levels of learning of the material	Materials of methodical software	Time (in minutes or %) of a hole lesson
1	**2**	**3**	**4**	**5**
I.	*The preparatory level*	1	The presentation, Auditory	5%
1.	The defining the purpose			
2.	Formation of the positive motivation			
II.	*The basic level.*	*II.*		85-90%
3.	The presentation of the lecture by plan 1. Anatomical and physiological features of skin and its derivatives, subcutaneous fat in children. 2. Semiotics of diseases.	*II.* *II.*	Illustrations	
III.	Final level.	*1.*		5%
4.	The conclusion of the lecture.			
5.	Answering questions.	*II.*		
6.	The task for self-control		Literature, questions, tasks.	

The content of the lecture

The text of the lecture:

Anatomical and physiological features of the skin. Semiotics of diseases. Morfological

and functional peculiarities of the skin and its derivatives. Peculiarities or the subcutaneous fat structure. Semiotics of its diseases. Semiotics of its main affections. Semiology of skin diseases and affections of the fatty tissue. Nursing children with skin diseases.

Skin, its components and derivatives.

The skin being the outside covering of the whole body is a compiex organ, performing important functions, ensuring normal vital activity of the organism. It protects the organism against harmful external influences, participates in the thermoregulation and metabolism, and executes the secretory and excretory functions, as well as a number of other important functions.

From the anatomic point of view the skin consists of three components, it is formed by epidermis, derma and subcutaneous fatty tissue.

Epidermis and its derivatives (hair, nails, sebaceous and sweat glands) develop from the outer embryonic layer − ectoderm, and the derma and subcutaneous fatty tissue − from the middle embryonic layer − mesoderm.

Embryogenesis of the skin.

At early stages of embryogenesis the skin consist of one layer of polygonal cells. In the 5-7th week of prenatal period the skin acquires two-layer structure, in which epidermis and derma are distinguished. The border between them is the basal membrane, which looks smooth.

In the third month the spine-form layer develops, therefore foveas and spines develop between the epidermis and derma, forming the unique skin pattern.

Granulous and corneous layers develop in the five month old fetus only on palms and soles, and by 7 month of prenatal development all skin layers, except the shining one, are formed.

Nails are formed in the third month of intrauterine period, grow slowly, and reach the finger tips by the last month of pregnancy.

In the 6-8th week of the intrauterine period of the development the germins of the hair are formed, and in the 3rd-4th month the fluffy hair (lanugo) develops from them. Alongside with the formation of hair follicles, the germins of sebaceous glands appear.

Germins of the sweat glands appear on the surface of palms and soles during the 4th month of prenatal period of life. The differentiation of their structure proceeds up to the 7th month, but secretion activity does not begin before the birth.

Clinical significance in the forming of the developmental anomalies.

The significance of these data consists in that they can explain the mechanisms of the forming of developmental anomalies in the skin and subcutaneous fatty tissue.

If the early development of epidermis or derma (or subcutaneous fatty tissue) is disturbed, local inherent defects may occur, that are a special kind of fetopathy, caused by the mother's exposure to bacterial or virus infections, intoxications or influence of radiation.

Immediately after birth local absence of skin, and sometimes subcutaneous cellular tissue is observed in the child. On examination these defects look like ulcers, the surface of which can be covered with crusts, and scars can develop later. The affection can be located in different parts of the body. As often as not the inherent absence of the skin can be accompanied by other disturbances of the development.

Morphological structure.

By the moment of birth at morphological examination it is visible, that the skin consists of two layers: epidermis and derma (skin proprea). These layers of skin have a number of

morphological and biochemical features, because with age the differentiation and growth of cell structures of the skin occurs.

Epidermis.

Epidermis in children, especially newborn infants is thin, gentle, and podgy. Its thickness is 2,5-3 times less, than In adults. The level of the development of epidermis in infants is uneven. It is especlally soft and thin in the area of face and skin folds, and on the palms and soles it is more morphologically mature.

Epidermis in the children as well as adults, consists of five layers: a) basal (embryonic layer); b) spinous; c) granulous (keratohyaline layer); d) shining; and e) corneous. However the specified layers of epidermis in children differ from those in adults and have some peculiarities.

1. The basal or embryonic layer consists of one row of cylindrical-shaped cells of smaller size, than in adults. The cytoplasm of these cells is basophilic owing to the large quantity of RNA. Among the cells of the basal layer there are melanocyts producing melanin. However the production of melanin is reduced, and up to six months melanin is practicalIy absent.
2. The spinous layer consists of two or five rows of cells, but their size is considerably smaller, than in adults.
3. The granulous layer is thinner, than in adults, and consists of one or three rows of cells, it is weakly expressed. It 1s well defined only on palms and soles, where as in adults the granulous layer is found in every part of the skin. The keratohyalyne pigment, that is usually found in the cytoplasm of the granulous cell layer and gives the skin a pale colouring, is absent in infants.
4. The shining layer in children is usual hardly defined on palms and soles. On all other surface of skin covering this layer practically isn't found. Owing to this fact some researchers don't even designate it as a separate layer of epidermis. The cells of the shining layer consist of solid dense substance, called eleidin, that reflects light intensively.
5. The corneous layer is weakly developed, thin, is presented by one or three rows of flat anuclear cells, resembling tile-shaped platelets. The connection between the cells of the corneous layer is weak, they are loosely arranged and easily get disattatched and separate from the skin.

The clinical significance of the specified features consists in the following: first, as far as thin, friable granulous layer easily gets disattatched and separated, in intertrigo develops very frequently children, manifested in the maceration of skin, occurrence of erosions and madescent sores. This is dangerous, it may cause the development of bacterial infections and occurrence of sepsis; secondly, low protective properties of the epidermis is the reason of frequent development of candidamycosis.

Dermis.

Dermis is the fibrous basis, containing blood and lymphatic vessels, fatty and sweat glands, nervous endings and hair papillae, i.e. it is a support for the derivatives of epidermis, located in it, for smooth muscle fibres, vessels and nerves.

By the moment of birth two layers, papillary and reticular are distinguished in the dermis, although they are not very distinct yet. Dermis in children has physiological and structural differences from the dermis in adults.

First, the papillary layer of the dermis is expressed weakly, or even is absent, especially in the immature newborn. It becomes morphologically formed oniy by 6 years.

Secondly, dermis in children preserves the embryonic structure, contains much cellular

elements and little fibrous structures. High biological activity of labrocytes is observed, and this explains special features of skin allergic reactions in children.

Thirdly, the fibrous structures of the dermis are differentiated insufficiently. There are few collagen fibres, they are considerably thinner, than in adults and consist of friable brunches. The elastic fibres are weakly developed. Only argyrophyllic fibres are well expressed, later they turn into collagen fibres.

In-fourth, the dermis in children differs also by biochemical features:

a) content of water in the children's dermis is higher, than in adults, it is hydropic. In newborns water content in the dermis makes 80 % and by one year − 50 %, and the skin contains 10-17 % of all water of the body, where as the skin of adults contains onIy 6-8 % of all water;

b) the dermis of newborns and children of early age has a higher level of acid mucopolysaccharides, containing hyaluronic and chondroitinsulfur acids. And in children the activity of hyaluronidase is increased, that read to its higher permeability.

In-fifth, in children the basal membrane, separating dermis from epidermis is immature. Because of this in case of various impacts or diseases the epidermis is easlly separated from the dermis, this explains possible occurrence of epidermolysis − exfoliation of epidermis with the development of blisters in various skin diseases. So, in the early congemital syphilis the so called syphilitic pemphigus develops, and the blisters are located predominantly on palms and soles. In the children of other age groups and in adults such changes are not observed in syphilis.

In the newborns the neonatal pemfigus (pyococcus pemphigoid) occurs as a reaction to the staphylococcus infection and surface blisters appear.

Clinical significance of the skin features.

The anatomical and morphological peculiarities of the skin promote peculiar course of many skin diseases in children. For example, in scabies the scabious elements in infants are frequently located on palms and soles, and they can also be found on the face and hairy part of head in the form of spots or blisters. ln adults such localization of scabious elements is never observed because of the physiologic thickening of the corneous layer of skin on palms and soles. Ignorance concerning the peculiar clinical picture of scabies in babies sometimes results in diagnostic errors.

Functions of the skin.

The skin executes important physiological functions, which in the newborns and babies of early age have some specific features, caused by the difference in anatomical and histological structure of the skin in children.

The protective function of skin is expressed insufficiently, because the epidermis is friable and soft, the corneous layer is thin, the basal membrane is immature, and the elastic and collagen fibres are morphologically insufficient. As the result, a child's skin is susceptible to mechanical, radiation, thermal and chemical damages, as well as can serve a place of infection penetration. This is especially important, because the skin is well visualized, so the microorganisms can spread very quickly.

However it should be emphasized, that the skin has bactericide properties, owing to the acid reaction of its surface (pH 4,2-5,6), created by the fatty and sweat glands. ln the newborn infants pH of the skin makes 6,1-6,7, i.e. it is close to neutral, and as the result provides most favorable environment for the growth of microbes.

Besides, the bactericide properties of the skin are connected with the existence of a water-

lipide covering on the surface of the epidermis, which contains small molecule free fatty acids and renders suppressive action on the growth of pathogenic microflora. Hence, the skin is something like a “personal sterilizer”. In the newborn infants and babies the systematic maceration of the skin with urine and excrements, as well as active secretion of sweat glands promote the dilution of the water-lipid covering. NaturalIy, it considerably reduces bactericide properties of the skin and its protective functions.

The thermoregulative function in babies is rather imperfect. lt is caused by that the process of heat-producing is not active enough in children, and heat emission for the account heat irradiation, conductabitity and sweating is very intensive. The high intensity of heat emission can be explained by thin skin, a big number of superficially located blood vessels, in the state of physiological dilatation. Besides at the cooling of the skin its surface doesn’t decrease, i.e. the children don’t develop the so called “goose flesh”. This is connected with immature muscle fibres of the hair follicles, which make for the phenomenon of “goose flesh”.

The breathing function of the skin in children is well expressed, that adds to breathing function of the imperfect lung apparatus. The intensity of the skin breathing in children is very great. This function in newborns is 8 times more intensive, than in adults. Well developed function of skin breathing in children is caused by soft and friable epidermis, its cells have partial keratosis; there is a great number of blood vessels in the dermis in the state of physlological dilatation.

Owing to the specified features of the skin,if a child has pneumonia, additional gas metabolism and oxygenation can be provided by his skin, and the child’s breathing can be improved. But it is necessary to keep the child’s skin clean. It is impossible toput ointment or animal fat on the child’s skin, as parents often do.

The resorption function of the skin is well developed, because the epidermis is thin, the skin is rich in blood vessels, contains a big number of fatty glands and hair follicles But it is necessary to emphasize, that resorption depends on a chemical nature of substances, that are subject to resorption. Substances, dissoluble in fats (iodine, chloroform, phenol, boracic and salicylic acids, mercury, sulfur, etc.) are easily absorbed and resorbed. This happens because substances are resorbed mostly through the fatty glands and hair follicles.

Taking this into account, it is necessary to be cautious with the application of ointments, creams and pastes, containing medicinal substances, that are easily resorbed. lt also concerns the application of hormones in ointments, because their action can be not only local, but also general, and the suppression of activity of the hypophysis and adrenal glands may occur.

The buffer function of the skin is not well developed, because in the newborn infants and children of early age pH of the skin is equal 6,7 and is close to neutral This circumstance causes insufficient neutralization of acids and alkalis, therefore the child skin is very sensitive to their damaging action.

The pigment producing function of the skin consists of the formation of the melanin pigment by the epidermal melanocytes. Melanin is formed from tyrosine, which slowly, under the influence of tyrosinaze, connected with copper, and UV beams, turns into 3,4-dihydrooxyphenilalanine (DOPA), and then under the impact of DOPA-oxydase - into melanin. Ir the newborn infants and babies the processes of production of melanin is immature. In the children skin the quantity of pigment granules is smaller than in adults. The significance of melanin consists in that it has an ability to absorb the UV sun beams, and thus to protect the skin from the damaging action of solar beams. Thus, it is necessary to be

extremely careful about the exposing of the child organism to the action of solar beams. We do not recommend to expose children to direct sun rays.

The vitamin producing function consists of the formation of Vitamin D and other bioiogically active substances. This function is well developed in children.

The secretory function of the skin in children is complex and various. The skin secrets keratin, squalen, calcium, phosphorus and other substances.

The formation of keratin, that begins in the embryonic period during the formation of the ectoderm, is expressed well in children. Owing to high secretion of keratin, which provides the keratosis in the epidermis, high regeneration ability of epidermis is observed in children.

The epidermis cells secret also a fat-like substance − squalen, one of the components of the water-lipide covering, which together with cholesterine makes 10-20% of the surface fatty membrane. This ability of the epidermis is expressed reasonably well.

Besides, the sweat glands in children produce more calcium and phosphorus, and the secretion of fatty and apocrine giands is maximal in the adolescent period.

The metabolic function of the skin is well developed. It explains high regenerative ability of the epidermis and dermis in children.

The significance of the skin in the maintenance of the homeostasis is especially high due to its high accumulative ability. In particular, a newborn's skin can accumulate water and mineral substances, owing to hydrophylity and lability of the osmotic state of the dermis fibrous components. This often causes the occurrence of exudative diathesis and other similar skin diseases.

The great quantity of intracelluiar and extracellular water, as well as mineral substances, in a child's skin supports high intensity of metabolism and realization of ferment processes in the epidermis and derma.

It should be emphasized, that in a childs skin the activity of ferments, participating In the proteins, fats, carbohydrates, mineral salts and water assimilation is insufficient. This explains the high frequency of dystrophic pathologic processes and occurrence of infIammatory diseases in the skin.

The receptor functions of the skin are reasonably well developed, because it contains numerous nervous endings, and thus is a peripheral analyzer, perceiving exogenic and endogenic stimules and sending them to the central nervous system. Therefore, the skin plays an exclusive role during the adaptation of newborn infants and children of early age to the environment conditions. Almost all the reflexes in a newborn baby are provoked by touching its skin. The skin of hands, soles, and face is the most sensitive to touches.

Epidermis appendages.

The epidermis appendages − fatty and sweat glands, hair and nails − also have a peculiar structure and function in newborns and babies.

There are two types sweat glands: eccrine (merocrine) and apocrine. The sweat glands of eccrine type are immature, because of their ducts end in the epidermis and are formed only by 3-4 months, and the process of their formation is finished after 7 years. The excretory ducts of the sweat glands of eccrine type in children are usually straight, instead of being corkscrew-shaped, as in adults. Therefore the sweat glands of eccrine type begin to function at 3 or 4 months. OnIy in the case of pathology (rickets, for example) the disposition to sweat occurs, especillIy in the skin of the hairy part of the head and on the forehead, because on these parts the sweat glands maturate first. Later these glands maturate in the skin of breast and back.

The sweat glands of apocrine type were preserved in human beings only in armpits, in the area of breast nipples, genital organs and anal orifice. In the children of early age these glands don't function at all. The beginning of their activity appears to 8-10 years.

Fatty glands in children are distinguished by large size, unicameral, superficial location, and a great number of them are located in the area of face and hairy part of the head.

Directly before birth the secretion of the fatty glands amplifies, and the products of their secretion participate in the formation of vernix caseosa, of greyish-white or yellowish colour, abundantly covering the skin of a newborn (vernix caseosa).

Sometimes at birth congestions of the secret of fatty glands can be seen as a kind of whitish or yellowish spots, slightly elevated over the surface of skin, by size of a millet grain, more often on the nose, less often on the forehead and cheeks. These congestions are called milia.

As the child grows, the intensity of secretion decreases and some of the glands atrophy, but in the period of sexual maturation the secretion increases again.

The excretory function of the skin is executed by the secretion of fatty and sweat glands. The products of estherification of lipoids, cholesterine, neutral fats, carbohydrates, as well as nitrous, sulfates and phosphorus metabolic products are excreted by fatty and apocrine glands, especially in the adolescent period. Despite of the immature state of fatty glands, this function is developed well.

Sweat glands in children practically don't function up to 3 or 4 months. Therefore the excretion of organic and inorganic substances (such 85 urea, ammonia, uric acid, salts of sodium potassium, phosphates, and sulphates) with sweat is insufficient.

It is important to emphasize, that overheating and excessive wrapping in newborn babies up to plentiful sweating. It causes the expansion of excretory ducts of sweat glands and small retention cysts can be formed on the baby's skin (miliaria).

The skin excretes many medicines, for example bromide, iodine, sulphur, iron, salicylates and some others, therefore the intake of these preparations can cause rash.

The hair begins to grow in the embryonic period. It is so called primary hair − lanugo, that doesn't have shafts in it. Soon after birth it falls out and is replaced with constant, secondary hair. Usually in the immature children the lanugo is more plentiful.

After the sexual maturation the setiform hair begins to grow in the armpits, on the pubis, in the area the anal orifice, and in boys − on the face.

Nails are well developed by the birth. They are very soft, elastic and contain a lot of water. In the first days after birth the growth of nails is slowed down. This happens because under the nail matrix the epidermal bends become thicker, infringing the blood and lymph circulation.

Clinical significance.

Some of the described features of the structure of the skin appendages have important clinical significance.

For example, in infants, especially newborns, staphylococcus ostioporites − vesicopustulosis − occur rather often. They are abscesses of the eccrine sweat gland ducts. The occurrence of this disease is explained by staphylococcal infection of the skin and type of straight the ducts of sweat glands. Therefore the infection easily penetrates through the straight duct and causes inflammation. The center of inflammation is located in the ducts of eccrine sweat glands.

Semiotics of the main skin diseases.

The semiotics of vesicopustulosis consists of the occurrence of multiple small-sized (miliar) pustules, surrounded by a ring of hyperemia, on the skin of the buttocks, hips, lower part of the abdomen and hairy part of the head. Later the vesicles burst and surface erosions develop, then they get dry and crusts are formed in their place, without following pigmentation. No marks remain on the skin after vesicopustulosis.

In the adolescents and adults staphylococcus infection of the apocrine sweat glands (hydradenites) occur quite often. In the newborn infants and children of early age the apocrine glands, as known, do not function, and consequently hydradenites are not observed in this age. As the hair follicles are immature in the newborns and infants, they don't have also furuncules, but they can develop.

Pseudofurunculosis (Finger's) – is plural purulent inflammation of sweat glands in newborns and infants. The illness begins with the inflammation of the mouths of the hair follicles and small pustules occur, surrounded with a circle of hyperemia. Later the process spreads deeper and passes onto the sweat glands.

Gradually an abscess is formed, which opens with discharge of puss and blood. Small hardly noticeable scars remain on the skin after healing of abscesses. Pseudofurunculosis differs from typical furunculosis by absence of a central necrotic shaft.

A very serious skin disease in the newborns caused by the golden staphylococcus infection is the neonatal exfoliative dermatitis (Ritter's disease). This disease has a very acute onset, sometimes in the first days after birth. The focus of the affection, in the form of erysipelas-like limited redness of the skin is located around the mouth and quickly spreads to the skin of the head, body and extremities. The skin becomes crimson-red, frequently with bluish shade. Later cracks, maceration and exfoliation of epidermis occurs, the epidermis comes off in layers. The skin of newborn looks like was burned. The Nikolsky symptom is often positive − epidermis is removed at the slightest touch of the skin. Usually no scars remain on the skin.

Exudative diathesis is the manifestation of the allergic tendency of a child's organism. Its typical attributes are: gneisis (fatty seborrhoea scales on the head), the "milky crust" (limited redness of the skin on the cheeks, with the development of white scales, and sometimes vesicles), intertrigo in the skin folds, erythematose and papular or vesicular rash on the skin of the face, extremities or body.

In the most children these skin changes are accompanied with itching, causing anxiety, irritability, sleep disorders, scratches on the skin and its secondary infection. The children with exudative diathesis also suffer from changes in the mucous membranes: "geographical tongue", allergic rhinitis, conjunctivitis, and other manifestations.

As a result of medicinal allergy an epidermal toxic necrolysis (Layel's syndrome) can develop. The onset of this disease is acute, usually in 10-24 hours after the intake of the medicine. Usually the progress of the disease is instantaneous, it manifests in the damage of mucous membranes (of the mouth cavity, lips, nasal part of the pharynx, larynx, trachea and bronches, esophagus, digestive tract and others), and the skin. The skin of the large natural pleats around natural of apertures and on sites, subject to traumatization is affected most seriously. The rash is polymorphic in the form of erythema, papules or blisters, that link with each other and form extensive erythematous-edematic zones. Soon on this background large (up to 30 centimetres) vesicles develop, with transparent serous or serous-hemorrhagic contents. After these vesicles opening extensive zones of maceration and necrosis are formed. The Nikolsky symptom is distinctly positive.

Subcutaneous fatty tissue.

In newborns and babies of early age subcutaneous fatty tissue has a number of specific features:

1. The fatty layer in children is relatively thicker, than in adults In adults it makes not more than 8% of the body weight, whereas in the babies − not less than 12%.
2. Adipose tissue is absent in the cavities, omentum and retroperitoneal space, and as a result the organs have great mobility and visceroptosis can easily develop.
3. ln the adipose tissue area of embryonic tissue are preserved, that has fat accumulating and hemopoietic functions.
4. The adipose cells are not enough differentiated, morphologically immature, like embryonic ones. They are small in size, and their nucleus are rather large. With age an increase of their sizes and reduction of the sizes of their nucleus is observed.
5. In a structure of the adipose cells there are more hard saturated fatty acids (palmytic and stearic) and less unsaturated fatty acids (oleic and others).
6. There is a quite big quantity (from 1 up to 3% of the body weight) of specific adipose tissue, called brown adipose tissue. It has a large contents of mytochondria, coenzimes and cytochrome, it is microgranular and has very intensive blood circulation.

The brown fatty tissue in newborns is located in the interscapular and axillar zones, in the areas of the thyreoid gland, pericardium, around the esophagus, kidneys, adrenal glands and in the mesenterium. Its main function consists of maintenance of non-contractile thermogenesis (i.e. heat production, not connected with muscular contractions). If a child is exposed to cold, the brown fatty tissue provides oxydation of fatty acids, that is accompanied with intensive heat production.

7. The accumulation of fat occurs in the definite order: face extremities- body (breast) – abdomen; disappearance (or reduction) of the adipose tissue − in the opposite order.
8. The fat accumulation in various periods of childhood occurs uneven: most intensively − at the age below 6 months, a little less at 9 months, and then more intensive fat accumulation is observed 7-10 years.
9. Sex distinctions in the accumulation of adipose tissue are most pronounced in a period of sexual maturation. In girls fatty tissue accumulates most intensively in the area of pelvis and extremities, and in boys - on the body.

Clinical significance.

These features of the subcutaneous fatty tissue in the children have important clinical significance.

A number of diseases and pathologic states can be explained by features of its morphologic and physiologic structure.

For example the neonatal adiponecrosis occurs after pathologic labour or when the forceps are used, is associated with the big quantity of hard fatty acids, especially palmitic, in the subcutaneous fatty tissue of the newborn babies, its content is 4 times more, than in adults. Adiponecrosis is the local necrosis of the subcutaneous fatty tissue, that appears in the first or second week after birth, and is usually occurs on the back, buttocks, shoulders, head and less often on extremities and face in the form of solid limited infiltrations. The skin over the infiltrates becomes of a purple-red colour, and then gradually grows pale. The disease has no unfavorable consequences.

Peculiar chemical structure of the adipose tissue (a lot of hard fatty acids), immaturity of the nervous regulation of the water metabolism and increased permeability of the skin lead to

frequent enough occurrence of sclerema and scleredema in the newborn infants as a result of over cold.

Semiotics of the main diseases of subcutaneous fatty tissue.

Sclerema occurs in the 3^{rd}-4^{th} day of life like diffuse infiltration of the skin and subcutaneous tissue on the back surface of legs, face and neck. It can spread to the hips, buttocks, body and upper extremities. The skin look like a hard, tense and tightly stretched shell, cold by touch. It is pale, with a yellowish or cyanotic shade, it is impossible to take it as a fold. Pit is not formed after pressing. The general state of a child gets worse.

Scleroedema is characterized by sudden occurrence of solid edema of the skin and subcutaneous tissue on the face, neck or lower extremities, and then on the body. The skin on the affected places is paste-like, pale, it quickly impresses, becomes tense and cold by touch, it is painful and after pressure a pit forms.

Materials to activate the students during lecture:

Questions :

1. Anatomical and physiological features of the skin and subcutaneous tissue in children.
2. Semiotics of diseases of the skin and subcutaneous tissue.

Materials of methodical software of the lecture:

- Auditory
- Software
- Illustrations

The materials for self study of students. (Literature for the student.)

g) For the topic of the current lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 205 – 224.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

h) For the topic of the next lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 225 – 246.
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Lecture № 6

Topic: «Anatomical and physiological features of respiratory system in children. Features of embryogenesis of respiratory system organs and anomalies of their development. Semiotics of defeats and main diseases of respiratory system in children. Syndromes of respiratory disorders and respiratory insufficiency, main clinical manifestations» – 2 hours.

The actuality of the topic: Knowledge of Morfological and functional peculiarities of the skin and its derivatives, Peculiarities or the subcutaneous fat structure, Semiotics of its diseases, Semiotics of its main affections, Semiology of skin diseases and affections of the fatty, ability to investigate a patient is an important part of diagnostics and treatment.

Aims of the lecture

2.1. Practical (training) aims: The development of the respiratory system encompasses three distinct processes: morphogenesis or formation of all the necessary structures, adaptation to postnatal atmospheric breathing, and dimensional growth. The first two processes take place primarily before or shortly after birth. Growth, in contrast, continues after birth at a pace that is generally dictated by the functional needs of the other growing organs and metabolic activity of the animal. The effects of an injury to the respiratory system depend, therefore, not only on the severity and chronic properties of the injury but also on the timing of the injury in relation to the developmental timetable of the lungs. Insults occurring during morphogenesis, for instance, tend to produce severe and irreversible disruptions of respiratory structure and function, often incompatible with survival. In contrast, injuries that take place during later stages of lung growth are frequently reversible and, if not, can be compensated for by the growth process itself.

It's very important for future doctors to know semiotics of damage and basic diseases respiratory system in children and syndromes of respiratory disorders and respiratory insufficiency, basic clinical manifestations. Also for the pediatrician have to well orient in care of children having respiratory disorders and emergency help for lung haemorrhage, haemoptysis and apnoea. To make the diagnosis one should be able to correctly estimate the data got during the investigation of a patient, data of laboratory analyses and then to make clinical analyses of the semiotic symptoms of disease.

Knowledge of the anatomical and physiological features of respiratory system in children, Semiotics of the affections and diseases of respiratory system is an actual problem due to its prevalence, diversity of clinical symptoms, often hidden proceeding with the following progress.

To make the diagnosis one should be able to correctly estimate the data got during the vestigation of a patient, data of laboratory analyses and then to make clinical analyses of the semiotic mptoms of diseases.

2.2. Educational aims:

- development of clinical thinking in students;
- bringing to the students the understanding of significance of the national clinical, scientific and dagogical schools in development of the problems mentioned in the lecture;
- to teach the students deontology and medical ethics.

Plan and structure of the lecture :

№№	**The main steps of the lesson**	**The aims in the levels of learning of the material**	**Materials of methodical software**	**Time (in minutes or %) of a hole lesson**
1	**2**	**3**	**4**	**5**
I.	*The preparatory level*	1	The presentation, Auditory	5%
1.	The defining the purpose			
2.	Formation of the positive motivation			
II.	*The basic level.*	*II.*		85-90%
3.	The presentation of the lecture by plan 1. Anatomical and physiological features of respiratory system. 2. Semiotics of diseases.	*II.*		
III.	Final level.	*II.*	Illustrations	5%
4.	The conclusion of the lecture.			
5.	Answering questions.			
6.	The task for self-control	*II.*		

			Literature, questions, tasks.	

The content of the lecture

The text of the lecture

Development of the Respiratory System

The development of the respiratory system encompasses three distinct processes: morphogenesis or formation of all the necessary structures, adaptation to postnatal atmospheric breathing, and dimensional growth. The first two processes take place primarily before or shortly after birth. Growth, in contrast, continues after birth at a pace that is generally dictated by the functional needs of the other growing organs and metabolic activity of the animal. The effects of an injury to the respiratory system depend, therefore, not only on the severity and chronic properties of the injury but also on the timing of the injury in relation to the developmental timetable of the lungs. Insults occurring during morphogenesis, for instance, tend to produce severe and irreversible disruptions of respiratory structure and function, often incompatible with survival. In contrast, injuries that take place during later stages of lung growth are frequently reversible and, if not, can be compensated for by the growth process itself.

Prenatal development: morphogenesis

In humans the morphogenesis of the respiratory system is divided into five periods. The first, or embryonic period, begins at approximately 4 wk of gestation, when the primitive airways appear as a ventral outpouching on the endodermal epithelium of the foregut. This outpouching divides almost immediately into two main stem bronchial buds, which burrow rapidly into the mesenchyme separating the foregut from the coelomic cavity. The bronchial bud start to branch, first by monopodal outgrowth (secondary branches grow out of a main branch) and then by asymmetric dichotomy (two secondary branches originate from one main branch).

The peribronchial mesenchyme or splanchnopleura plays an essential role in shaping the lungs during the embryonic period. Close contact between this mesenchyme and the epithelium of the bronchial buds is essential for the continued branching of the airways. Although the factors that promote bronchial division are not fully identified, steroid-induced secretion of growth factors by the mesenchymal fibroblasts, specific interactions with acellular components of the mesenchyme, and even direct molecular communications between fibroblasts and endodermal cells across gaps in the basal membrane have been proposed as signaling mechanisms. The interactions between mesenchyme and the bronchial bud endoderm are organ-specific.

The pulmonary vasculature is a mesenchymal derivative. Soon after their appearance, the bronchial buds are surrounded by a vascular plexus, which originates from the aorta and drains into the major somatic veins. This vascular plexus connects with the pulmonary artery and veins to complete the pulmonary circulation at the 7^{th} wk of gestation but retains some aortic connections that form the bronchial arteries. All the supporting structures of the lungs,

including the pleura, the septal network of the lungs, and the smooth muscle, cartilage, and connective covers of the airways, originate from the mesenchyme.

Toward the 6th wk of gestation, at the beginning of the second or pseudoglandular period, the lungs resemble an exocrine gland with a thick stroma crossed by narrow ducts lined by an epithelium of tall cells that almost fill the lumen. The major airways are already present and are in close association with pulmonary arteries and veins. The trachea and the foregut are now separated after the progressive fusion of epithelial ridges growing from the primitive airway. The incomplete fusion of these ridges results in a tracheoesophageal fistula, a common congenital malformation. During the pseudoglandular period, the airways continue to branch until the entire conducting airway system is formed, including the primitive bronchioles that eventually give rise to the air-exchanging portions of the lungs. Simultaneously, the pluripotential cells that line the airways differentiate, starting from the trachea and main bronchi, in a process that also appears to be under some degree of mesenchymal control. They soon form a thinner, pseudostratified epithelium containing ciliated, secretory (Clara), globular, and neuroendocrine (Kulchitsky) cells of neuroectodermal origin. Mucous glands, cartilage, and smooth muscle can be easily distinguished by the 16th wk of gestation.

The diaphragm is formed during this period. Its central tendon originates from the transverse septum, a plate of mesodermal tissue located between the pericardium and the stalk of the yolk sac. Its lateral portions are formed by the pleuroperitoneal folds, which grow from the body Wall until they fuse with the esophageal mesentery and the transverse septum. The fusion eliminates the communication between thorax and abdomen and establishes a barrier to the caudal growth of the lungs. Its failure, usually on the left side, causes the congenital diaphragmatic hernia of Bochdalek. This defect, which is the most frequent type of diaphragmatic hernia, allows the abdominal organs to enter the primitive pleural cavity and interferes with airway and pulmonary vascular branching. The result is severe hypoplasia of the lung, particularly on the side of the hernia. Initially membranous, the normal diaphragm is eventually invaded by striated muscle derived from cervical myotomes.

During the third or canalicular period, between the 16th and 26-28th wk of gestation, epithelial growth predominates over mesenchymal growth. As a result, the bronchial tree develops a more tubular appearance, whereas its distal regions subdivide further to lay the structural foundations of the pulmonary acinus. The epithelial cells in these regions become more cuboidal and start to express some of the antigen markers that characterize cells as type II pneumocytes. Some cells become flatter and can be identified as potential type I pneumocytes by the presence of a sparse endoplasmic reticulum and abundant cytoplasmic glycogen. The capillaries contained in the distal bronchial mesenchyme form a denser network and grow closer to the potential air spaces, making limited gas exchange possibly by 22 wk of gestation.

Between the 26th and the 28th wk of gestation, lung morphogenesis enters its saccular period, during which the terminal airways continue to widen and form cylindrical structures known as saccules. Initially smooth, the internal surface of the saccules soon develops ridges or secondary crests, which originate as folds of the epithelium and peribronchial mesenchyme and contain a double capillary layer. The distance between the capillaries and the potential air spaces narrows further until eventually only a thin basal membrane separates them.

Exactly when the saccular period ends and the alveolar period begins, depends on the definition of what constitutes an alveolus. Formation of alveoli before birth is not a requisite for survival, as demonstrated by the observation that in altricial or no precocious species, such as the rat or the rabbit, alveoli are not present until several days after birth. In the human fetus, the saccular septation initiated with the appearance of the secondary crests continues at a rapid rate so that multifaceted structures analogous to the alveoli of the mature lung can be seen at 32 wk of gestation. In more precocious species such as the sheep and the horse, the lungs contain even more alveoli at birth than are present in humans. There is substantial evidence that the timing and progression of alveolar septation is under endocrine regulation. Thyroid hormones stimulate septation, whereas glucocorticoids impair it in a fashion that, at least in the rat, can be irrevocable (even though they accelerate the thinning of the alveolar capillary membranes). Alveolarization is also influenced by physical stimuli. Both the stretch by the liquid contained in the fetal lung and the periodic distention provided by the action of the respiratory muscles during fetal breathing, for instance, appear to be necessary for the development of the acini. Their absence when the lungs or chest are compressed (as in the case of a diaphragmatic hernia or oligohydramnios) or when fetal breathing is abolished (e.g., by spinal cord lesions) results in pulmonary hypoplasia with reduced numbers of alveoli.

A number of gene families have been identified as being essential for development. The homeo-domain or homeo-box (hox) gene family was discovered first in Drosophila and was later shown to be well preserved in mammals and critical in mammalian organ development, including that of the respiratory system. Hoxa-1, 2, 3, 5, and Hoxb-3, 4,6,7,8 mRNA transcripts have been identified using molecular biologic techniques in branching regions of the developing mouse lung. These Hox genes were differentially expressed in time and space in early lung development, indicating that they play a role in the differentiation, maturation, and proliferation of various lung cells throughout the various phases of lung development. Furthermore, some of these genes seem to be important in distal versus proximal branching and differentiation. Hoxa-2 seems to be tied to a proximal role, whereas Hoxb-6 is involved in distal airway branching. Also of importance is the fact that the expression of some of these genes is controlled by retinoic acid. This may be related to a possible therapeutic role of retinoic acid at later stages of lung development or in injured lungs.

Adaptation to air breathing

The transition from placental dependence to autonomous gas exchange requires adaptive changes in the lungs. These changes include the production of surfactant in the alveoli, the transformation of the lung from a secretory to a gas-exchanging organ, and the establishment of parallel pulmonary and systemic circulations.

As soon as the newborn takes the first breath of air, an air-liquid interface becomes established inside the lungs. Unless the surface tension generated at this interface is reduced, the walls of the air spaces would tend to stick together and collapse, threatening the geometric stability of the lungs. The pulmonary surfactant makes such a reduction possible by forming a hydrophobic lipid monolayer at the very surface of the liquid film that lines the air spaces (see Chapter 97.3). Pulmonary surfactant is a heterogeneous mixture of phospholipids and proteins secreted into the saccular or alveolar sub-phase by the type Π pneumocytes. Its presence is first recognized in characteristic secretory organelles known as

lamellar bodies as early as the 24th wk of gestation However, surfactant lipids, of which the most abundant is phosphatidylcholine, are not detectable in the amniotic fluid until the 30th wk of gestation, suggesting that there is a chronologic gap between surfactant synthesis and secretion. Labor probably shortens this gap because phospholipids are consistently found in the air spaces of infants born before the 30th wk of gestation. Three apoproteins (SP-A, SP-B, SP-C) identified in pulmonary surfactant (a fourth lectin-like glycoprotein, SP-D, has been isolated, but its function and regulation are still poorly under stood) promote the spreading of the surfactant layer and are therefore essential for the effective reduction of surface tension. Apoproteins also appear to be important for the reuptake and recycling of surfactant products and for the formation of tubular myelin (the structures in which surfactant is stored in the liquid sub phase).

Surfactant apoproteins and phospholipids share some, but not all, of their regulatory influences. Glucocorticoids, for instance, increase the synthesis of both apoproteins and lipids; accordingly, their prenatal administration has been used to prevent the respiratory distress syndrome associated with prematurity. Because many actions of the steroids involve direct stimulation of response elements in apoprotein and phospholipid enzyme genes and therefore require messenger RNA production, sufficient time must elapse between steroid administration and birth. Thyroid hormones also enhance the synthesis of phospholipids by a receptor-mediated mechanism, but, unlike the glucocorticoids, have little or no effect on surfactant apoprotein synthesis. Conversely, β-adrenergic agonists and other agents that raise cellular cyclic adenosine monophosphate content increase apoprotein synthesis and phosphatidylcholine secretion into the air spaces but have no effect on phospholipid synthesis. Insulin, hyperglycemia, ketosis, and androgens may have negative effects on the production of surfactant proteins and phospholipids, thus explaining the high incidence of respiratory distress syndrome in infants of diabetic mothers and the slight maturational delay of the lungs of male fetuses compared with female fetuses.

Surfactant proteins and lipids also may play an important role in lung immunity, although the molecular details are not known. Surfactant proteins A and D are lectins (bind to carbohydrates) and belong to the collectins family of genes. These proteins, present in the serum and lungs, stimulate phagocytosis and chemotaxis, produce reactive oxygen species, and regulate the production and release of cytokines by immune cells. Alternatively, surfactant lipids can suppress immunity. It is possible that the ratio between surfactant lipids and proteins is important in regulating the immune status of the lungs. This may be critical in premature infants and in new-born who lack surfactant proteins; knock-out mice with SP-A deficiency have a major problem with infections. The fetal lung is a secretory organ. Throughout gestation, a C1-, K^+- and H^+-enriched fluid is produced in its peripheral air spaces with the help of a Cl pump. The presence of this fluid appears to be important for the development of the acinus because chronic drainage of the trachea in experimental animals results in lung hypoplasia. Fluid secretion, however, is incompatible with air-breathing. Accordingly, and in preparation for birth, lung fluid production decreases slowly at the end of gestation. This decrease, which is accelerated by the beginning of labor, denotes a transformation in the ion transfer activities of the pulmonary epithelium from Cl (and water) secretion to Na^+ (and water) absorption. In experimental animals, such a transformation can be precipitated by the administration of β-adrenergic agonists at doses that result in serum levels comparable to those found during labor. Stimulation of β-receptors is not the only labor-related signal because fluid clearance in the fetal lung is delayed by the Na^+ channel

blocker amiloride but not by β-blockers. After birth, the still substantial amount of fluid left in the lungs is absorbed over several hours into the circulation either directly through pulmonary vessels or indirectly through an already very effective lymphatic system. The cellular elements responsible for fluid secretion and absorption in the lungs are not fully identified. It is obvious that a mature alveolar epithelium is not essential in fluid secretion, which is already taking place before alveoli or even saccules exist. Alveolar cells, in contrast, probably play a protagonistic role in fluid absorption. Type II pneumocytes may be involved because they cover a larger portion of the air space surface in the newborn than in the adult and their metabolic machinery appears to be particularly well adapted 10 active ion transport A number of transporters and channels that have importance on water and solute transport in early life have been cloned and identified in the past decade. Most prominent has been the epithelial sodium channel or ENaC. It is the amiloride sensitive and apical channel that is responsible for sodium and water absorption in the luminal surface of the airways and renal tubular cells. This channel is made up of three types of subunits, α, β, and γ. This channel seems to be critical in early life; knock-out mice for this channel develop pulmonary edema and die soon after birth.

At birth, the pulmonary circulation change from a high-resistance to a low-resistance system and, as a consequence, pulmonary blood flow becomes capable of accommodating systemic venous return. The change in resistance is brought about by the combined effects of the mechanical forces applied on the pulmonary vascular walls by the expanding lung tissue and the relaxation of the pulmonary arterial smooth muscle caused by the increased alveolar concentrations of oxygen and probably by endogenous release of vasodilators. The subsequent closure of the foramen ovale and the ductus arteriosus completely separates the pulmonary from the systemic circulation. Arterial oxygen tension then rises sharply and becomes homogeneous throughout the body. Pulmonary vascular resistance continues to decrease gradually during the first few weeks after birth through a process of structural remodeling of the pulmonary vessel musculature.

Postnatal development

The postnatal development of the lungs can be divided into two phases depending on the relative rates of development of the various components of the lungs. During the first phase, which extends to the first 18 mo after birth, there is a disproportionate increase in the surface and volume of the compartments involved in gas exchange. Capillary volume increases more rapidly than air space volume, which, in turn, increases more rapidly than solid tissue volume. These changes are accomplished primarily through a process of alveolar septation. This process is particularly active during early infancy and, contrary to previous belief, may reach completion within the first 2 yr instead of the first 8 yr of life. The configuration of the air spaces becomes progressively more complex, not only because of the development of new septa but also because of the lengthening and folding of the existing alveolar structures. Soon after birth, the double capillary system in the alveolar septa of the fetus fuses into one single, denser system. At the same time, new arterial and venous branches develop within the circulatory system of the acinus and muscle starts to appear in the medial layer of the intra-acinar arteries.

During the second phase, all compartments grow more proportionately to each other. Although there is little question that new alveoli can still be formed, the majority of the growth occurs through an increase in the volume of existing alveoli. Alveolar and capillary surfaces expand in parallel with somatic growth. As a result, taller individuals tend to have

larger lungs. However, the final size of the lungs and, ultimately, the dimensions of the individual constituents of the acinus are also influenced by factors such as the subject's level of activity and prevailing state of oxygenation (altitude), which allow for a better adaptation of lung structure and function. The same factors are probably operative in the compensatory responses to pulmonary disease and injury.

Chemoreceptor, temperature receptors, mechanoreceptors, and laryngeal receptors and their afferent play an important part in modulating respiration and rhythmic behavior

The neonate is more exquisitely sensitive to afferent input than is the adult. Laryngeal reflexes are extremely potent in inhibition respiration in the newborn. Aspiration and stimulation of laryngeal chemoreceptor in premature infants (who lack the ability of a strong cough), especially when these infants are anamic or hypoglycemic or even during normal sleep, can cause life-threatening respiratory events.

Effective ventilation requires coordinated interaction between the respiratory muscles of the chest wall (including the diaphragm and intercostals) and those of the upper airway (including the pharynx and the larynx) under various conditions of altered respiratory drive. In infants, a specific sequential pattern of nerve and muscle activation occurs so that some upper airway muscles contract prior to and during the early inspiratory flow: the genioglossus muscle contracts, moving the tongue forward, which prevents pharyngeal obstruction and the vocal cords abduct, reducing inspiratory laryngeal resistance. Laryngeal muscles also modulate expiratory flow and thus may influence lung volume.

Imbalance of pharyngeal and diaphragmatic activities or their responses to chemoreceptor or mechanoreceptor stimulation may contribute to obstructive apnoe in infants and children. Because the respiratory muscles are responsible for executing central neural responses and because muscle and chest wall properties change with age in early life, it is likely that neural responses can be influenced by pump properties. Thus, it is important to consider the maturational changes of respiratory muscles and the chest wall. For example, one of the important maturational aspects of respiratory muscles (e.g., in skeletal muscles) is their pattern of innervations. In the adult, one muscle fiber is innervated by one motor neuron. Therefore, i.e.: a motor neuron innervates certain muscle fibers (e.g., about 200 muscle fibers in the case of the diaphragm), these fibers do not receive innervations from any other motor neuron. In the newborn, however, each fiber is innervated by two or more motor neurons, and the axons of different motor neurons can synapse on the same muscle fiber—thus the term polyneuronal innervations. Synapse elimination takes place postnatally and, in the case of the diaphragm, the adult type of innervations is reached by several weeks of age depending on the animal species. The time course of polyneuronal innervations of the diaphragm in the human newborn is not known.

The neuromuscular junction folds, postsynaptic membranes, and acetylcholine receptors and metabolism undergo major postnatal maturational changes. The acetylcholine content per end plate potential is lower in the newborn than in the adult rat diaphragm. The newborn diaphragm is also more susceptible to neuromuscular transmission failure than is that of the adult, especially at higher frequencies of stimulation. Whether this is the result of differences in acetylcholine metabolism between the newborn and the adult or whether this is related to the neuromuscular junction itself is not known.

In addition to an increase in cross-sectional area and muscle mass, muscle fiber types in the diaphragm change as a function of gestational and postnatal age. However, there are conflicting reports about the composition of fiber types in young muscle, and it is not known whether human newborn muscles are more oxidative or fatigue-resistant than those of the adult. The sarcoplasmic reticulum of the premature diaphragm is, however, underdeveloped compared with that of the adult. This is one major reason for the delay in the release and uptake of Ca^{2+}, which may have functional significance. The poorly developed sarcoplasmic reticulum in the newborn causes increased contraction and relaxation time in neonatal muscle fibers. This increased relaxation time may be an important factor in impeding blood flow and limiting oxidative metabolism when the muscle is under a load.

The chest wall in newborn infants is highly compliant. Because of this and because young infants spend a large proportion of time in REM sleep, during which the intercostal muscles are inhibited, there is little splinting of the chest wall for diaphragmatic action. Therefore, with every breath in supine infants, the chest wall is sucked in paradoxically at a time when the abdomen expands. This creates an additional load on the respiratory system and results in a higher work of breathing per minute ventilation in the infant than in the adult. Some believe that this may be an important reason for the newborn's susceptibility to muscle fatigue and respiratory failure.

The young child and neonate respond to various stimuli in a different way than does the adult. In response to low O_2, the newborn does not sustain an increase in ventilation, and often ventilation decreases to below baseline levels. CO_2 levels do not increase at a time when ventilation is decreasing, suggesting that ventilation is matching metabolic needs. This neonatal response to low O_2 can be considered an intermediate response between those of the fetus and the adult; the fetus shuts off all respiratory efforts in response to O_2 deprivation, and the adult hyperventilates as long as the stimulus is present. The mechanism or mechanisms for the lack of sustained increase in ventilation during hypoxia in the newborn is not well understood. In addition to differences in metabolic rate during hypoxemia among neonates and adults, changes in the mechanical properties of the lung and airways, maturation of carotid chemoreceptors, and alterations in the cellular and membrane properties of central neurons have all been proposed as potential individual or combined mechanisms. It is clinically important that neonatal tissues resist O_2 deprivation and do not injure as easily as those of the adult. This is true for the heart, brain, and kidneys, organs known to be sensitive to hypoxia and ischemia in the mature animal or human. These differences between newborn and adult sensitivity to anoxic injury are not well understood but possibly relate to the ability of the newborn cell to metabolize lactate and ketone, to down regulate metabolism during severe O_2 deprivation, and to regulate certain protein synthesis (e.g., heat-shock proteins) differently from the adult.

CO_2 response is also reduced in the young. Whether this is a reflection of an inherent difference in sensitivity or the result of differences in mechanical function is not known and, presumably, is related to both mechanical and central differences.

Although alterations in responsiveness can be secondary to a number of differences between the young and the mature organism, the central neuronal changes with maturation seem especially important. For example, the soma of lumbar and phrenic motor neurons increases with age, and their input resistance (or inverse of membrane conductance) decreases with age. The decrease in input resistance results in major part from the increase in soma size, but other mechanisms, such as a change in the geometry of the dendrites and their

outgrowth, a change in the number of ion channels per surface area, and an increase in the number of synapses onto motor neurons, cannot be ruled out. Axonal velocity also increases with age, and action potentials of phrenic and hypoglossal motor neurons decrease in duration. There are also major maturational changes in active cellular properties in some motor neurons or premotor neurons. For example, with increasing postnatal age, neurons in the area of the nucleus tractus solitarius develop cellular properties important for repetitive firing. Changes in neuronal properties could play an important role in the integrative abilities of neuronal cells and, therefore, in their response to stimulation.

Clinical implications

Apnoea, although there are numerous studies on apnoea in the newborn and adult human, there are also a number of controversies. Further, the length of the respiratory pause that has been defined as apnoea has varied. Apnoea can be defined statistically as a respiratory pause that exceeds 3 SD of the mean breath time for an infant or a child at any particular age. This definition requires data from a population of infants at that age, lacks physiologic value, and does not differentiate between relatively shorter or longer respiratory pauses. Alternatively, the definition of apnoea may be based on the fact that respiratory pauses are associated with cardiovascular or neurophysiologic changes. Such definition relies completely on the functional assessment of pauses and is, therefore, more relevant clinically. Because infants have higher O_2 consumption (per unit weight) than the adult and relatively smaller lung volume and O_2 stores, it is possible that short (e.g., seconds) respiratory pauses that may not be clinically important in the adult can present serious consequences in the very young or premature infant.

Independent of age group, respiratory pauses are more prevalent during sleep than during the waking state. The frequency and duration of respiratory pauses depend on the sleep state in human infants. Respiratory pauses are more frequent and shorter in REM than in quiet sleep and are more frequent in younger than in older infants.

Although there is controversy regarding the pathogenesis of respiratory pauses, there is a consensus about certain observations. Normal full-term infants, children, and adult humans exhibit respiratory pauses during sleep. Paradoxically, some believe that the presence of respiratory pauses and breathing irregularity is a “healthy” sign and that the complete absence of such pauses may be indicative of abnormalities. However, prolonged apnoea can be life-threatening. The pathogenesis of these apnoeas may relate to the clinical condition of the patient at the time of the apnoea, associated cardiovascular (systemic or pulmonary) changes, the chronicity of the clinical condition, the perinatal history, and whether the cause is central or peripheral. Prolonged apnoea spells require therapy and, optimally, treatment should be targeted to the underlying pathophysiology. A septic infant should be treated for the infection and a seizing infant with antiepileptic medication. The child with congenital hypoventilation syndrome, in the absence of pharmacologic therapy, should be placed on mechanical ventilation until properly paced with phrenic stimulators.

Upper airway obstruction. Upper airway obstruction (UAO) during sleep is recognized with increasing frequency in children. In contrast to adults with UAO, in whom the cause of obstruction often remains obscure, many children have anatomic abnormalities. A common cause of UAO in children is tonsillar and adenoidal hypertrophy due to repeated upper respiratory infections. Other associated abnormalities include craniofacial malformations, micrognathia, and muscular hypotonia. The usual site of obstruction in UAO in both infants and adults is the oropharynx, between the posterior pharyngeal wall, the soft palate, and the

genioglossus. During sleep, upper airway muscles, including those of the oropharynx, lose tone and trigger an episode of UAO.

Snoring with recurrent periods of respiratory pauses commonly occurs during sleep in children. Parents frequently describe periods of increasing chest wall movement without air flow and with cyanosis. In older children, the syndrome may include failure to thrive, developmental delay, and poor school performance. Hypertension and daytime hypersomnolence are less common abnormalities in children than in adults. Children with long-standing signs and symptoms of UAO during sleep can present with right ventricular failure and corpulmonale. The treatment, therefore, varies but should be targeted primarily at the underlying cause of obstruction. Some of these infants benefit from tonsillectomy and adenoidectomy, if obese, then by a reduction in weight. In some refractory cases, successful treatment has included continuous positive airway pressure applied through the nose.

Diagnostic Approach to Respiratory Disease

Carefully obtained family and personal histories are essential in the diagnosis of respiratory system diseases. Only after the history is obtained can the physical findings be interpreted with a good chance of arriving at the correct diagnosis.

Physical examination

Respiratory dysfunction usually produces detectable alterations in the pattern of breathing. Respiratory control abnormalities may cause the child to breathe at a low rate or periodically. Mechanical abnormalities, on the other hand, produce compensatory changes that are generally directed at maintaining or increasing ventilation. These changes include variable increases in the breathing rate, chest wall retractions, and nasal flaring. Children with restrictive disease breathe at faster rates, and their respiratory excursions are shallow. An expiratory grunt is common, as the child attempts to raise the functional residual capacity by closing the glottis at the end of expiration. Children with obstructive disease take slower, deeper breaths. When the obstruction is extrathoracic (from the nose to the midtrachea), inspiration is more prolonged than expiration, and an inspiratory stridor can usually be heard When the obstruction is intrathoracic, expiration is more ostensibly prolonged and the patient often has to make use of accessory expiratory muscles. Lung percussion is usually dull in restrictive lung disease and tympanic in obstructive disease but has limited value in small infants because it cannot discriminate between noises originating from tissues that are close to each other. Auscultation confirms the presence of inspiratory and expiratory prolongation and provides information about the symmetry and quality of air movement. In addition, it often detects abnormal or adventitious sounds such as stridor (a predominant inspiratory monophonic noise, usually caused by upper airway obstruction), rales or crackles (high pitch, interrupted sounds found during inspiration and more rarely during early expiration, which denote opening of previously closed air spaces), or wheezes (musical, continuous sounds usually caused by the development of turbulent flow in narrow airway areas).

Blood gas analysis

Cyanosis is influenced by skin perfusion and blood hemoglobin concentration, and is therefore an unreliable sign of hypoxemia. Arterial hypertension, tachycardia, and diaphoresis are late and by no means exclusive signs of hypoventilation. Blood gas exchange is evaluated most accurately by the direct measurement of arterial pO_2, pCO_2, and pH.

Respiratory function testing

The measurement of respiratory function in infants and young children may be difficult by lack of cooperation. Attempts have been made to overcome this limitation by creating

standard tests that do not require the patient's active participation. Respiratory function tests still provide only a partial insight into the mechanisms of respiratory disease at early ages.

Whether restrictive or obstructive, most forms of respiratory disease cause alterations in lung volume and its subdivision. Restrictive diseases typically decrease total lung capacity (TLC), which is the total volume of gas contained in the lungs at the end of a maximal inspiration. TLC includes residual volume (the volume of gas contained in the lungs at the end of a forced expiration), which is not accessible to direct determinations. It must therefore be measured indirectly by gas dilution methods or, preferably, by plethysmography. Restrictive disease also decreases vital capacity (VC), which is the total amount of gas that can be inhaled after a forced expiration. VC can be measured by spirometry and is commonly used at the bedside to assess the progression of neuromuscular disorders. Obstructive diseases produce gas trapping and thus increase residual volume and function residual capacity (RC, the volume contained in the lungs at the end of a tidal expiration), particularly when these measurements are considered with respect to TLC.

Measurements of elastic recoil and respiratory compliance require knowledge of the lung volume at which the measurements were made in order to be properly interpreted. Similarly, measurements of airway and total respiratory resistance are technically cumbersome and difficult to interpret because of the large and variable contribution of the upper airway (nose, pharynx, and larynx) and lung and chest wall tissues to these resistances.

Airway obstruction is more frequently evaluated from determinations of gas flow in the course of a forced expiratory maneuver. The peak expiratory flow is reduced in advanced obstructive disease. The wide availability of simple devices that perform this measurement at the bedside makes it useful for assessing children with airway obstruction. Evaluation of peak flows requires a voluntary effort and peak flows may not be altered when the obstruction is moderate or mild. Other gas flow measurements require that the child inhale to TLC and then exhale as far and as fast as possible for several seconds. Cooperation and good muscle strength are therefore necessary for the measurements to be reproducible. The forced expiratory volume in 1 sec (FEV1) correlates well with the severity of obstructive diseases. The maximal midexpiratory flow rate, the average flow over the middle 50% of the forced vital capacity, is a more reliable indicator of mild airway obstruction. Its sensitivity to changes in residual volume and vital capacity, however, limits its use in children with more severe disease. The construction of flow-volume relationships during the forced vital capacity maneuvers overcomes some of these limitations by expressing the expiratory flows as a function of lung volume.

Ventilation/perfusion studies

The ventilation/perfusion ratio can be examined in children with the help of radionuclide tracers.

Exercise testing

Exercise testing is a more direct approach to detect diffusion impairment as well as other forms of respiratory disease. Measurements of heart and respiratory rate, minute ventilation, oxygen consumption, carbon dioxide production, and arterial blood gases during incremental exercise loads often provide invaluable information about the functional nature of the disease. Often a simple assessment of the patient's exercise tolerance in conjunction with other more static forms of respiratory function testing may allow a distinction between respiratory and non-respiratory disease in children.

Sleep studies

The sleep state has an important influence on respiratory function, particularly in the newborn and young infant. Polysomnographic studies are often helpful when abnormalities of central respiratory control, muscular disorders, or respiratory complications from gastroesophageal reflux are suspected. These studies, which usually include the simultaneous assessment of ventilatory effort, airway gas flow, gas exchange, and sleep state, are also useful in the diagnosis and management of nocturnal hypoxemia and hypercarbia in children with chronic respiratory disease.

Respiratory distress syndrome

This condition is a major cause of death in the newborn.

The failure to develop a functional residual capacity and the tendency of affected lungs to become atelectatic correlate with high surface tensions and the absence of surfactant.

Clinical manifestation

Signs usually appear within minutes of birth, although they may not be recognized for several hours until rapid, shallow respirations have increased to > 60/min. The late onset of tachypnea should suggest other conditions. Characteristically, tachypnea, prominent (often audible) grunting, intercostal and subcostal retractions, nasal flaring, and duskiness are seen. There is increasing cyanosis, which is often relatively unresponsive to oxygen administration. Breath sounds may be normal or diminished with a harsh tubular quality, and, on deep inspiration, fine rales may be heard, especially over the lung bases posteriorly. The natural course is characterized by progressive worsening of cyanosis and dyspnea. If inadequately treated, blood pressure and body temperature may fall; fatigue, cyanosis, and pallor increase, and grunting decreases or disappears as the condition worsens. Apnoea and irregular respirations occur as infants tire and are ominous signs requiring immediate intervention. There may also be a mixed respiratory-metabolic acidosis, edema, ileus, and oliguria. Signs of asphyxia secondary to apnoea or partial respiratory failure occur when there is rapid progression -of the disease. The condition rarely progresses to death in severely affected infants, but in milder cases, the symptoms and signs may reach a peak within 3 days, after which gradual improvement sets in.

Apnoea

Periods of apnoea, particularly in the premature infant, may be associated with a variety of disturbances. When apnoeas recur or when the intervals are longer than 20 sec or are associated with cyanosis or bradycardia, they warrant an immediate diagnostic evaluation.

Clinical manifestation

In preterm infants, serious apnoea is defined as cessation of breathing for longer than 15-20 sec or any duration if accompanied by cyanosis and bradycardia. The incidence of associated bradycardia increases with the length of the preceding apnoea and correlates with the severity of hypoxia. Short apnoeas (10 sec) are rarely associated with bradycardia, whereas longer apnoeas (>20 sec) have a higher incidence of bradycardia. Bradycardia is associated with apnoea in more than 95% of cases; vagal responses and, rarely, heart block are causes of bradycardia without apnoea.

Emergency care

Infants at risk for apnoea should be monitored with apnoea monitors Gentle cutaneous stimulation is often adequate therapy for the neonatal infant having mild and intermittent episodes. Infants having recurrent and prolonged apnoea require immediate bag and mask ventilation.

Pulmonary haemorrhage

Bleeding in the lower respiratory tract in children is unusual but can be life threatening. During infancy and prepubertal childhood, infections, trauma, and foreign bodies are probably the most common causes of pulmonary hemorrhage.

Most of the clinical manifestations are related to blood in the alveoli and to the effects of chronic blood loss. Symptoms are those of recurrent or chronic pulmonary disease and include cough, hemoptysis, dyspnea, wheezing, and occasional cyanosis associated with fatigue and pallor. The cough may be productive of bloody sputum or the infant or child may simply vomit large quantities of blood. During acute attacks, which usually last 2-4 days, the child may be febrile. Digital clubbing is often present.

Materials to activate the students during lecture:

Questions :

1. Anatomical and physiological features of respiratory system in children.
2. Features of embryogenesis of respiratory system organs and anomalies of their development.
3. Semiotics of defeats and main diseases of respiratory system in children.
4. Syndromes of respiratory disorders and respiratory insufficiency, main clinical manifestations.

Materials of methodical software of the lecture:

- Auditory
- Software
- Illustrations

The materials for self study of students. (Literature for the student.)

i) For the topic of the current lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 247 – 307; 780 – 781.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

j) For the topic of the next lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 454 – 533; 783 – 784.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

Literature used by lector during preparation the lecture:

37. Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – 808 pp.
38. Partha,s Fundamentals of Pediatrics. Ajanta offset &Packagings Ltd., New Delhi.-2013.- 782 pp.
39. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

40. Vicky R. Bowden, Cindy S. Greenberg. Pediatric nursing procedures. - Lippincott Williams & Wilkins. - 2011. - 822 pp.
41. Pediatric Nursing Procedures. Vicky R. Bowden, Cindy S. Greenberg. - Wolters Kluwer Health.- 2015 -728pp.
42. Nelson. Essentials of Pediatrics. Sixth Edition. Canada: 2011. - 831p.
43. Robert M. Kliegman. Nelson Textbook of Pediatrics 19th ed. – W.B. Saunders Company, 2011. – 2680 pp.
44. Pocket Book of Hospital Care for Children. – World Health Organization, 2013. – 438 pp.
45. Nelson Textbook of pediatrics / R. M. Kliegman, B. F. Stanton, J. W. St Geme III, N. F. Schor ; ed. by R. E. Behrman. – 20th ed. – Canada : ELSEVIR, 2016. – 5315 p.

Lecture № 7

Topic: «Anatomical and physiological peculiarities of cardiovascular system in children. Clinical signs of affection of cardiovascular system in children. Semiotics of hereditary cardiovascular diseases in children. Semiotics of acquired diseases of heart and vessels in children. Features of ECG and FCG in healthy children of different age» – 2 hours.

The actuality of the topic: Modem Clinical Cardiology childhood reflects the most significant achievements obtained in recent years in the basic sciences. Scientific advances in pediatric cardiology have significantly reduced the gap between theory and practice, and thereby opening up vast horizons for the prevention and treatment of many cardiovascular diseases, ranging from the neonatal period. Advances in diagnosis and treatment of congenital heart disease (CHD), valvular heart surgery and transplantation does not diminish the value of the old classical methods of diagnosis, and helps to learn that this lecture. It's very important for future doctors to know semiotics of damage and basic diseases of heart and vessels in children and syndromes of diseases of heart and vessels, basic clinical manifestations. Also for the pediatrician have to well orient in care of children having diseases of heart and vessels and its impossible without knowledge on semiotics of acquired diseases of heart and vessels at children. The main method of diagnostic of pathology of cardiovascular system is ECG and FCG. To make the diagnosis one should be able to correctly estimate the data got during the investigation of a patient, data of laboratory analyses and then to make clinical analyses of the semiotic symptoms of diseases.

Aims of the lecture

2.1. Practical (training) aims:

- Repeat embryogenesis and fetal circulation - questions that have been examined at the department of anatomy, but with lighting a critical period of formation of the anomalies of the heart and blood vessels that is necessary for knowledge on congenital heart disease To introduce students to anatomo-physiological peculiarities of the cardiovascular system of children in all periods of childhood, with clinical evidence of cardiac and vascular and modern methods of their investigation.
- to tell the students knowledge on the Semiotics of acquired diseases of heart and vessels in children.
- to tell the students knowledge on Features of ECG and FCG in healthy children of different age.

2.2. Educational aims:

- Mark merits domestic scientists in the development of Pediatric Cardiology (I.M. Rudney, S.V. Ignatov, M. Amosov);
- development of clinical thinking in students;
- bringing to the students the understanding of significance of the national clinical, scientific and dagogical schools in development of the problems mentioned in the lecture;
- to teach the students deontology and medical ethics.

Plan and structure of the lecture :

№№	**The main steps of the lesson**	**The aims in the levels of learning of the material**	**Materials of methodical software**	**Time (in minutes or %) of a hole lesson**
1	**2**	**3**	**4**	**5**
I.	*The preparatory level*	1	The presentation, Auditory	5%
1.	The defining the purpose			
2.	Formation of the positive motivation			
II.	*The basic level.*	*II.*		85-90%
3.	The presentation of the lecture by plan 1. Anatomical and physiological peculiarities of cardiovascular system in children. 2. Clinical signs of affection of cardiovascular system in children. 3. Semiotics of hereditary cardiovascular diseases in children. 4. Semiotics of acquired diseases of heart and vessels in children.	*II.* *II.* *II.*	Illustrations	

	5. Features of ECG and FCG in healthy children of different age.			
III.	Final level.		Literature, questions, tasks.	5%
4.	The conclusion of the lecture.			
5.	Answering questions.			
6.	The task for self-control			

The content of the lecture

The text of the lecture

Embryogenesis of cardiovascular system and critical periods of anomalies of heart and vessels in children of different age groups. Anatomical and physiological peculiarities of cardiovascular system in children

Knowledge of the cellular and molecular mechanisms of cardiac development is necessary to understand congenital heart defects and develop strategies for prevention. Developmental cardiologists traditionally grouped defects based on common morphologic patterns, for example, abnormalities of the outflow tracts (the conotruncal lesions such as tetralogy of Fallot and truncus arteriosus) or abnormalities of atrioventricular septation (primary atrial septal defect, complete atrioventricular canal defect). These morphologic categories do not provide an understanding of the mechanisms of heart malformations and their linkage to genetic alterations.

Early Cardiac Morphogenesis

In the early presomite embryo, the first identifiable cardiac precursors are angiogenetic cell clusters arranged on both sides of the embryo's central axis, which form paired cardiac tubes by 18 days of gestation. These tubes fuse in the midline on the ventral surface of the embryo to form the primitive heart tube by 22 days. Premyocardial cells continue their migration into the region of the heart tube, including epicardial cells and cells derived from the neural crest. The regulation of this early phase of cardiac morphogenesis is controlled in part by the interaction of specific signaling molecules or ligands, usually expressed by one cell type, with specific receptors, usually expressed by another cell type. For example, positional information is conveyed to the developing cardiac mesoderm by the retinoids, isoforms of vitamin A, which bind to specific nuclear receptors and regulate gene transcription. Migration of epithelial cells into the developing heart tube is directed by extracellular matrix proteins, such as fibronectin, interacting with cell surface receptors such as the integrins. The importance of ligands is noted clinically by the spectrum of human cardiac teratogenic effects caused by the retinoid-like drug isotretinoin. As early as 20-22 days, prior to cardiac looping, the embryonic heart begins to contract, exhibiting phases of the cardiac cycle that are surprisingly similar to those in the mature heart. Morphologists identified segments of the heart tube that were believed to correspond to structures in the

mature heart: the sinus venosus and atrium (right and left atria), the primitive ventricle (left ventricle), the bulbus cordis (right ventricle), and the truncus arteriosus (aorta and pulmonary artery). Studies that carefully map the fate of individual heart tube cells demonstrate that this model is oversimplified. Only the trabecular (most heavily muscularized) portions of the left ventricular myocardium are present in the early cardiac tube, the cells that will become the inlet portion of the left ventricle migrate into the cardiac tube at a later stage (after looping is initiated). Even later to appear are the primordial cells that give rise to the great arteries (truncus arteriosus), including cells derived from the neural crest, which are not present until after cardiac looping is complete.

At approximately 22-24 days, the heart tube begins to bend ventrally and toward the right through unknown biomechanical forces. Looping brings the future left ventricle leftward and in continuity with the sinus venosus (future left and right atria), whereas the future right ventricle is shifted rightward and in continuity with the truncus arteriosus (future aorta and pulmonary artery). This pattern of development explains the relatively common occurrence of the cardiac anomalies double-outlet right ventricle and double-inlet left ventricle, and the extreme rarity of double-outlet left ventricle and double-inlet right ventricle. Cardiac looping, one of the first manifestations of right-left asymmetry in the developing embryo, is critical for the successful completion of cardiac morphogenesis When cardiac looping is abnormal, there is a high incidence of serious cardiac malformations.

Potential mechanisms for cardiac looping include differential growth rates for myocytes on the convex vs. the concave surface of the curve, differential rates of cell death, and mechanical forces generated within myocardial cells via their actin cytoskeleton. The signal for this directionality may be contained in a concentration gradient between the right and left sides of the embryo by the expression of critical signaling molecules, (e.g., members of the TGF [tumor growth factor]-P family of peptide growth factors, and signaling peptides such as Sonic hedgehog). Immune models of abnormal looping, the specific defect resides in the dynein gene.

Cardiac Septation

When looping is complete, the external appearance of the heart is similar to that of the mature heart, internally the structure resembles a single tube, although now it has several bulges resulting in the appearance of primitive chambers. The common atrium (comprising both right and left atria) is connected to the primitive ventricle (future left ventricle) via the atrioventricular canal.

The primitive ventricle is connected to the bulbus cordis (future right ventricle) via the bulboventricular foramen. The distal portion of the bulbus cordis is connected to the truncus arteriosus via an outlet segment (the conus). The heart tube now consists of several layers of myocardium and a single layer of endocardium separated by the cardiac jelly, an acellular extracellular matrix secreted by the myocardium. Septation of the heart begins at approximately day 26 with the ingrowth of large tissue masses, the endocardial cushions, at both the atrioventricular and conotruncal junctions. These cushions consist of protrusions of cardiac jelly, which, in addition to their role in development also serve a physiologic function as primitive heart valves. Endocardial cells differentiate and migrate into the cardiac jelly in the region of the endocardial cushions, eventually becoming mesenchymal cells that will form part of the atrio-ventricular valves.

Complete septation of the atrioventricular canal occurs with fusion of the endocardial cushions. Most of the atrioventricular valve tissue is derived from the ventricular

myocardium, resulting from an undermining of the ventricular walls. As this process occurs asymmetrically, the tricuspid valve annulus sits closer to the apex of the heart than does the mitral valve annulus. The physical separation of these two valves produces the atrioventricular septum, the absence of which is the primary common defect in patients with atrioventricular canal defects. If the process of undermining is incomplete, one of the atrioventricular, one of the atrioventricular from the ventricular myocardium, a possible cause of Ebstein's anomaly.

The septation of the atria begins at approximately 30 days, with the growth of the septum primum downward toward the endocardial cushions. The orifice that remains is the ostium primum. The endocardial cushions then fuse and, together with the completed septum primum, divide the atrioventricular canal into right and left segments. A second opening appears in the posterior portion of the septum primum, the ostium secundum, which allows a portion of the fetal venous return to the right atrium to pass across to the left atrium. Finally, the septum secundum grows downward, just to the right of the septum primum. Together with a flap of the septum primum, the ostium secundum forms the foramen ovale, through which fetal blood passes from the inferior vena cava to the left atrium.

Septation of the ventricles begins at about day 25 with protrusions of endocardium in both the inlet (primitive ventricle) and outlet (bulbus cordis) segments of the heart. The inlet protrusions fuse into the bulboventricular septum and extend posteriorly toward the inferior endocardial cushion, giving rise to the inlet and trabecular portions of the interventricular septum. Ventricular septal defects can occur in any portion of the developing interventricular septum. The outlet or conotruncal septum develops from ridges of cardiac jelly, similar to the atrioventricular cushions. These ridges fuse to form a spiral septum, bringing the future pulmonary artery into communication with the anterior and rightward right ventricle, and the future aorta into communication with the posterior and leftward left ventricle. Differences in cell growth of the outlet septum lead to a lengthening of the segment of smooth muscle beneath the pulmonary valve (conus), which separates the tricuspid and pulmonary valves. In contrast, the segment beneath the aortic valve disappears, leading to fibrous continuity of the mitral and aortic valves. Defects in these processes are responsible for conotruncal and aortic arch defects (truncus arteriosus, tetralogy of Fallot, pulmonary atresia, double-outlet right ventricle, interrupted aortic arch), a group of cardiac anomalies often associated with deletions of the DiGeorge critical region of chromosome 22qll.

Aortic Arch Development

The aortic arch, head and neck vessels, proximal pulmonary arteries, and ductus arteriosus develop from the aortic sac, arterial arches, and dorsal aortae. When the straight heart tube develops, the distal outflow portion bifurcates into the right and left first aortic arches, which join the paired dorsal aortae. The dorsal aortae will fuse to form the descending aorta. The proximal aorta from the aortic valve to the left carotid artery arises from the aortic sac. The 1st and 2nd arches largely regress by about 22 days, with the 1st aortic arch giving rise to the maxillary artery and the 2nd to the stapedial and hyoid arteries. The 3rd arches participate in the formation of the innominate artery and the common and internal carotid arteries. The right 4th arch gives rise to the innominate and right subclavian arteries, and the left 4th arch participates in the formation of the segment of the aortic arch between the left carotid artery and the ductus arteriosus. The 5th arch does not persist as a major structure in the mature circulation. The 6th arches join the more distal pulmonary arteries, with the right 6th arch giving rise to a portion of the proximal right pulmonary artery and the left 6th arch giving

rise to the ductus arteriosus. The aortic arch between the ductus arteriosus and the left subclavian artery is derived from the left-sided dorsal aorta, whereas the aortic arch distal to the left subclavian artery is derived from the fused right and left dorsal aortae. Abnormalities in development of the paired aortic arches are responsible for right aortic arch, double aortic arch, and vascular rings.

Cardiac Differentiation

The process by which the totipotential cells of the early embryo become committed to specific cell lineages is called differentiation. Precardiac mesodermal cells differentiate into mature cardiac muscle cells, developing an appropriate complement of cardiac-specific contractile elements, regulatory proteins, receptors, and ion channels. Expression of the contractile protein myosin occurs at an early stage of cardiac development, even before fusion of the bilateral heart primordia. Differentiation in these early mesodermal cells is regulated by signals from the anterior endoderm, a process known as induction. Several putative early signaling molecules include fibroblast growth factor, activin, and insulin. Signaling molecules interact with receptors on the cell surface that activate, second messengers that activate specific nuclear transcription factors, (GATA-4, MEF2, Nkx, bHLH and the retinoic acid receptor family), which induce the expression of specific gene products to regulate cardiac differentiation. Some of the primary disorders of cardiac muscles and the cardiomyopathies may be related to defects in some of these signaling molecules. Developmental processes are chamber-specific. Early in development, ventricular myocytes express both ventricular and atrial isoforms of several proteins, for example, atrial natriuretic peptide (AMP) and myosin light chain (MLC). Mature ventricular myocytes do not express ANP and express only a ventricular-specific MLC 2v isoform, whereas mature atrial myocytes express atrial natriuretic peptide (ANP) and an atrial-specific MLC 2a isoform. Many pathologic cardiac conditions, for example, heart failure, volume overload, and pressure overload hypertrophy, are associated with a recapitulation of fetal cell phenotypes: Mature myocytes re-express fetal proteins. Since different isoforms have different contractile behavior (fast vs. slow activation, high versus low adenosine triphosphatase activity), the expression of different isoforms may have important functional consequences.

Developmental Changes in Cardiac Function

During development, there are profound changes in the composition of the myocardium, resulting in an increase in the number and size of myocytes. During prenatal life, this process involves myocyte division (hyperplasia), whereas after the first few postnatal weeks subsequent cardiac growth occurs by an increase in myocyte size (hypertrophy). The myocytes themselves change shape from round to cylindrical, the proportion of myofibrils (which contain the contractile apparatus) increases, and the myofibrils become more regular in their orientation. The plasma membrane (known as the sarcolemma in myocytes) is the location of ion channels and transmembrane receptors that regulate the exchange of chemical information from the cell surface to the cell interior. Ion fluxes through these channels control the processes of depolarization and repolarization. Developmental changes have been described for the sodium-potassium pump, the sodium-hydrogen exchanger, and voltage-dependent calcium channels. As the myocyte matures, extensions of the sarcolemma develop toward the interior of the cell (the t-tubule system), which dramatically increases its surface area, and enhances rapid activation of the myocyte. The membrane's α- and β-adrenergic receptors are regulated with development, enhancing the ability of the sympathetic nervous system to control cardiac function as the heart matures.

The sarcoplasmic reticulum (SR), a series of tubules surrounding the myofibrils, controls the intracellular calcium concentration. A series of pumps regulate calcium release to the myofibrils, initiating contraction (ryanodine-sensitive calcium channel), and calcium uptake, initiating relaxation (adenosine triphosphate-dependent SR calcium pump). In immature hearts, this SR calcium transport system is less well developed, resulting in an increased dependency on transport of calcium from outside the cell for contraction. In the mature heart, the majority of calcium activating contraction comes from the SR. This developmental phenomenon may explain the sensitivity of the infant heart to sarcolemmal calcium channel blockers such as verapamil, often resulting in a marked depression of contractility and cardiac arrest.

The major contractile proteins (myosin, actin, tropomyosin, and troponin) are organized into the functional unit of cardiac contraction, the sarcomere. Each has several isoforms, which are expressed differentially by location (atrium vs. ventricle) and by developmental stage (embryo, fetus, newborn, adult).

Changes in myocardial structure and myocyte biochemistry result in easily quantifiable differences in cardiac function with development. In isolated cardiac muscle strips, force of contraction increases with maturation from fetus to adult. Fetal cardiac function is poorly responsive to changes in both preload (filling volume) and afterload (systemic resistance). The most effective means of increasing ventricular function in the fetus is through increasing heart rate. After birth and with further maturation, preload and afterload play an increasing role in regulating cardiac function. The rate of cardiac relaxation is also developmentally regulated. Decreased ability of the immature SR calcium pump to remove calcium from the contractile apparatus is manifested as a decreased ability of the fetal heart to enhance relaxation in response to sympathetic stimulation. This inability of the immature myocardium to use preload effectively may partly explain the difficulty most premature infants have in compensating for the left-to-right shunt through a patent ductus arteriosus.

The fetal and neonatal circulatory transition

The Fetal Circulation

Much of the information concerning the fetal circulation has been derived from animal studies, especially those in fetal sheep. Although there may be some species differences, the human fetal circulation and its adjustments after birth are probably qualitatively very similar to those of other large mammals.

In the fetal circulation, the right and left ventricles exist in a parallel circuit as opposed to the series circuit of the newborn or adult. In the fetus, gas and metabolite exchange are provided for by the placenta. The lungs do not provide gas exchange, and vessels in the pulmonary circulation are vasoconstricted. There are three cardiovascular structures unique to the fetus that is important for maintaining this parallel circulation: the ductus venosus, foramen ovale, and the ductus arteriosus.

Oxygenated blood returning from the placenta, with a pO_2 of about 30-35 mm Hg, flows to the fetus through the umbilical vein. Approximately 50% of umbilical venous blood enters the hepatic circulation, whereas the rest bypasses the liver and joins the inferior vena cava via the ductus venosus, where it partially mixes with poorly oxygenated inferior vena caval blood derived from the fetal lower body. This combined lower body plus umbilical venous blood flow (pO_2 of about 26-28 mm Hg) enters the right atrium and is directed preferentially across the foramen ovale to the left atrium. This blood then flows into the left ventricle and is ejected into the ascending aorta. Fetal superior vena caval blood, which is considerably less

oxygenated (pO_2 of 12-14 mm Hg), enters the right atrium and preferentially traverses the tricuspid valve, rather than the foramen ovale, and flows primarily to the right ventricle.

From the right ventricle this blood is ejected into the pulmonary artery. Because the pulmonary arterial circulation is vasoconstricted, only about 10% of right ventricular outflow enters the lungs. The major portion of this blood (which has a pO_2 of about 18-22 mm Hg) bypasses the lungs and flows through the ductus arteriosus into the descending aorta to perfuse the lower part of the fetal body as well as to return to the placenta via the two umbilical arteries. Thus, the fetal upper body (including the coronary and cerebral arteries, and those to the upper extremities) is perfused exclusively from the left ventricle with blood having a slightly higher pO_2 than the blood perfusing the lower fetal body, which is derived mostly from the right ventricle. Only a small volume of blood from the ascending aorta (10% of fetal cardiac output) flows across the aortic isthmus to the descending aorta.

The total fetal cardiac output the combined ventricular output (CVO) of both the left and right ventricles amounts to about 450 mL/kg/min. Approximately 65% of descending aortic blood flow returns to the placenta; the remaining 35% perfuse the fetal organs and tissues. In the sheep fetus, right ventricular output is approximately two times that of the left ventricle. In the human fetus, with a larger percentage of blood flow going to the brain, right ventricular output is probably closer to 1.3 times left ventricular flow. Thus, during fetal life the right ventricle is not only pumping against systemic blood pressure but is performing a greater volume of work than the left ventricle.

The transitional circulation

At birth, the mechanical expansion of the lungs and increase in arterial pO_2 results in a rapid decrease in pulmonary vascular resistance. Concomitantly, the removal of the low resistance placental circulation results in an increase in systemic vascular resistance. The output from the right ventricle now flows entirely into the pulmonary circulation, and because pulmonary vascular resistance is lower than systemic vascular resistance, the shunt through the ductus arteriosus reverses and becomes left to right. Over the course of several days the high arterial pO_2 constricts the ductus arteriosus and it closes, eventually becoming the ligamentum arteriosum. The increased volume of pulmonary blood flow returning to the left atrium increases left atrial volume and pressure sufficiently to functionally close the foramen ovale, although the foramen may remain probe-patent for many years.

The removal of the placenta from the circulation also leads to closure of the ductus venosus. Thus, within several days almost a total transition from a parallel (fetal) to a series (adult) circulation is completed. The left ventricle is now coupled to the high resistance systemic circulation, and its wall thickness and mass begin to increase. In contrast, the right ventricle is now coupled to the low resistance pulmonary circulation, and its wall thickness and mass decrease slightly. The left ventricle, which in the fetus pumped blood only to the upper body and brain, must now deliver the entire systemic cardiac output (approximately 350 mL/kg/min), an almost 200% increase in output. This marked increase in left ventricular performance is achieved through a combination of hormonal and metabolic signals, including an increase in circulating catecholamines and an increase in the level of the myocardial receptors (β-adrenergic) through which these catecholamines have their effect.

When congenital structural cardiac defects are superimposed on these dramatic physiologic changes, they often impede this smooth transition and markedly increase the burden on the newborn myocardium. Also, because the ductus arteriosus and foramen ovale do not close completely at birth, they may remain patent in certain congenital cardiac lesions.

Patency of these fetal pathways may provide either a life-saving pathway for blood to bypass a congenital defect (e.g., a patent ductus in pulmonary atresia or coarctation of the aorta or a foramen ovale in transposition of the great vessels) or may present an additional stress to the circulation (patent ductus arteriosus in a premature infant, pathway for right-to-left shunting in infants with pulmonary hypertension). The cardiologist has available pharmacologic means to either maintain these fetal pathways (e.g., prostaglandin E1) or to hasten their closure (indomethacin).

Neonatal circulation

At birth the fetal circulation must immediately adapt to extrauterine life as gas exchange is transferred from the placenta to the lung. Some of these changes are virtually instantaneous with the 1st breath, and others are effected over hours or days. After an initial slight fall in systemic blood pressure, there is a progressive rise with increasing age. The heart rate slows as a result of a baroreceptor response to an increase in systemic vascular resistance when the placental circulation is eliminated. The average central aortic pressure in the term neonate is 75/50 mm Hg.

With the onset of ventilation a marked decrease in pulmonary vascular resistance" occurs due to both active (pO_2-related) and passive (mechanical-related) vasodilation. In the normal neonate, closure of the ductus arteriosus and the fall of pulmonary vascular resistance result in a fall of pulmonary arterial and right ventricular pressures. The major decline of pulmonary resistance from the high fetal levels to the low "adult" levels in the human infant at sea level usually occurs within the first 2-3 days but may be prolonged for 7 days or more. Over the first several weeks of life, pulmonary vascular resistance decreases even further secondary to remodeling of the pulmonary vasculature, including thinning of the vascular smooth muscle and recruitment of new vessels. This decrease in pulmonary vascular resistance significantly influences the timing of the clinical presentation of many congenital heart lesions that are dependent on the relative systemic and pulmonary vascular resistances. For example, the left-to-right shunt through a ventricular septal defect (VSD) may be minimal during the 1st wk after birth when pulmonary vascular resistance is still somewhat high. As pulmonary resistance decreases over the next week or two, the volume of the left-to-right shunt through the VSD increases, leading eventually to symptoms of congestive heart failure.

Significant differences between the neonatal circulation and that of older infants may be summarized as follows: (1) right-to-left or left-to-right shunting may persist across the patent foramen ovale; (2) in the presence of cardiopulmonary disease, continued patency of the ductus arteriosus may allow left-to-right, right-to-left, or bidirectional shunting; (3) the neonatal pulmonary vasculature constricts more vigorously in response to hypoxemia, hypercapnia, and acidosis; (4) the wall thickness and muscle mass of the neonatal left and right ventricles are almost equal; and (5) newborn infants at rest have a relatively high oxygen consumption, which is associated with a relatively high cardiac output. The newborn cardiac output (about 350 mL/kg/min) falls over the first 2 months of life to about 150 mL/kg/min, then more gradually to the normal adult cardiac output of about 75 mL/kg/min. The high percentage of fetal hemoglobin present in the newborn may actually interfere with delivery of oxygen to the tissues in the neonate, requiring an increased cardiac output for adequate delivery of oxygen to the tissues.

The foramen ovale is functionally closed by the 3rd month of life, although it is possible to pass a probe through the overlapping flaps in a large percentage of children and in 15-25%

of adults. Functional closure of the ductus arteriosus is usually complete by 10-15 hr in the normal neonate, although the ductus may remain patent much longer in the presence of congenital heart disease, especially associated with cyanosis. In premature newborn infants an evanescent systolic murmur with late accentuation or a continuous murmur may be audible, and in the context of the respiratory distress syndrome, the presence of a patent ductus arteriosus should be suspected.

The normal ductus arteriosus differs morphologically from the adjoining aorta and pulmonary artery in that the ductus has a significant amount of circularly arranged smooth muscle in its medial layer. During fetal life, patency of the ductus arteriosus appears to be maintained by the combined relaxant effects of low oxygen tension and endogenously produced prostaglandins, specifically prostaglandin E2 (PGE2). In the full-term neonate, oxygen is the most important factor controlling ductal closure. When the pO_2 of the blood passing through the ductus reaches about 50 mm Hg, the ductal wall constricts; the mechanisms by which oxygen activates ductal constriction are not completely understood. The effects of oxygen on the ductal smooth muscle may be direct or mediated by its effects on prostaglandin synthesis. Gestational age also appears to play an important role; the ductus of the premature infant is less responsive oxygen, even though its musculature is developed.

Epidemiology of congenital heart diseases

Incidence. Congenital heart disease occurs in approximately 8 of 1,000 live births The incidence is higher among stillborns (2%), abortuses (10-25%), and premature infants (about 2% including ventricular septal defect [VSD], but excluding transient patent ductus arteriosus [PDA]) This overall incidence does not include mitral valve prolapse, the PDA of the preterm infant, and bicuspid aortic valves (present in about 0. 9% of adult series). Among infants with congenital cardiac defects, there is a wide spectrum of seventy. About 2-3 out of 1,000 total newborn infants will be symptomatic with heart disease in the 1st yr of life. The diagnosis is established by 1 wk of age in 40-50% of patients with congenital heart disease and by 1 mo of age in 50-60% of patients. Since palliative or corrective surgery has evolved, the number of children surviving with congenital heart disease has increased dramatically.

Most congenital defects are well tolerated during fetal life because of the parallel nature of the fetal circulation. Even severe cardiac defects, for example, severe hypoplasia of the left ventricle, can usually be well compensated for by the fetal circulation. It is only after the maternal circulation is eliminated, the fetal pathways (ductus arteriosus and foramen ovale) closed or restricted, and the cardiovascular system independently sustained that the full hemodynamic impact of an anatomic abnormality becomes apparent. One major exception is the case of regurgitated lesions, most commonly of the tricuspid valve. In these lesions, for example, Ebstein anomaly, the parallel fetal circulation cannot adequately compensate for the volume load imposed on the right heart. In utero heart failure, often with fetal pleural effusions and ascites (hydrops fetalis), may occur.

Although the immediate prenatal period marks the time of the most significant transitions in the circulation, the infant's circulation continues to undergo change after birth, and these later changes may also have a hemodynamic impact on cardiac lesions and their apparent incidence. For example, as pulmonary vascular resistance falls over the 1st several weeks of life, left-to-right shunting through intracardiac defects increases and symptoms become more apparent. The relative significance of various defects can also change dramatically with growth, some ventricular septal defects may become much smaller as the child ages. Alternatively, stenosis of the aortic or pulmonary valve, which may be mild m the newborn

period, may become worse if valve orifice growth does not keep pace with patient growth. The physician should be aware of the spectrum of severity for the various congenital heart malformations and their evolution with time, and always be alert for associated congenital malformations, which can adversely affect the patient's prognosis.

Etiology. The etiology of most specific congenital heart defects is still unknown. However recent advances in molecular genetics may soon permit identification of specific chromosomal abnormalities associated with many of these defects. It has long been appreciated that genetic factors played some role in congenital heart disease, the recurrence risk of congenital heart disease increases from 0.8% to about 2-6% if a 1st degree relative (parent or sibling) is affected. Currently, approximately 3% of patients with congenital heart disease have an identifiable single gene defect, such as Marfan or Noonan syndrome. Five to eight per cent of patients with congenital heart disease have an associated chromosomal abnormality: heart disease is found in greater than 90% of patients with trisomy 18, 50% of patients with trisomy 21, and 40% of those with XO (Turner syndrome).

Two to four per cent of cases of congenital heart disease are associated with environmental or adverse maternal conditions and teratogenic influences, including maternal diabetes mellitus, phenylketonuria, systemic lupus erythematosus, congenital rubella syndrome, and drugs (lithium, ethanol, thalidomide, anticonvulsant agents). Associated non cardiac malformations noted in identifiable syndromes may be seen in as many as 25% of patients with congenital heart disease.

Genetic counseling. Parents who have a child with congenital heart disease require counseling regarding the probability of a cardiac malformation occurring in subsequent children. With the exception of syndromes known to be due to a single gene mutation, most congenital heart disease is the result of a multifactorial inheritance pattern, which results in a low risk of recurrence. There is an approximately 0.8% incidence of congenital heart disease in the normal population, and this incidence increases to 2-6% for a 2nd pregnancy following the birth of a child with congenital heart disease, depending on the type of lesion in the 1st child. When two siblings have congenital heart disease, the risk for a 3rd affected child may reach 20-30%. In general, when a 2nd child is found to have congenital heart disease, it will tend to be of a similar class as the lesion that was discovered in the 1st instance. However, the degree of severity may be quite disparate, and associated defects may be variable. Certain cardiac lesions, for example, left-sided obstructive lesions, may be associated with a much higher rate of recurrence because of the presence of mild and clinically silent defects, for example, a bicuspid aortic valve, in other family members.

Fetal echocardiography has improved the rate of detection of congenital heart lesions in high risk patients. However, the resolution and accuracy of fetal echocardiography is not perfect. Furthermore, congenital heart lesions may evolve during the course of the pregnancy, for example, moderate aortic stenosis with a normal-sized left ventricle at 18 wk may evolve into aortic atresia with a hypoplastic left ventricle by 34 wk because of decreased flow through the left heart during the later half of gestation.

The question often arises as to whether a woman with congenital heart disease, either unoperated or operated, will be able to carry a fetus to term. The major factor in determining this is the mother's cardiovascular status. In the presence of a mild congenital heart defect, or after successful repair of a more severe lesion, normal childbearing is likely. The increased hemodynamic burden on a patient with poor cardiac function may result in significantly increased risk to the mother as well as to the fetus. The incidence of spontaneous abortion in

the presence of severe congenital heart disease is high, especially when the patient is cyanotic. The maternal risk in these situations is also quite high. Therefore, it is important to discuss various methods of birth control with young women with repaired or palliated congenital heart lesions. Antibiotic prophylaxis against endocarditis is also indicated at the time of delivery.

Relative Frequency of Congenital Heart Lesions

Lesions	% of all lesions
Ventricular septal defect	25-30
Atrial septal defect (secundum)	6-8
Patent ductus arteriosus	6-8
Coarctation of aorta	5-7
Tetralogy of Fallot	5-7
Pulmonary valve stenosis	5-7
Aortic valve stenosis	4-7
d-Transposition of great arteries	3-5
Hypoplastic left ventricle	1-3
Hypoplastic right ventricle	1-3
Truncus arteriosus	1-2
Total anomalous pulmonary venous return	1-2
Triscuspid atresia	1-2
Single ventricle	1-2
Double-outlet right ventricle	1-2
Others	5-10

Evaluation of the infant or child with congenital heart disease

The initial evaluation of the infant or child with suspected congenital heart disease involves a systematic approach with three major components. First, congenital cardiac defects can be divided into two major groups based on the presence or absence of cyanosis, which can be determined by physical examination, aided by transcutaneous oximetry. Second, these two groups can be further subdivided based on whether the chest radiograph shows evidence of increased, normal, or decreased pulmonary vascular markings. Finally, the electrocardiogram can be used to determine whether right, left, or biventricular hypertrophy exists. The character of the heart sounds and the presence and character of any murmurs further narrows the differential diagnosis. The final diagnosis is then confirmed by echocardiography and/or cardiac catheterization.

Acyanotic congenital heart lesions.

Acyanotic congenital heart lesions can be classified according to the predominant physiologic load they place on the heart. Although many congenital heart lesions induce more than one physiologic disturbance, it is helpful to focus on the primary load abnormality for purposes of classification. The most common lesions are those that produce a volume load, and the most common of these are the left-to-right shunt lesions. Atrioventricular valve regurgitation and some of the cardiomyopathies are other causes of increased volume load. The second major class of lesions causes an increase in pressure load, most commonly secondary to ventricular outflow obstruction (e.g., pulmonic or aortic valve stenosis) or narrowing of one of the great vessels (e.g., coarctation of the aorta). The chest radiograph and electrocardiogram are useful tools for differentiating between these major classes of volume and pressure overload lesions.

Lesions resulting in increased volume load.

The most common lesions in this group are those that cause left-to-right shunts: atrial septal defect (ASD), ventricular septal defect (VSD), atrioventricular septal defects (AVSD, AV canal), and patent ductus arteriosus (PDA). The pathophysiologic common denominator in this group is a communication between the systemic and pulmonary sides of the circulation, resulting in the shunting of fully oxygenated blood back into the lungs. This shunt can be quantitated by calculating the ratio of pulmonary to systemic blood flow, or Qp:Qs. Thus, a 2:1 shunt usually implies that there is twice the normal pulmonary blood flow.

The direction and magnitude of the shunt across such a communication depends on the size of the defect and the relative pulmonary and systemic pressures and pulmonary and systemic vascular resistances. These factors are dynamic and may change dramatically with age: intracardiac defects may grow smaller with time; pulmonary vascular resistance, which is high in the immediate newborn period, decreases to normal adult levels by several weeks of life; chronic exposure of the pulmonary circulation to high pressure and blood flow will result in a gradual increase in pulmonary vascular resistance. Thus, in a lesion such as a large VSD, there may be little shunting and few symptoms during the 1st week of life. When the pulmonary vascular resistance decreases over the next several weeks, the volume of the left-to-right shunt increases, and symptoms begin to appear.

The increased volume of blood in the lungs decreases pulmonary compliance and increases the work of breathing. Fluid leaks into the interstitial space and alveoli, causing pulmonary edema. The infant develops the symptoms we refer to as "heart failure", such as tachypnea, chest retractions, nasal flaring, and wheezing. However, the term heart failure is a misnomer; total left ventricular output is actually several times greater than normal, although much of this output is ineffective because it returns directly to the lungs. To maintain this high level of left ventricular output, heart rate and stroke volume are increased, mediated by an increase in sympathetic nervous system activity. The increase in circulating catecholamines, combined with the increased work of breathing, result in an elevation in total body oxygen consumption, often beyond the oxygen transport ability of the circulation. This leads to the additional symptoms of sweating, irritability, tachycardia, and failure to thrive. If left untreated, pulmonary vascular resistance eventually begins to rise, and by several years of age the shunt volume will decrease and eventually reverse to right-to-left.

Additional lesions that impose a volume load on the heart include the regurgitated lesions and the cardiomyopathies. Regurgitation of the atrioventricular valves is most commonly encountered in patients with partial or complete atrioventricular septal (AV canal) defects. In this lesion, the combination of a left-to-right shunt with atrioventricular valve regurgitation increases the volume load on the heart and leads to more severe symptomatology. Isolated regurgitation of the tricuspid valve is seen in Ebstein anomaly. Regurgitation of one of the semilunar valves is usually also associated with stenosis; however, aortic regurgitation may be encountered in patients with a VSD directly under the aortic valve (supracristal VSD).

As opposed to the left-to-right shunts, in which intrinsic cardiac muscle function is usually either normal or increased, in the cardiomyopathies heart muscle function is decreased. Cardiomyopathies may affect systolic contractility, diastolic relaxation, or both. Decreased cardiac function results in increased atrial and ventricular filling pressures, and pulmonary edema occurs secondary to increased capillary pressure. The major etiologies of cardiomyopathy in infants and children include viral myocarditis, a large range of metabolic

disorders, and endocardial fibroelastosis.

Lesions resulting in increased pressure load.

The pathophysiologic common denominator of these lesions is an obstruction to normal blood flow. The most common are obstructions to ventricular outflow: valvular pulmonic stenosis, valvular aortic stenosis, and coarctation of the aorta. Less common are obstruction to ventricular inflow: tricuspid or mitral stenosis and coarctation. Ventricular outflow obstruction can occur at the valve, below the valve (e.g., double-chambered right ventricle, subaortic membrane), or above it (e.g., branch pulmonary stenosis or supravalvar aortic stenosis). Unless the obstruction is severe, cardiac output will be maintained and symptoms of heart failure will be either subtle or absent. This compensation involves an increase in cardiac wall thickness (hypertrophy).

The clinical picture is quite different when obstruction to outflow is severe, usually encountered in the immediate newborn period. The infant may become critically ill within several hours of birth. Severe pulmonic stenosis in the newborn period (critical PS) results in signs of right-sided heart failure (hepatomegaly, peripheral edema) and cyanosis due to right-to-left shunting across the foramen ovale. Severe aortic stenosis in the newborn period (critical AS) presents with signs of left-sided heart failure (pulmonary edema, poor perfusion), rightsided failure (hepatomegaly, peripheral edema), and may progress rapidly to total circulatory collapse.

In older children, coarctation of the aorta usually presents with upper body hypertension and diminished pulses in the lower extremities. In the immediate newborn period, the presentation of coarctation may be delayed due to the presence of a patent ductus arteriosus. In these patients, the aortic end of the ductus may serve as a conduit for blood flow to partially bypass the obstruction. These infants become symptomatic when the ductus finally closes.

Cyanotic congenital heart lesions.

This group of congenital heart lesions can also be further divided based on pathophysiology: whether pulmonary blood flow is decreased (tetralogy of Fallot, pulmonary atresia with intact septum, tricuspid atresia, and total anomalous pulmonary venous return with obstruction) or increased (transposition of the great vessels, single ventricle, truncus arteriosus, total anomalous pulmonary venous return without obstruction). As with the acyanotic lesions, the chest radiograph is a valuable tool for differentiating between these two categories.

Cyanotic lesions with decreased pulmonary blood flow.

These lesions must include both an obstruction to pulmonary blood flow (at the tricuspid valve, right ventricular, or pulmonary valve level) and a pathway by which systemic venous blood can shunt right to left and enter the systemic circulation (via a patent foramen ovale, ASD, or VSD). Common lesions in this group include tricuspid atresia, tetralogy of Fallot, and various forms of single ventricle with pulmonary stenosis. In these lesions, the degree of cyanosis depends on the degree of obstruction to pulmonary blood flow. If the obstruction is mild, cyanosis may be absent at rest. However, these patients may develop hypercyanotic ("tet") spells during conditions of stress. In contrast, if the obstruction is severe, pulmonary blood flow may be dependent on the patency of the ductus arteriosus. When the ductus closes during the 1st few days of life, the neonate presents with profound hypoxemia and shock.

Cyanotic lesions with increased pulmonary blood flow.

In this group of lesions, there is no obstruction to pulmonary blood flow. Cyanosis is

caused by either abnormal ventricular-arterial connections or by total mixing of systemic venous and pulmonary venous blood within the heart. Transposition of the great vessels (TGV) is the most common of the former group of lesions. In TGV, the aorta arises from the right ventricle and the pulmonary artery from the left ventricle. Systemic venous blood returning to the right atrium is pumped directly back to the body and oxygenated blood returning from the lungs to the left atrium is pumped back into the lungs. The persistence of fetal pathways (foramen ovale and ductus arteriosus) allows for a small degree of mixing in the immediate newborn period, however, when the ductus begins to close, these infants develop extreme cyanosis.

The total mixing lesions include those cardiac defects with a common atria or ventricle, total anomalous pulmonary venous return, and truncus arteriosus. In this group, deoxygenated systemic venous blood and oxygenated pulmonary venous blood mix completely in the heart, resulting in equal oxygen saturations in the pulmonary artery and aorta. If there is no obstruction to pulmonary blood flow, these infants present with a combination of cyanosis and heart failure. In contrast, if pulmonary stenosis is present, these infants present with cyanosis alone, similar to patients with tetralogy of Fallot.

Acquired heart diseases

Infective endocarditis.

Clinical manifestations. The early symptoms and signs are usually mild.

Prolonged fever, without other manifestations (except occasionally weight loss), persisting for as long as several months, may often be the only medical history. Alternatively, the onset may be acute and severe, with high, intermittent fever and prostration. Usually, however, the onset and course vary between these two extremes. The symptoms are usually nonspecific and consist of low-grade fever with afternoon elevations, fatigue, myalgia, arthralgia, headache, and at times chills, nausea, and vomiting. New or changing heart murmurs are common, particularly when there is associated congestive heart failure. Splenomegaly is relatively common, and petechiae may occur. Serious neurologic complications, such as embolism, cerebral abscesses, mycotic aneurysms, and hemorrhage: these complications are manifested by meningitis, increased intracranial pressure, altered sensory and focal neurological signs. Myocardial abscesses may also occur and may rupture into the pericardium. Pulmonary and other systemic emboli are infrequent, except with fungal disease. Many of the classic skin manifestations develop late in the course of the disease; hence, they are seldom seen in the appropriately treated patient. These include Osier nodes (tender, pea-sized intradermal nodules in the pads of the fingers and toes), Janeway lesions (painless small erythematous or hemorrhagic lesions on the palms and soles), and splinter hemorrhages (linear lesions beneath the nails). These lesions probably represent vasculitis produced by circulating antigen-antibody complexes.

Rheumatic heart disease.

Rheumatic involvement of the valves and endocardium is the most important manifestation of rheumatic fever. The valvar lesions begin as small verrucae composed of fibrin and blood cells along the borders of one or more of the heart valves. The mitral valve is affected most often, followed in frequency by the aortic valve; right-sided heart manifestations are rare. As the inflammation subsides, the verrucae tend to disappear and leave scar tissue. With a repeated attack of rheumatic fever, new verrucae form near the previous ones, and the mural endocardium and chordae tendineae become involved.

Patterns of valvular disease

Mitral insufficiency

Clinical manifestations. The principal physical signs of mitral insufficiency depend on its severity. With mild disease, signs of heart failure will not be present, the precordium will be quiet, and auscultation will reveal a holosystolic murmur at the apex, radiating to the axilla. With severe mitral insufficiency, signs of chronic congestive heart failure, including fatigue, weight gain, weakness, and dyspnoe on exertion, may be noted. The heart is enlarged with a heaving apical left ventricular precordial impulse and often an apical systolic thrill. The 1st heart sound is normal; the 2nd heart sound may be accentuated if pulmonary hypertension is present. A 3rd heart sound is usually prominent. There is rarely a midsystolic ejection click, as seen in patients with nonrheumatic mitral valve prolapse. A holosystolic murmur is heard at the apex radiating to the axilla and the sternal edge. In addition, a short mid-diastolic rumbling murmur follows the 3rd heart sound; it is caused by increased blood flow from the volume-loaded left atrium across the mitral valve as a result of the massive insufficiency. The presence of a diastolic murmur associated with mitral insufficiency does not necessarily mean that mechanical mitral stenosis is present.

Rheumatic mitral stenosis

Clinical manifestations. Generally, there is a good correlation between symptoms and the severity of obstruction. Patients with mild lesions are asymptomatic. More severe degrees of obstruction are associated with effort intolerance and dyspnea. Critical lesions can result in orthopnoe, paroxysmal nocturnal dyspnea, and over pulmonary edema. These symptoms may be precipitated by uncontrolled tachycardia, atrial fibrillation, or pulmonary infections. Congestive heart failure is usually but not invariably associated with moderate or severe pulmonary hypertension. Right ventricular dilatation may result in functional tricuspid insufficiency, hepatomegaly, ascitis, and edema. Hemoptysis due to ruptured bronchial or pulmonar veins and, occasionally, pulmonary infarction may occur. Blood-streaked sputum appears during episodes of pulmonary edema. With chronic severe mitral stenosis, cyanosis and a malar flush are noted.

The jugular venous pressure is increased in the presence of congestive heart failure, tricuspid valve disease, or severe pulmonary hypertension. The heart size is normal with minimal disease. Moderate cardiomegaly is usual with severe mitral stenosis and sinus rhythm, but cardiac enlargement can be massive, especially when atrial fibrillation and heart failure supervene. The apical impulse is normal, but a parasternal right ventricular lift is palpable when pulmonary pressure is high. The principal auscultatory findings are a loud 1st heart sound, an opening snap of the mitral valve, and a long, low-pitched, rumbling mitral diastolic murmur with presystolic accentuation at the apex. The mitral diastolic murmur may be virtually absent in patients who are in congestive heart failure. A holosystolic murmur owing to tricuspid insufficiency may also be audible. In the presence of pulmonary hypertension, the pulmonic component of the 2nd heart sound is accentuated. An early diastolic murmur may be caused by associated aortic insufficiency or secondary pulmonary valvular insufficiency (Graham Steell murmur).

Aortic insufficiency

In chronic rheumatic aortic insufficiency, sclerosis of the aortic valve results in distortion and retraction of the cusps. Regurgitation of blood results in a volume overload with dilatation and hyperthrophy of the left ventricle.

Clinical manifestations. Symptoms are unusual except in severe aortic insufficiency. The large stroke volume and forceful left ventricular contractions may result in palpitations. Excessive sweating and heat intolerance are related to vasodilatation. Dyspnea on effort can progress to orthopnea and pulmonary edema; angina may occur during heavy exertion. In adolescents with severe insufficiency, nocturnal attacks with sweating, tachycardia, chest pain, and hypertension may occur.

The pulse pressure is wide with bounding peripheral pulses. The systolic blood pressure is elevated, and the diastolic pressure is lowered. In severe aortic insufficiency, the heart is enlarged and there is a left ventricular apical heave. There may be a diastolic thrill. The typical murmur begins immediately with the 2nd heart sound and continues until late in diastole. The murmur is heard over the upper and middle left sternal border with radiation to the apex and to the aortic area. Characteristically, it has a hollow, high-pitched blowing quality. Generally, the murmur is more easily audible in full expiration, with the diaphragm of the stethoscope placed firmly on the chest and the patient leaning forward. Occasionally, it may be louder in the recumbent position. A systolic ejection murmur sometimes preceded by a click is frequent and is produced by the large stroke volume. An apical presystolic murmur (Austin Flint) resembling that of mitral stenosis is sometimes heard and is the result of the large regurgitant aortic flow in diastole that prevents the mitral valve from opening fully.

Tricuspid valvular disease

Primary tricuspid involvement is rare following rheumatic fever. Tricuspid insufficiency secondary to right ventricular dilatation resulting from severe left-sided lesions can occur in patients in whom surgery is not carried out. The signs produced by tricuspid insufficiency include prominent pulsations of the jugular veins with a "c-v" wave, systolic pulsations of the liver, and a blowing holosystolic murmur in the 4th and 5th left parasternal spaces that increases in intensity during inspiration. Concomitant signs of mitral or aortic valve disease, with or without atrial fibrillation are frequent. Signs of tricuspid insufficiency decrease or disappear when heart failure produced by the left-sided lesions is successfully treated. However, tricuspid valvuloplasty may be required in some cases.

Pulmonary valvular disease

Pulmonary insufficiency occurs on a functional basis secondary to pulmonary hypertension or dilatation of the pulmonary artery. This is a late finding with severe mitral stenosis. The murmur (Graham Steell murmur) is similar to that of aortic insufficiency, but the peripheral arterial signs (bounding pulses) are absent.

Disease of the myocardium.

The status of the myocardium is a critical factor in the prognosis of cardiac disease.

Unlike in adults, where ischemic myocardial damage is often a prominent component of cardiac disease in children the myocardium is relatively unimpaired in the majority of congenital heart lesions. In some congenital lesions, for example, left-to-right shunts, myocardial function may actually be supranormal. However, in children with unoperated congenital heart disease, longstanding volume or pressure load or chronic hypoxia may lead to eventual myocardial dysfunction. In addition to injury resulting from congenital heart lesions, infections, mesenchymal diseases, endocrine disorders, metabolic and nutritional diseases, neuromuscular diseases, blood diseases, tumors, hypertension, and primary congenital anomalies may also directly affect the myocardium.

Myocarditis

Myocarditis refers to inflammation, necrosis, or myocytolysis that may be caused by many infectious, connective tissue, granulomatous, toxic, or idiopathic processes affecting the myocardium with or without associated systemic manifestations of the disease process or involvement of the endocardium or pericardium. Coronary pathology is uniformly absent. The most common manifestation is congestive heart failure, although arrhythmias and sudden death may be the first detectable signs. Viral infections are the most common etiology.

Clinical manifestations. The presentation depends on the age and acute or chronic nature of the infection. The neonate may present with fever, severe heart failure, respiratory distress, cyanosis, distant heart sounds, weak pulses, and tachycardia out of proportion to the fever, mitral insufficiency caused by dilatation of the valve annulus, a gallop rhythm, acidosis, and shock. There may be evidence of viral hepatitis, aseptic meningitis, and an associated rash. In the most fulminant form, death may occur within 1-7 days of the onset of symptoms. The chest roentgenogram demonstrates an enormously enlarged heart and pulmonary edema, the electrocardiogram reveals sinus tachycardia, reduced QRS complex voltage, and ST segment and T wave abnormalities. Arrhythmias may be the first clinical manifestation and in the presence of fever and a large heart strongly suggest acute myocarditis.

The older patient with acute myocarditis may also present with acute congestive heart failure; however, more commonly patients will present with the gradual onset of congestive heart failure or the sudden onset of ventricular arrhythmias. In these patients, the acute infectious phase has usually passed and an idiopathic dilated cardiomyopathy is present.

Diseases of the pericardium

Clinical manifestations. The first symptom of pericardial disease is often precordial pain. The major complaint is a sharp, stabbing sensation over the precordium and often the left shoulder and back; the pain may be exaggerated by lying supine and relieved by sitting, especially leaning forward. Because there is no sensory innervation of the pericardium, the pain is probably referred pain from diaphragmatic and pleural irritation. Cough, dyspnea, and fever may also occur. The presence of symptoms or signs associated with other organs and systems depends on the basic etiology of the pericarditis.

On physical examination, many of the findings relate to the degree of fluid accumulation in the pericardial sac. The presence of a friction rub is helpful but may be a variable sign in acute pericarditis, becoming apparent only after the effusion is reduced. When the effusion is larger, muffled heart sounds may be the only auscultatory finding. Narrow pulses, tachycardia, neck vein distention, and an increased pulsus paradoxus suggest significant fluid accumulation.

The pulsus paradoxus is caused by the normal slight decrease in systolic arterial pressure during inspiration. With cardiac tamponade this normal phenomenon is exaggerated, probably because of decreased filling of the left side of the heart with the inspiratory phase of respiration.

The degree of the pulsus paradoxus is determined with a mercury manometer. The patient is told to breathe normally, without exaggeration. Allowing the manometer to slowly fall, the first Korotkoff sound will initially be heard intermittently (varying with respirations). This first point is noted and the manometer then allowed falling until the first Korotkoff sound is heard continuously. The difference between these two systolic pressures is the pulsus paradoxus A pulsus paradoxus of >20 mm Hg in a child with pericarditis is a reliable indicator of the presence of cardiac tamponade; a 10-20 mm Hg change is equivocal.

Cardiopulmonary arrest

Children are more likely to have a respiratory arrest due to sepsis, infections, aspiration of foreign bodies, trauma including head injury and near-drowning, upper and lower respiratory tract disease, and sudden infant death syndrome. Other less common causes of arrest in children include metabolic abnormalities, cardiac disease and dysrhythmias, and distributive, hypovolemic, and cardiogenic shock. Half of all children who require cardiopulmonary resuscitation are infants.

Basic life support circulation. If ventilation is adequate, the rescuer then assesses circulation by checking for a femoral or brachial pulse in the infant under 1 yr of age or the carotid pulse in a child. If the pulse is not palpable, chest compressions are begun. If the pulse is palpable but the child is not breathing, ventilation is provided. The apnoic child may also require chest compression, as heart rate and stroke volume are frequently inadequate. The patient should be placed on a hard surface, and in a hospital setting a bedboard, which supports the entire width of the chest from shoulders to waist, may be placed below the patient.

If the infant is being carried, the body may be supported along the rescuer's forearm with the head supported by the rescuer's palm. The head is not allowed to be higher than the body. The rescuer's hand closest to the child's feet is placed with the index finger just below the intermamillary line, the index finger is raised, and the 3rd and 4th fingers are used to deliver compressions to the lower one third of the chest. Alternatively, two hands may be used to encircle the chest and compressions are delivered with both thumbs. The lower one third of the sternum is compressed one-third to one-half the depth of the chest, approximately 1/2-1 in chest compressions in children ages 1-8 yr. The hand closest to the child's feet is used to locate the xiphoid notch. The middle finger is placed in the xiphoid notch, and the index finger is placed next to it. The position of the index finger is noted, and the same hand is moved up so that the heel is adjacent and proximal to the line where the index finger rested. The heel of the hand delivers compressions at a depth of 1-1 1/2 in; care is taken to keep the fingers off the chest. Open chest cardiac compression is of little value and is not recommended except under unusual circumstances (cardiac surgery). The role of active compression-decompression CPR for in-hospital or out-of-hospital cardiac arrests in children has not been established.

A rate of 5 compressions to 1 ventilation is appropriate for both infants and children, whether there are one or two rescuers. The chest is compressed at a rate that would result in at least 100 compressions/min, but because compressions are interrupted to deliver ventilation, the effective compression rate is 80/min. A second rescuer or member of the resuscitation team should frequently reassess adequacy of pulses resulting from chest compressions.

Electrocardiogram (ECG) in children

Changes in cardiac anatomy and hemodynamics soon after birth are reflected in the evolution of the ECG of the neonate. Because vascular resistances in the pulmonary and systemic circulations are nearly equal in the fetus at term, the intrauterine work of the heart results in virtually an equal mass of both the right and left ventricles. After birth, systemic vascular resistance rises when the placental circulation is eliminated, and pulmonary vascular resistance falls when the lungs expand. These changes are effected over a period of hours or days, and are eventually reflected in the ECG as the right ventricular wall begins to thin.

The ECG demonstrates these anatomic and hemodynamic features principally by changes in the QRS and T-wave morphology. It is recommended that a 13-lead ECG be earned out in pediatric patients, including either lead VSR or V4R. These right precordial leads are extremely important in the evaluation of right ventricular hypertrophy in childhood. On occasion, lead V1 is positioned too far leftward to reflect right ventricular forces accurately and may display the usual R/S pattern of the mid-precordial leads. This problem is particularly present in premature infants in whom the ECG electrode gel may produce contact between all of the precordial leads. At the same time, V3R or V4R may reflect a dominant R or S pattern, which is also important diagnostically.

During the 1st days of life, right axis deviation, large R waves, and upright T waves in the right precordial leads (VSR or V4R and VI) are normally. When pulmonary resistance decreases and right ventricular pressure reaches its normal level, the right precordial T waves become negative. In the great majority of instances this occurs within the first 48 hr of life If upright T waves persist in leads V3R, V4R, or VI beyond 1 wk of life, this represents an abnormal finding, indicating right ventricular hypertrophy or strain, sometimes even in the absence of QRS voltage criteria. The T wave in VI should never be positive before 6 yr of age and may remain negative into adolescence. This finding represents one of the most important yet subtle differences between the pediatric and adult ECG.

In the frontal plane leads of the standard ECG, the mean QRS axis in the newborn normally lies in the range of 110 to 180 degrees. The right-sided chest leads reveal a larger positive (R) than negative (S) wave and may do so for months or years because the right ventricle remains relatively thick throughout infancy. Furthermore, owing to proximity, the voltage recorded by the right precordial leads is influenced to a greater extent by right ventricular depolarization. Left-sided leads (V5 and V6) also reflect right-sided dominance in the early neonatal period when the RS ratio in these leads may be less than 1. However, because the left precordial leads are in direct proximity to the left ventricle, a dominant R wave reflecting left ventricular forces quickly becomes evident within the 1st few days of life. Over the years, the QRS axis gradually shifts leftward and right ventricular forces slowly regress. As the left ventricle becomes dominant, the ECG evolves to the characteristic pattern of the older child, and finally the typical adult electrocardiogram emerges.

With the growth of the infant there is a slow regression of right ventricular dominance and an increase in left ventricular forces. Leads VI, V3R, and V4R will display a prominent R wave until 6 months to 8 yr of age. The majority of children will have an RS ratio greater than 1 in lead V4R until they are 4 yr of age. The T-waves are inverted in V4R, VI, V2, and V3 during infancy and may remain so into the middle of the 2nd decade of life and beyond. The processes of right ventricular thinning and left ventricular growth are best reflected in the QRS-T pattern over the right precordial leads. Thus, the diagnosis of right or left ventricular hypertrophy in the pediatric patient can be made only with an understanding of the normal developmental physiology of these chambers at various ages until adulthood is reached.

Ventricular hypertrophy may result in increased voltage in the R and S waves in the chest leads. However, the height of these deflections is governed by the proximity of the specific electrode to the surface of the heart, and by the sequence of electrical activation through the ventricles, resulting in variable degrees of cancellation offerees, as well as by hypertrophy of the myocardium. Because the chest wall in infants and children as well as in adolescents may

be relatively thin, the diagnosis of ventricular hypertrophy should not be based on voltage changes alone in the entire pediatric age range.

The diagnosis of pathologic right ventricular hypertrophy is difficult in the 1st week of life, as physiologic right ventricular hypertrophy is a normal finding. Serial tracings are often necessary to determine whether marked right axis deviation and potentially abnormal right precordial forces or T-waves, or both, will persist beyond the neonatal period. In contrast, an adult ECG pattern seen in a neonate suggests left ventricular enlargement. The exception is the premature infant, who may display a more "mature" ECG than his or her full-term counterpart as a result of lower pulmonary vascular resistance secondary to underdevelopment of the medial muscular, layer of the pulmonary arterioles. Thus, the electrocardiogram may simulate that of the older child, with left ventricular dominance manifested by a more mature R-wave progression across the precordium (qR in V6, R/S ratio in V4R, V3R and VI J{less than or equal} 1). Some premature infants display a pattern of generalized low voltage across the precordium.

Materials to activate the students during lecture:

Questions :

Anatomical and physiological peculiarities of cardiovascular system in children.
Clinical signs of affection of cardiovascular system in children.
Semiotics of hereditary cardiovascular diseases in children.
Semiotics of acquired diseases of heart and vessels in children.
Features of ECG and FCG in healthy children of different age.

Materials of methodical software of the lecture:

- Auditory
- Software
- Illustrations

The materials for self study of students. (Literature for the student.)

k) For the topic of the current lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 454 – 533; 783 – 784.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

l) For the topic of the next lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 308 – 384; 774 – 775; 781 – 783.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

Literature used by lector during preparation the lecture:

46. Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – 808 pp.
47. Partha,s Fundamentals of Pediatrics. Ajanta offset &Packagings Ltd., New Delhi.-2013.- 782 pp.
48. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.
49. Vicky R. Bowden, Cindy S. Greenberg. Pediatric nursing procedures. - Lippincott Williams & Wilkins. - 2011. - 822 pp.
50. Pediatric Nursing Procedures. Vicky R. Bowden, Cindy S. Greenberg. - Wolters Kluwer Health.- 2015 -728pp.
51. Nelson. Essentials of Pediatrics. Sixth Edition. Canada: 2011. - 831p.
52. Robert M. Kliegman. Nelson Textbook of Pediatrics 19th ed. – W.B. Saunders Company, 2011. – 2680 pp.
53. Pocket Book of Hospital Care for Children. – World Health Organization, 2013. – 438 pp.
54. Nelson Textbook of pediatrics / R. M. Kliegman, B. F. Stanton, J. W. St Geme III, N. F. Schor ; ed. by R. E. Behrman. – 20th ed. – Canada : ELSEVIR, 2016. – 5315 p.

Lecture № 8

Topic: «Age anatomical and physiological features of digestive system in children. Semiotics of defeats digestive organs and main diseases (gastritis, ulcer disease, cholecystitis, dyskinesia of bilious ways etc.). Syndrome of "acute abdomen» – 2 hours.

The actuality of the topic: Knowledge of anatomical and physiological features of the digestive system, The ability to review patient is an important part of the complex medical diagnostic process. Questions considered in the lecture are very important today because diseases of the digestive organs take a leading place in the whole structure of Pathology that occurs in childhood. Besides syndrome of "acute abdomen" may be extremely dangerous for a child, because it is necessary to know deeply and thoroughly anatomical and physiological features, to be able to properly investigate the digestive system in children.

Aims of the lecture

2.1. Practical (training) aims:

- To acquaint students with Age anatomical and physiological features of digestive system in children and lesions of the gastrointestinal tract,
- to acquaint students with the semiotics of lesions of the digestive organs and main diseases;
- to acquaint students with the syndrome of "acute abdomen".

2.2. Educational aims:

- Training students in current clinical thinking,
- Ensuring the development of students leading domestic value of clinical, scientific and educational schools in the development of the problems of the lectures,
- Emphasize the need to fight for further reducing child morbidity and mortality from digestive diseases,
- Skills to teach ethics and medical ethics.

Plan and structure of the lecture :

№№	The main steps of the lesson	The aims in the levels of learning of the material	Materials of methodical software	Time (in minutes or %) of a hole lesson
1	**2**	**3**	**4**	**5**
I.	*The preparatory level*	1	The presentation, Auditory	5%
1.	The defining the purpose			
2.	Formation of the positive motivation			
II.	*The basic level.*	*II.*		85-90%
3.	The presentation of the lecture by plan 1. Anatomical and physiological peculiarities of digestive system in children. 2. Clinical signs of affection of digestive system system in children. 3. Semiotics of diseases of digestive system in children. 4. Semiotics of Syndrome of "acute abdomen.	*II.* *II.* *II.*	Illustrations	
III.	Final level.			5%
4.	The conclusion of the lecture.		Literature, questions, tasks.	
5.	Answering questions.			
6.	The task for self-			

	control			

The content of the lecture

The text of the lecture

Diseases of the digestive system take one of the first places in the incidence of children illness. Their spreadness constitutes 70-90% in the different climatic and geographic regions. For the last ten years this number has been increasing because of urbanization, unsuccessful ecological situation, early physical maturity and influence of the stress factors. The girls fall ill more often than the boys.

Predominantly the stomach and duodenum diseases, high frequency of their combining affection, tendency to prolong and recurrent course are peculiarities of gastroenterologic pathology. Study of anatomical and physiological peculiarities of gastroenteric tract organs helps to understand the essence of pathologic process, to estimate the functional condition of digestive organs more objectively, to be able to tell the difference between age functional peculiariries and pathology.

The digestive system is the complex of organs, including the *oral cavity, throat, esophagus* and *stomach, small and large intestine, salivary glands, liver and pancreas.* This is an integrated formation with typical common plan of structure.

At the same time the different parts of the digestive tract are distinguished by some peculiarities in their structure, conditioned by their function.

According to morphological and physiological signs digestive apparatus has 3 parts: front, middle and back.

The front part consists of the oral cavity with its derivatives and components, the throat and esophagus.

- The mechanical cutting of the food,
- partial digestion of the food particles and
- pushing it through into the stomach occur in this part.

The middle part consists of the stomach, small and large intestines, liver and pancreatic gland and

- realizes mainly chemical food digestion,
- absorption of the decomposited products (proteins, fats, carbohydrates, vitamins, microelementes, etc.) and
- formation of the scubalum mass.

The back part consists of the caudal part of the rectum and realizes evacuation of the scubalum mass from the digestive tract.

The digestive tract has *common plan of the structure* in spite of the morphological peculiarities of its different parts.

Its wall consists of <u>4 basic parts:</u>

a) the mucous membrane lining the digestive tract from inside;
b) submucous basis;
c) muscle layer;
d) external membrane, which is serous or adventicial tissue.

Mucous membrane consists of 3 plates (records): epithelium; proper plate of mucous membrane, muscle plate of mucous membrane.

Submucous basis is built from crumbly fibrillar unformed connective tissue, which

connects the mucous membrane with muscle layer together.

Muscle layer consists of internal circular and external longitudinal layers.

Serous membrane covers most part of the digestive tract as visceral peritoneum.

Now we dwell on the each digestive tract parts structure peculiarities.

The oral cavity of the first year life baby has some features that differ from those of an adult. These differences provide the children with the capability to more effectively suck milk from mother's breasts. The features of the oral cavity of a child of breast feeding age are:

- ✓ is relatively small
- ✓ in the center of the upper lip there is so called procheilon (3-4 mm)
- ✓ transversal folds on the lips
- ✓ the mucous membrane is gentle, dry and rich in blood vessels, very vulnerable
- ✓ the palate is almost flat
- ✓ the tongue is large and wide
- ✓ mass of the cheeks has the well developed fat pads (corpus adiposum buccae of Bitchat)
- ✓ the gums have torusable widening
- ✓ white and yellow nodules (Bones nodules) on the middle line of palate

Salivary glands (hypoglossal, submaxillary and parotid) in newborn is in the germinal condition and develop by 3-4th months of life.

By this age the quality of saliva increases and contains more diastase. The quantity of saliva achieves 1/5 or 1/10 part of the taking food and it is the physiological hypersalivation of a newborn.

Saliva pH of a newborn is neutral or sour. Saliva is secreted only by reflex.

Saliva contains amylase and ptyalin, which digest carbohydrates.

Suckling act is a congenital instinctive reflex. It consists of 3 phases:

1) squeezing of the nipple;
2) aspiration;
3) swallowing of milk. Behavior al reflexes (turning over of a newborn, definite situation near the breast, milk smell, etc.) play important role is the suckling act. Swallowing reflex is also congenital.

The features of the esophagus in children of early age are:

- ❖ well expressed funnel-shaped form
- ❖ glands absence
- ❖ insufficient development of elastic and muscle tissues
- ❖ well developed blood vessels
- ❖ relatively big in size:
  - newborn (is equal to) – 10 cm (1/2 of the length of the trunk)
  - 1 years – 12 cm
  - 5 years – 15 cm
  - 15 years – 20 cm
  - Adult – 25 cm (1/4 of the length of the trunk)
- ❖ Anatomical esophageal stenoses are expressed very bad
- ❖ The level of entrance into the esophagus
  in newborn – between III – IV cervical vertebras
  at 12 years – between VI – VII cervical vertebras

The place of the passage between the esophagus and the stomach is situated at the level of

10-11 thorax vertebrae during all childhood periods

After birth the thickness of the esophagus wall increases, chiefly, because of thickening of the mucous layer epithelium. It provides safety to the esophagus wall when the thick food bolus pass through it.

The stomach of the newborn has the following *peculiarities*:

- *Position* of the stomach of the first year old baby is horizontal and the lesser curvature of the stomach is addressed by concavity to the back. The stomach obtains vertical position when a baby starts walking (at 9-12 months).
- The *muscular layer* of the stomach at early age is insufficiently developed (The stomach form is impermanent and changes its position easily under the influence of filling with food and air):
  - The sphincter of the cardiac part of stomach is bad-developed and with weak muscle development promotes often regurgitation and aerophagy at breast feeding age
  - The muscles of the pyloric part, in contrary, are well developed, that promotes the development of pylorospasm
- The *volume of the stomach* increases according to the age:
  - Newborn – 7 ml after birth and up to 80 ml on the 10^{th} day
  - 3 months − 100 ml
  - 1 year − 200-250 ml
  - 3 years − 500 ml
  - 12 years − 1500 ml
- *Mucous membrane of the stomach* is abundantly vascularized and rather thicker. The number of the gastric glands are relatively small – 2 million, with growth the number increases and in 1^{st} year of age there are 10 million, in 10 years – 20 million, in adult person – 25 million. The mucous membrane of a newborn is characterized by the following histological features:
  - Chief cells (zymogenic) secreting pepsin, are not yet fully developed
  - Oxyntic cells (parietal) secreting hydrochloric acid – are sufficient in quantity, however their function is minimized
  - The small amount of goblet (calciform=beaker=chalice) cells secretes insoluble protective mucus which covers all the surface of the mucous membrane

  After 2 years of age the histological structure corresponds to that of an adult.
- *Secretory function of the stomach* has a number of peculiarities due to the degree of the central nervous system development:

a) gastric juice has the *total acidity* (2,5-3,0 times less than in adults) – it is equal to 20-40;

b) secretion of pepsin and labferment (renneting bag) and their force is less than in adults;

c) covering epithelium of the mucous membrane of the gastric body, additional cells and pylorus glands secrete extremely important protective factor-mucin. The pylorus glands contain endocrine cells producing bombesin. This factor stimulates production of the gastrin, secretion of HCL and pancreatic ferments;

d) any disturbance of the babies physiological condition leads to the decrease of acidity and ferments activity;

e) the ferment secretion depends on the food composition and babies condition: woman breast milk demands less digestive ferments; proteins increase secretion and fats depresses it.

Gastric digestion consists of: coagulation of milk; decomposition of fat and digestion of

casein (50% fats of the breast milk are decomposed in the stomach, but with artificial feeding fats decomposition in the stomach is absent), absorbtion. Absorbing gastric function of a baby is poor: sugar, water and protein decomposition products are absorbed here.

The pancreas of the children of early age is

- ✓ insufficiently differentiated
- ✓ more mobile
- ✓ weight in a newborn – 3 g, in the 15 year old child – 50 g.

It secretes:

- *pancreatic juice* into the duodenum (organic and inorganic substances, enzymes – trypsin, chymotrypsin, carboxypeptidase, elastase, amylase, maltase, lipase)
- *insulin* into blood.

The liver is the largest gland in the organism of a person. The liver is the basic hemopoetic organ in the intrauterine life After birth decomposition of the blood cells takes place here. The liver participating in:

- The process of digestion
- Metabolism
- The system of blood circulation
- Realization of enzymatic functions
- Carrying out excretory functions

Anatomo-physiological features of the liver:

- The liver in newborn is functionally undeveloped
- In children, especially of the first year of life is relatively large:
  - the newborn liver constitutes 4% of the body mass
  - adult liver − 2%
- The big size of the left lobe in the liver of a newborn decreases at the age of 1.5 years
- In a newborn the lobes of the liver are vaguely expressed, it is formed by the end of the 1st year of life
- Normally the lower edge of the liver till 7 years of age is palpated below the edge of the right costal margin along on the right medioclavicularis line:

Till 6 months	2-3 cm lower
6 months – 2 years	1.5 cm lower
3 – 7 years	0.5-1 cm lower
>7 years	It does not protrude from under the costal margin

- From 7 years of age, along the median line of the stomach, the liver does not descend lower than the upper third of the distance between umbilicus and xiphoid process
- In children of breast feeding age the liver is characterized by
  - Abundant vascularization
  - Imperfect differentiation of parenchyma
  - Weak development of connecting tissues

The histological structure of the liver of an infant corresponds to the adult at 8 years of age.

The liver grows up chiefly in width and thickness. The liver has vagal and sympathetic inervation, receptors are under the control of regulated action of the brain cortex.

The liver in its turn influence to CNS. This leads to different neurological manifestations. The liver is involved in pathologic process with any gastro-intestinal tract diseases.

Peculiarities of the structure and role of the children liver are following.

The liver of a child is plethoric. Development of the liver cells continues till 6-8 years old. Besides the bile production, neutralization, detoxicity, protein- and pigmentopoesis functions, taking part in metabolism (carbohydrating bile, fatty, watering and vitaminal), the babies liver carries out a number of functions. First of all, the liver is the basic hemopoetic organ in the intrauterine life. After birth decomposition of the blood cells takes place here.

Liver acts as storage of the glycogen, fat and protein in the liver. Part of the liver cells (kupfer's cells, endothelium of the portal vein) composes reticulo-endotelial apparatus. It takes part in phagocytosis, iron, lipid and cholesterol metabolism.

Functional deficiency of the newborn liver provokes physiological hyperbilirubinemia and sometimes - physiological jaundice of a newborn in the first days of life.

One of the main functions of the liver is bile formation. The gallbladder of a newborn is small and narrow and reaches the liver border in 2 years old. Bile secretion begins from 2-3 months of the intrauterine life, but after birth its excretion is very small.

Bile peculiarities at the baby's age are:

1.Quantity of the bilious acids is very small.

2.Predomination of taurocholic acid over glycocholic.

3.Significant contents of the mucous, water and pigments.

These peculiarities of bile composition are not accidental. They are necessary for children organism because taurocholic acid is more powerful antiseptic medicine than glycocholic acid. Taurocholic acid intensifies excretion of the pancreatic juice since ferments are necessary for digestion of fatty and sweet breast milk. Functions of the bile are the same in adults. The functions of bile are the following:

- It intensifies peristaltics of the large intenstine
- It neutralizes the content in the duodenum
- Emulsifies fats
- Activates lipase of the pancreas
- Dissolves and promoted the adsorption of fat-soluble vitamins

The small intestine of a newborn is relatively longer than that of an adult person. It consists of duodenum (7-10 cm after birth and 25-30 cm in the adult person, i.e. the increases is relatively small), jejunum and ileum (make accordingly 2/5 and 3/5 of the total length of small intestine). Mucous membrane is thin and highly vascularized. The epithelial cells are quickly regenerated. In comparison with adults, the intestinal glands are larger.

Large intestine is undeveloped till birth. So, the formation of cecum comes to the end by the end of the 1st year of life. The cecum and appendix are mobile. Apart from that appendix takes untypical position - behind the cecum and even may be in the small pelvis. Till 4 years of the age the *ascending part* is bigger than its descending part. The *transversal colon* occupies the horizontal position only in 2 years. The *sigmoid colon* in a newborn is very long and mobile, till 5 years of age it is located in the peritoneal cavity, and then goes down to the pelvis minor. The structure of the large intestine corresponds to that of the adult person only at 3-4 years of age. The digestive, motor and absorbing intestinal functions have definite peculiarities in the childhood.

The rectum in a newborn is relatively long. Its mucous and submucous membranes are fixed weakly, therefore the rectum might fall out easily.In rectum the final formation of fecal

mass (feces=stool) and absorption of water take place. *The age features of feces in children*

Age	The name	External features		
		Color	*The consistency*	*Smell*
1-3 day	Meconium	Dark green	Thick, homogenous	–
3-5 day	Transitional	Fragments of different colors – whitish, yellowish, greenish	Liquid, watery, with mucous and lumps	Gradually turns into sour
Since 5-6 day till 6 months	Usual natural feeding Artificial feeding	Golden-yellow Light yellow	Like liquid yoghurt Porridge-like (semi-liquid=doughy)	Acidic (sour) Putrefactive, harsh
Over 6 months	Usual	Brown	Thick (or formed =solid=shaped)	Usual (normal)

Digestive function.

Digestion in the intestine takes place under the influence of pancreatic and liver secretion and intestinal juice. Peculiarities of the digestive function are the following:

1) from birth the intestine juice contains the following ferments: enterokinase, erepsin, amylase, lactase, maltase and invertin;

2) children of the senior age have more lipase;

3) pH of the breast baby intestinal juice is usually weak acid, even neutral;

4) digestion of food substances to assimilation degree is connected with action of the pancreatic juice, which contains trypsin, amylase and lipase.

These enzymes of breast baby are insufficiently active and in the future their digestive force increases according to complicating of the child's alimentary regimen (diet). Lipolitic ferment is the most active.

Vigorous motorics is the peculiarity of the intestine motor function of the early age babies.

Therefore the defecation act comes as a reflex. Defecation becomes free from the end of the first year of life.

The absorption is the main functional activity of intestine. In a child absorption activity has the following peculiarities:

If a baby is breast fed, protein of breast milk is absorbed without digestion in the same form and in the form of aminoacids.

Products of fats, proteins, carbohydrates and soils decomposition absorb in the small intestine.

The large intestine is the organ for absorption of iron, phosphorus and alkalies.

Processes of fermentation predominate in the breast baby intestine, and putrefaction is practically absent.

In artificial feeding, absorption in the large intestine gets worse because of soaps and phosphates presence.

Duration of food passage through the intestine depends on age: with newborn-from 4 till 18 hours, with elder age children-about 24 on the average. In artificial feeding the duration of the intestinal digestion is about 2 days and nights.

Permeability of the intestinal epithelium for the products of incomplete digestion and microorganisms more in early age. Newborn intestine is sterile. Microorganism appear in the intestine 2-3 days after birth. They get in intestine from air, nipples of mother's breast and by nursing objects.

E. Coli predominates in the intestinal microflora in the artificially fed babies and bifido- and lactobacterium in breast-fed babies. Composition of microflora of 5-7 days baby is the same as in adult (according to american datas).

Microorganisms are spread in the intestine irregularly: most of them are in the rectum and colon and are practically absent in the small intestine.

Microorganisms have a very important role: they take part in the digestion, promote soaping of fats, dissolve cellulose, synthesize vitamins K and B-group.

Peritoneum.

The peritoneum is joined with abdominal wall more loosely, than in adults.

The mesentery can get longer and as a result hernia and invagination may appear in children.

Clinical conclusion.

In this way the system of the digestive organs of children is different from adults by a number of anatomical- and physiological peculiarities, which influence the functional promotion of these organs and pathology of digestion and nutrition at the early child age.

Modern methods of investigations are very helpful in the diagnosis of gastrointestinal tract diseases.

However questioning, inspection, palpation, auscultation along with instrumental and morphofunctional methods are very helpful in differential diagnostic criteria.

Diagnostic process usually starts with the patient's complaints anamnesis. All complaints which directly characterized diseases of gastrointestinal tract we can divide into 3 groups:

- Violation of appetite
- Dyspeptic disorder
- Pain in the abdomen

Appetite. Disturbance in appetite can be conditionally divided into 4 kinds:

1. Reduced appetite
2. Anorexia
3. Increased appetite
4. Perverted appetite

All this complaints can be a sign of appearance of diseases of gastrointestinal tract, and also a clinical feature of some diseases of other organs and systems. More often children suffer from loss of appetite (reduced appetite). It can be caused by:

- ✓ Disorders in digestion at diseases of gastrointestinal tract (enterocolitis, gastroduodenitis, etc.)
- ✓ Intoxication on the background of different diseases of the inflammatory character (flu, pneumonia, pyelonephritis, scalet fever, etc.)
- ✓ Violation of feeding and composition of food (overfeeding, irregular regime, excessively fatty food, etc.)

- ✓ Neurogenic reasons (defects in moral education, unsatisfactory conditions at home, as a result of which neuropathy can arise)

As a result of long violation accompanying with bad appetite the child may absolutely refuse to take food. The absence of appetite refers to anorexia. At the puberty stage anorexia occurs as physiological decrease of an appetite.

Increase in appetite (bulimia) is observed very rare in pediatrics. It occurs during diabetes mellitus, tumor of pancreas − insulinoma, brain tumor and dementia. Use of certain medicines (hormones, ftivaside) likewise, produces increase in appetite. Bulimia also may be in a child of breast feeding age at overfeeding with artificial mixes, when food flows easily from the bottle (big holes in the nipple).

The Perverted appetite is the condition in which the child willing eats uneatable substances like chalk, sand, soil, raw meat (lack of education of a child or lack of some substances in an organism (Ca) and in anemia).

The Dyspeptic disorder includes:

- Nausea
- Vomiting
- Rumination
- Belching
- Neartburn
- The disorders of defecation:
  - Diarrhea
  - Constipation

Defect of swallowing (Dysphagia) is return of swallowed food or liquid through nose, fear of swallowing, and retrosternal pain during swallowing and aspiration of food.

In general dysphagia is found in the early age of childhood as innate or congenital defects like undeveloped hard palate, atresia or asteronosis of esophagus.

In childhood burn of esophagus, post operative sternosis of esophagus, sorting of esophagus after esophagitis and collagenosis are also main defects.

Change in nearby organs like palate, retropharyngeal abscess, mediastinal tumor and others may cause dysphagia.

Vomiting (emesis) is the throwing out the contents of stomach and the upper part of the intestine through the mouth. It is a complex nervous reflux act. Its mechanism is controlled by vomiting center, which is located in medulla oblungata. More often vomiting precedes nausea - unpleasant sensation in epigastrium, sometimes accompanied with vegetative vascular reaction − paleness, weakness, dizziness.

Nausea is seen in pathology of digestive system, acute and chronic intoxication.

Vomiting is the early sign of the different gastrointestinal diseases and deseases of other organs including meningitis, toxicosis, diabetic acidosis and others. *Type of vomited mass has diagnostic value*. Vomiting of blood (haematoemesis) seen in haemorrhagic diethesis, gastric ulcer, duodenal ulcer and varicosis of esophageal vein and others. Light clotted blood indicates the point of bleeding upper to the cardiac part of gastric.

Vomiting with blood indicates the bleeding is in the stomach and duodenum. If bile is absent in vomiting it indicates obstruction in bile duct.

“Coffee grounds” vomiting – emetic mass of brown color is a sign of the presence of small quantity of blood in the stomach where it gets dark color under the influence of gastric juice; or if the blood stays in the stomach for a long time (bleeding at stomach and duodenal

ulcer, swallowing of blood from bleeding organs like nose, oral cavity, respiratory tract).

Obstructive process in the lower part of large and small intestine gives stool vomiting. Then vomiting has characteristic smell of stool.

Vomiting of fresh milk in breast-feeding babies is seen in esophageal obstruction and undigested food in children in esophageal constriction or obstruction in cardiac part of stomach.

If the structure of emetic mass looks like curd – curds milk – vomiting arose in 1.5-2 hours after feeding of the child of breast feeding age.

Vomiting may be of mechanical reflux and central origin when stimulant act directly on vomiting center and it is independent of age.

Vomiting of mechanical origin occurs often due to obstructive process in digestive tract. In the 1st 20-24 hours after birth occurrence of vomiting with increase in stomach due to obstruction in intestine. In such cases X-ray of digestive tract is of important value. Athresia of intestine or anal foramen, meconial illius are dangerous.

Pyloric spasm and pyloric stenosis causes vomiting after taking food. [Firstly it occurrs due to intense constriction in pyloric sphincter in hypotonia in the muscles of cardiac part of stomach and secondly congenital hypertonia of the mussels of pyloric part of stomach. Fountain (projective) vomiting is one of the main sign of pyloric stenosis at breast feeding babies. In this pathology vomiting occurs after each feeding, progressive hypertrophia non-constant emptying of intestine scanty stool. Lower half of stomach looks filled, upper dilated, visible peristaltic of stomach directed towards left to right. Final diagnosis gives X-rays.

Reflex vomiting often occurs in child. Reflex may be occurring with receptors pathological changes in digestive organs and urogenital system, internal ear, liver, bile secreting duct, cardiovascular system and other internal organs. Reflex vomiting in the first day of life of child is due to stimulation of mucous membrane of stomach due to ingested amniotic fluid. In infectious diseases vomiting is characteristic feature for gastroenteritis of different etiology, scarlet fever, mononucleosis, candidosis of intestine. Reflex vomiting occurs in some internal ear diseases for example. Acute medium otitis, concerned with labyrinth or occur due to increased excitation in the organ of equilibirium during transport, voyage and air transportation.

Central origins of vomiting in children arises are seen in edema or in brain tumor, intracranial haemorrhage, subdural haemotoma. Any disease causing increase in intracranial pressure may precedes with vomiting (hydrocyphalli,meningitis and meningoencephalytis, cranial trauma).

In child there is cyclic or periodic acetonemic vomiting due to spontaneous development of acetic acid. It generally occurs at the age of 3-8 years. Acetonemic vomiting characterizes spontaneous occurrence without any reason. After some hour it goes off spontaneously. Just after occurrence of vomiting exhaled air smell like acetone, in urine there is acetone, aceto acidic acid and hetaoxybutiric acid. During attack of acetonemic vomiting general condition of the children severs, sunken stomach but pain less attacks occurs 2-3 times a year.

Rumination is a rare kind of vomiting in children of breast feeding age. It characterized swallow emetic mass back, nausea usually is absent.

Belching (eructation) is spontaneous involuntary movement of gastric content (food eructation) in very small amounts or gases (gaseous (air) eructation) back to buccal cavity from the stomach.

In breast feeding babies belching occurs during aerophagia. Belching is seen in the 1st

weeks of life of the child. In children it occurs due to fermentation of gastric content of food with abundence of gases. In normal body weight and addition of food in accordance his belching is not taken as a sign of any disease. Step by step addition of food with solid consistency decreased frequency of belching decreases and after word it's completely disappears. In some conditions belching may be up to 1st year of life. Sometimes belching may occur due to violation of feeding rules. During breast feeding air may enter with milk in the stomach. Accumulated air in the stomach periodically comes from the stomach in the form of gas bubbles. With these gas bubbles food comes out.

Waste gases may have different smells. Foul-smelling eructation - Rotten smell characterizes fermentation and seen in gastritis with decrease acid production of pyloric sternosis. Acidic smell occurs in case of hyper acidosis and ulcer of gaster or duodenum. Belching with bitter taste characterizes diseases of bile secreting ducts and is concerned with reflux. Belching is one of the symptoms of insufficiency of gastroesophageal sphincter.

Pyrosis or heartburn is retrosternal feeling of burn or in epigastral region, sometimes refered up to pharynx because of movement of gastric contents back to esophagus. It is a sign of hyperacid gastritis and esophageal gastro reflux.

Diarrhea is too frequent defecation, at which the stool is liquid by consistence. The liquidness of stool is caused by the allocation of water. There are many courses of diarrhea (dystrophic diarrhea, "hungry stool", lienteric diarrhea, allergic, drug, antibiotic diarrhea, neurogenous diarrhea).

Constipation is the reduction of the frequency of defecation acts (it is quite often accompanied by difficulty) with the passage of thick stool mass (scybalous= "sheep's" stool). The sign of constipation in the child of the first year of life is the absence of excrement for more than a day, in the senior child – more than 2 days (or daily, but with difficulty). The reason of constipation at early age is the disorder of diet, in older age – disturbances in peristaltic of intestines, undevelopment of nervous-muscular system, psychogenic factor.

Abdominal pain is the most common complaint in the child age. In infants, abdominal pain is characterized by at least three episodes in a 3-months period in a child whose physical examination is normal. It affects 10-15% of children between 4 and 12 years of age. Abdominal pain may be severe, is sometimes associated with palor and emesis (30%), and frequently interferes with school attendance. Fewer than 10% have organic disease. The incidence of organic disease increases in children under 3 years and in the presence of symptoms such as diarrhea, weight loss, dysuria, fever, or neurologic symptoms. The pain varies in intensity, duration and location. The presence of nocturnal pain does not rule out the diagnosis. Headache, constipation and limb pains are common findings. Emotional stress related to family or school may be found in 30-50% of patients. Laboratory evaluatproportionto the apparent degree of pain.

In typical cases of intestinal invagination unexpected acute abdominal pain occurs. Besides pain vomiting and weakness are seen. Attack of pain repeats. Temperature may be normal at first. In 1st 12 hours of disease in the stool found blood. Stomach and intestine are large. Tumor like elongated formation are defined during palpation with different localization in the intestine, often right half of the stomach, somewhat higher to the nevus. Attack of pain causes increase in size of these elongated tumors like formation. At the tip of finger blood is found during manual rectal examination. Final diagnosis can be made with X-ray of intestine.

Pain syndrome	Hiatal hernia	Ulcer		Gastritis	Gallbladder Inflammation (cholecystitis)	Chronic pancreatitis
		Gastric	Duodenal			
Regularity of pain	-	+	+	-	+	Irregular (come in attack or are permanent)
Time of occurrence	Sometimes at night	30-40 min after meals (early pain)	1.5-2 h after meals (late pain), nocturnal, hunger	Short interval after eating	- (chronic – 1-3 h after taking fat and roasted food)	-
Character of pain	Colic	Colic		Aching	Acute (sharp), colic	Indefinite
Length (duration) of pain	Some minutes, rarely	1-2 hour	-	-	1-2 hour	-
Duration of pain period	-	3-4 weeks	-	-	2-3 days	-
Localization of pain	Area of xiphoid process, irradiated to the area of stomach and heart	Epigastric region, often clear limited		Epigastric region	Right hypochondrium, irradiated to the right shoulder and right scapula	Upper abdomen or in epigastrium and irradiates to the left shoulder, scapula, neck
Relation between pain and intake meals	Often increases	Increases	Increases or decreases	Intensifying or lessening	Increases after taking fatty and roasted food	Intensifies after taking fatty food
Nausea	-	-	+	+	+	+
Periodicity	-	+ seasonal		-	-	-
Vomiting	-	+	+	+	+ With a small amount of bile	+

Appendicitis is the most common lesion of the intestinal tract requiring surgery in childhood. The cause is not clear, although some cases result from impaction of a fecalith in the lumen of the appendix. Pinworms may occasionally cause appendicitis.

Symptoms and signs. Periumbilical or generalized abdominal pain occurs. After 1-5 hours, the pain becomes localized in the right lower quadrant. Urinary pain or frequency may be present if the appendix lies near the bladder. Constipation occurs frequently, but diarrhea is only occasionally seen. Vomiting is a late symptom.

Fever is low-grade or may be absent early in the course. Very high fevers are suggestive of appendiceal perforation, with peritonitis. Localization of tenderness may be difficult, but an opinion may be formed by palpating each area and noting the voluntary guarding or involuntary spasm of the abdominal musculature. Most children tend to flex the right thigh in an effort to decrease the spasm of the psoas muscle. However, the elicitation of a positive psoas sign is of doubtful value in small children. There may be rectal tenderness, a mass consisting of peritoneal fluid, or an indurated omentum wrapped around an inflamed appendix.

Laboratory findings. Two or three consecutive determinations of white blood cells (WBCs) will frequently show a rise in the total WBC, with an accompanying shift to the left in the neutrophilic series. A urinalysis should be performed in order to rule out infection of the kidney or bladder.

Imaging. In uncomplicated appendicitis, plain films of the abdomen may show a fecalith, scoliosis, or an abnormal gas pattern. When exudate has formed, evidence of peritoneal inflammation may cause disappearance of the properitoneal fat line along the right wall of the abdomen or obliteration of the psoas shadow.

A barium enema may be of value if the diagnosis is not clear since filling of the appendix tends to exclude the diagnosis of appendicitis. Abdominal ultrasound may show an enlarged appendix or abscess.

Differential diagnosis includes mesenteric adenitis, pyelitis, cystitis, pneumonitis (especially pneumococcal), gastroenteritis, peritonitis, constipation, Meckel’s diverticulitis, and inflammatory bowel disease (especially Crohn’s ileitis/ileocolitis).

Appendectomy should be performed as soon as the child has been prepared by adequate fluid and electrolyte administration. If there is doubt as to diagnosis, exploratory laparotomy or laparoscopy, with removal of the appendix and culture of peritpneal fluid, should be performed. Intravenous antibiotic therapy is indicated if peritoneal contamination has occurred.

Mechanical obstruction of intestine always causes abdominal pain following nausea, vomiting, eructation, absence of gases and stool. In the vomited mass found bile in complete obstruction symptoms are severe and increased. In complete obstruction symptoms are mild. X-ray gives final diagnosis. Abdominal pain is characteristic of strangulated hernia. Especially in the region of hernial folding, which increases in size.

Complication of intestinal invagination, appendicitis, mechanical obstruction, and strangulated hernia may be purulent peritonitis for which diffuse abdominal pain is characteristic feature, which increases during deep inhalation and on coughing, hyperstagia of skin of the anterior wall of the abdomen. Abdomen does not participate in respiration. Intestinal peristalsis slows down due to which different degree of flutulence. Constipation occurs, later on vomiting is added, symptoms of intoxication, painless, frequent superficial

respiration sunken eye, lack of interest in the surroundings, pulse is rare with continuous decreased filling and tension. Decreases in blood pressure, muscle of anterior abdominal wall are intense during palpation. In the blood signs of inflammatory process: leucocytosis with increased neutrophillic granulocytes, shifting of leucocyte formula to the left, mercased ESR.

Abdominal pain in children may occurs in pathological condition like mickels, diverticulum, doubling of intestine, primary constipation and helmenthosis, liamblesis, ulcer, regional enteritis, gastrointestinal allergy, cystic fibrosis, gastroduodenesis, gastric and duodenal ulcer, dyskinesia of gall bladder and bile duct, cholecystocolongitis, pancreatitis.

Diseases of small and large intestine of infectious origin often causes pain syndrome in abdomen followed by fever, vomiting, and loose motion.

Subdiaphragmatic process characterizes acute process in right hypochondrium, which increases in each inhalation. Pain irradiates to scapula and shoulder. Body temperature increases.

Abdominal pain may occur in affection of other organs of the abdominal cavity − kidney, urinary bladder, genital organs, capillary toxicosis, systemic diseases of connective tissue.

Constipation is a defect of physiological mechanism of defecation followed by disturbance and tone and peristaltic of intestine.

Pilorospasm is a primary spasm of pylorus without organic changes in pyloric canal.

Etiology. The clinical not know or unknown. It's generally found in children, with pathological perinatal period after long influence of intrauterine hypoxia and sensibilization, pathological labor and with diseases of neonatal period.

Pilorospasm is subdivided into primary and secondary. The later one develops in children with severe enteral and parenteral diseases with toxicose.

Clinical. Vomiting from the first days of life, oftenly, but not each feeding, not excessively less in volume than the milk consumed. There may be bile in the vomit. Gradually grows the deficit of body mass. Number of urination is less than normal (around 10). Stool 1-2 times in a day or at a gap of a day. Peristaltic is almost absent in the region of stomach. Skin is white.

Diagnosis. Diagnosis is made with the help of X-ray report. On the X-ray gram the pylorus is permeable, may be prolonged and stomach is distended. Symptoms of distonia in the spastic and relaxed parts. In the endoscopic examination on empty stomach: stomach is free from food parts. Pylorus is in round form, freely communicating with duodenum, X-ray and endoscopic examination are recommended after the effect for 4-7 days.

Pylorostenosis is more apt to occur in firstborn infants and is more common in males then in females (4:1 ratio) between 3 and 12 weeks of age. Circular musculature of the pylorus is hypertrophied, causing obstruction of the lumen.

Symptoms and signs. Emesis is mild at first but becomes progressively more projectile over 3-7 days. The vomitus does not contain bile. The infant appears hungry. Stools are small. Weight loss, dehydration, and hypochloremic alkalosis may be severe. Jaundice develops in 2-5% of cases. Gastric stasis results in gastritis and hematemesis in some cases. On examination, the infant is alert, irritable, dehydrated, and hungry. The epigastrium may be distended and the gastric outline obvious. Gastric peristalsis passing from left to right during feeding can be seen on the abdomen. An olive-shaped mass is palpable in the right upper quadrant, especially immediately after vomiting. Inguinal hernias develop in 10% of cases secondary to forceful emesis. Tetany as a result of alkalosis may occur.

Imaging. If the typical mass in the right upper quadrant is not palpable, the pyloric muscle may be demonstrated by ultrasonography. The upper GI series shows an enlarged stomach with a narrow, elongated pyloric channel and prolonged gastric retention of barium. The impression of the pyloric muscle on the antrum can be seen.

Treatment is surgical. Ramstedt pyloromyotomy divides the hypertrophied muscle bundles that obstruct the pylorus. Surgery should not be performed until rehydration and correction of alkalosis are complete. Complete relief is to be expected following surgical repair. Mortality is low.

The differential diagnosis of pilorospasm and pylorostenosis

Clinical signs	*Pilorospasm*	*Pylorostenosis*
Time of occurrences of the main symptom – vomiting	From the first days after birth	Mostly in 2-4 weeks after birth
Structure of emetic mass	Mainly uncurdled milk, a short time after feeding	Mainly curdled milk, may be with last feeding
Volume of emetic mass	Always less than the volume of the eaten food, can be insignificant quantity	Large, can be more than the volume of the eaten food
Frequency and regularity of vomiting	Often (2-4 times day), irregular	In the 2nd months becomes less frequent (1-2 times a day), but regular
Character of vomiting	Belching	Fountain-like
Physical development	Normal or slightly delayed	Delayed – hypotrophy develops
Stool	Constipation alternates with normal stool	Constipation
Volume of urine and frequency of urinations	Can be reduced 1.5-2 times	Considerably reduced (3-4 times)
Condition of the child	Often cries, restless, shouts before and during vomiting	Mostly quiet, sometimes may get excited before vomiting
Symptom of “hour-glass”	Absent	Is present
The results of X-ray	There is no contraction like “hour-glass”, barium remains in the stomach for about 6-8 hours	There is a contraction of the stomach like “hour-glass”, barium remains in it for about 24 hours and more
The result of ultrasound	Normal	Pyloric part: ▪ Hypertrophy of the wall > 4 mm ▪ Increase in length > 20 mm

		▪ On the cross-section – symptom of “cockade”
Efficiency of treatment with cholinolytics	Positive result after some days	No effect

Dyskinesia of biliferous tracts is the functional disorder of the motility of gallbladder and bile ducts as consequence of contraction of gallbladder and (or) sphincter apparatus.

Etiology and Pathogenesis. In mechanism of bile ducts motor function disorders and excretory function of liver have leading role neurovegetative innervating belongs to changes and intractory function of digestive system.

Clinical. Hypogenetical dyskinesia of biliferous tracts always observed in children with predominant tone of sympathetic path of vegetative neuro system. Pain syndroms are arises from the result of dissolution of gall bladder. It assists to exit of anticholynocystinokinase, more quality of its creation decrease the production of cholynocystinokinase in duodenum. Due to this moveable function of gall bladder decreases. Character of clinical hypermotor dyskinesia is sharp, weak, sometime permanent pain arise in right side in intercostals spaces of the ribs, strong emotion and mistake in diet will increase the pain feeling. Sometime occurs dispeptic occurrence – disease hebetite, telch sickness, bitter test, pain in abdomen, and discenty. On the palpation may be found pain in right side of intercostals spaces of the ribs, liver may be increased.

Hyperkinetical dyskinesia of gallbladder mostly observed in children with increased parasympathetic tone of vegetative nervous system. Patient have critical complaint – penetrating pain arise at a right side of intercostals space of the ribs. Pain may be radiated from right arm to right shoulder. It is short periodic. Sometime accompanied of sickness, vomiting. Pain syndrome occurs when there is a mistake in diet and emotional stress. On the objectical analysis, difines the projection of the pain in the gallbladder. It gives the count that character of pain syndrome in dyskinesia provide influence on such condition of insphincter apparatus. In spite of pain expression dispeptical syndrome, the occurrence of complete intoxication mostly absent in the period of afragetive palpation of abdomen the inflammatory change are absent or without pain or found the unknown pain in region of right interribs.

Diagnosis. Dyskinesia of gallbladder duct covers on main characteristic symptom and conform result of laboratory and instrumental analysis.

Meconum ileus with small bowel obstruction is the presenting sign in 15% of newborn infants with CF. Lack of pancreatic trypsin causes thick meconium, which obstructs the lower 10-20 cm of ileum. The ileocecal valve and the entire colon are normal, albeit small.

Symptoms and signs. Progressive bilious vomiting and abdominal distension occur in the first day or two of life. Firm masses within dilated bowel loops strongly suggest meconium ileus. In most cases, no meconium will have been passed per rectum.

Imaging. Marked intestinal dilatation is seen. A granular, mottled appearance to the intestinal content in the right lower abdomen is characteristic of inspissated meconium. Microcolon may be seen on barium enema. Free air in the peritoneal cavity or fluid between the loops of bowel indicates perforation. Calcification of the peritoneum is seen in antenatal perforation and meconium peritonitis.

Laboratory findings. The sweat shows an increase in the chloride concentration (>60 meq/L). Serum immunoreactive trypsinogen, usually elevated in newborns with CF, may be falsely normal in patients with meconium ileus, because of the severe antenatal reduction in exocrine pancreatic secretion.

Treatment and prognosis. Provide continious gastric suction through a nasogastric tube. Administer diatrizoate (Gastrografin) enemas but only when the infant is adequately hydrated. The hypertonic contrast medium draws water into the bowel and may "float out" the inspissated meconium. This procedure must be done under fluoroscopy by a radiologist familiar with newborn infants. Surgery is required if obstruction is not relieved. The ileum is opened and meconium removed. The portion of distal ileum containing the greatest amount of meconium may have to be resected with temporary ileostomy. There is no relationship between meconium ileus and the severity of the respiratory symptoms of CF.

Materials to activate the students during lecture:

Questions :

1. Age anatomical and physiological features of digestive system in children.
2. Semiotics of defeats digestive organs and main diseases (gastritis, ulcer disease, cholecystitis, dyskinesia of bilious ways etc.).
3. Syndrome of "acute abdomen.

Materials of methodical software of the lecture:

- Auditory
- Software
- Illustrations

The materials for self study of students. (Literature for the student.)

m) For the topic of the current lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 308 – 384; 774 – 775; 781 – 783.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

n) For the topic of the next lecture
Literature:
1**.** Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 621 – 670; 779 – 780.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

Literature used by lector during preparation the lecture:

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56. Partha,s Fundamentals of Pediatrics. Ajanta offset &Packagings Ltd., New Delhi.-2013.- 782 pp.
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58. Vicky R. Bowden, Cindy S. Greenberg. Pediatric nursing procedures. - Lippincott Williams & Wilkins. - 2011. - 822 pp.

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60. Nelson. Essentials of Pediatrics. Sixth Edition. Canada: 2011. - 831p.
61. Robert M. Kliegman. Nelson Textbook of Pediatrics 19th ed. – W.B. Saunders Company, 2011. – 2680 pp.
62. Pocket Book of Hospital Care for Children. – World Health Organization, 2013. – 438 pp.
63. Nelson Textbook of pediatrics / R. M. Kliegman, B. F. Stanton, J. W. St Geme III, N. F. Schor ; ed. by R. E. Behrman. – 20th ed. – Canada : ELSEVIR, 2016. – 5315 p.

Lecture № 9

Topic «Anatomical and physiological features of urinary system in children. Semiotics of most wide-spread diseases of urinary system in children (pyelonephritis, glomerulonephritis, cystitis etc.). Semiotics of microscopic changes of urinary deposit (proteinuria, erythrocyturia, leucocyturia,cilindruria etc.). Syndrome of acute and chronic renal insufficiency» – 2 hours.

The actuality of the topic: Knowledge of anatomical and physiological features of the urinary system, The ability to review patient is an important part of the complex medical diagnostic process. Questions considered in the lecture are very important today because diseases of the urinary organs take a leading place in the whole structure of Pathology that occurs in childhood. Semiotics of microscopic changes of urinary sediments (protein-, erytrocit-,leucocite-,& cylindruria. Syndrom of acute and chronic renal insufficiency, ability to investigate a patient is an important part of diagnostics and treatment. Diagnostics of the renal system affection is an actual problem due to its prevalence, diversity of clinical symptoms, often hidden proceeding with the following progress and development of the chronic renal deficiency. As a result, patients often become invalids in their productive age.
To make the diagnosis one should be able to correctly estimate the data got during the investigation of a patient, data of laboratory analyses and then to make clinical analyses of the semiotic symptoms of disease.

Aims of the lecture

2.1. Practical (training) aims:
- To acquaint students with Age Anatomical-physiological peculiarities of urinary system in children,
- to tell the students knowledge on the main diseases of the renal system and renal system affection semiotics.
- to tell the students knowledge on the syndrom of acute and chronic renal insufficiency
- to teach the students the methods of investigation and taking care of the patients.

2.2. Educational aims:
- Training students in current clinical thinking,
- Ensuring the development of students leading domestic value of clinical, scientific and educational schools in the development of the problems of the lectures,
- Emphasize the need to fight for further reducing child morbidity and mortality from digestive diseases,
- Skills to teach ethics and medical ethics.

Plan and structure of the lecture :

№№	**The main steps of the lesson**	**The aims in the levels of learning of the material**	**Materials of methodical software**	**Time (in minutes or %) of a hole lesson**
1	**2**	**3**	**4**	**5**
I.	*The preparatory level*	1	The presentation, Auditory	5%
1.	The defining the purpose			
2.	Formation of the positive motivation			
II.	*The basic level.*	*II.*		85-90%
3.	The presentation of the lecture by plan 1. Anatomical and physiological features of urinary system in children. 2. Semiotics of most wide-spread diseases of urinary system in children (pyelonephritis, glomerulonephritis, cystitis etc.). 3. Semiotics of microscopic changes of urinary deposit (proteinuria, erythrocyturia, leucocyturia,cilindruria etc.). 4. Syndrome of acute and chronic renal insufficiency.	*II.* *II.* *II.*	Illustrations Literature, questions, tasks.	
III.	Final level.			5%
4.	The conclusion of the			

	lecture.			
5.	Answering questions.			
6.	The task for self-control			

The content of the lecture

Diagnostics of the renal system affection is an actual problem due to its prevalence, diversity of clinical symptoms, often hidden proceeding with the following progress and development of the chronic renal deficiency. As a result, patients often become invalids in their productive age.

To make the diagnosis one should be able to correctly estimate the data got during the investigation of a patient, data of laboratory analyses and then to make clinical analyses of the semiotic symptoms of disease.

We consider the peculiarities of the embryogenesis of the urine organs their anatomical, histological and physiological peculiarities. It is necessary because anatomical and physiological peculiarities of the urinary organs in children of different age group define the character of pathology and symptoms of the disease.

Lets consider the peculiarities of the embryogenesis of the organ's of the urinary system. I will attract your attention to those peculiarities, knowledge of which is a ”clue” to the understanding of the essence of different anomalies in the development of kidney and urinal tracks. According to statistical facts' 10-14% (ten to fourteen percents) of children are born with different anomalies of the urogenital organs.

The urinary organs of fetus are developed from indifferentiated mesodermal tissue. The source of the development is metanephrogenal ropes (nephrotoms) which stretch along the embryo’s body between somits and celom. The development of the kidneys is continued in the some consecutive stages on the third week of embryogenal development from nephrotoms the pronephros is formed in embryo’s cranial part. The pronephos consist of 2-5 tubules, which open into the cavity of the body, and they are gathered in to the general duct (VOLFOV CANAL) of pronephros, which opens to the cloaca. At once non- functional pronephros is exposed to the reverse development (till the sixth week), because it has no connection with the circulatory blood system and does not lose the connection with celoms cavity.

Mesonephros develops on the 5-6 week. It is located in the trunk segments region. Mesonephros are the bilateral ropes, which consist of one line of convoluted tubule of kidney, at the free end of that the cuplike growth – it’s glomerular (boumenov) capsule and immersing in it glomerulus are formed. The tubules connect with the longitudinal mesonephral volfov duct, opening to urinal sinus.

Consequently, the mesonephros has the connection with the circulatory blood system and loses the connection with the celom’s cavity. The mesonephros functions during the second-third monthes and it is exposed to the reverse development (till the fourteenth week) and only its caudal parts take part in the formation of the genital glands.

At the second month (aproximately at the sixth week of the intrauterine life) the metanephros (secondary kidney) begins to developed in the caudal part of the embryon. It is formed from two parts of the mesoderm – nephrotoms and diverticules of the volfov canal.

The renal secretory apparatus (this is corpusculum renalis, tubules proximalis, tubules distalis, Henles loop) is formed from nephrotoms, and excretory apparatus (ductus papillares, pelvis renalis, calyces renalis, ureters) are formed from the diverticules of the volfov duct, which grows to the cranial side and which are divided into two-three blind sacks: calyces renalis major and calyces renalis minor. Ductus papillares are grown from them. Nephrons fix to ductus papillares between the twentieth and twenty sixths weeks of embryo's period.

So, in embrional development of the kidney the two main peculiarities may be isolated: first of all, the formation of the urogenital system goes from two different parts of the mesoderm, which are connectiveted then; secondly, the kidneys are formed in the caudal embryo's part and then they are raised from the pelvic cavity to the lumbar region.

These peculiarities of the urinary system embroygenesis have great clinical importance. For example, the kidney and ureter formation from different layers of the embryonic tissue may be the cause of the appearance of anatomical anomalies of the kidneys and ureter number. So, absence of the development of ureter sprout from the volfov duct lead to agenesia, that is the absence of the kidney in one or two sides together with ureter spourt. In case, when nephrotoms from the nephrogenal tissue are absent or they don t react upon the stimulation from the ureter spourt, aplasia is appears, that is the kidney is absent, but the ureter is present.

More that one ureter sprout are formed as result of influence of the unfavorable factors during pregnancy (on the seventh tenth week), that lead to doubling of the ureters or kidneys.

Besides, distrubence of embriogenesis lead to renal polycystosis, thoracic, lumbar, cross, and pelvic, iliac types of disposition.

During the embryo period the kidneys are lifted to their final position and turn round at their vertical axis. Under the influence of different causes the lifting and the rotation of the kidneys are impaired. As a rule In this anomaly the renal pelvis and gate of the kidney turn forward. But in excessive rotation of the renal pelvis can be behind or along the external border of the kidney.

During of embryogenes the insufficient development of the renal tissue results in deficit of the parenchyma and beginning of hypoplasia in which the kidney is diminished and does not increase with the age.

Anatomical and morphological peculiarities of structures of the kidneys and other urine system organs

The mass and capacity in newborn kidneys are more than adult ones. Mass of the newborn kidney is 1/100 of baby's mass; the mass of the adult's kidney is 1/200-1/250 of body mass.

The form of newborn kidney is almost round; form of a bean becomes later. The length of the kidneys does not exceed the height of four lumbar vertebrae. As a rule the right kidney is bigger than the left one by one centimetre. The width of the one-year baby kidney is sixty-five percent (65%) of its length. With the age the growth in length exceeds the growth in width. The width of the elder children's kidney is about fifty percent (50%) of the length of the organ, in adult thirty-thirty five percent (30-35%).

Children's kidneys are relatively bigger than adult's ones. That's why location of children's kidneys is a little lower. The location of the newborn kidney is at the level of the first-fifth lumbar vertebrae. The location of the elder children's kidney is between the

eleventh thoracic and the fourth lumbar vertebrae. The location of the right kidney is lower, but differences between the locaton of kidneys never exceed the height of one corpus vertebra.

The children till seven-eight year old have lower location of the kidneys. The kidneys are located in the niche of the retroabdominal's space, which is formed by muscles (M. quadrates lumborum and M. psoas major). From the outside the kidney is covers by the fibrous capsule, which in newborn is very thin and attached directly to the parenchyma, because of the fat capsule is absent.

The fixing apparatus of the kidneys consists of the some anatomical formations, such as fibrous capsule of the kidneys, ligaments (lig. hepatorenale, lig. lienorenale, lig. duodenale), vessels, near kidney fat cellulose, kidney fascia. The fixing apparatus in the newborns and babies kidneys is insufficient. It is explained by the absence of the fat cellulose and by weak development of the ligaments apparatus. The fixing mechanism is finally formed by five years, sometimes by eight years old.

The insufficient development of the kidneys fixing apparatus promotes more physiological agility of the kidneys in babies. The physiological agility of the kidneys is conditioned on respiration act and changing position of the body. Physiological agility of the kidney does not exceed the height of corpus of one lumbar vertebrae corpus and makes up one-two centimeters.

Displacement of the kidneys on 1,5 and more lumbar vertebrae testify about pathologic agility. It leads to lowering of the kidney – **nephroptosis.**

In the pubertal period, when the child is intensively growing in heigth, the insufficient development of the mechanism of the kidneys fixation promotes nephroptosis.

The kidney agility and their lower position allows to palpat of three years' babies throught anterior abdominal wall.

So, babies kidneys have next anatomical peculiarities:

a) the kidneys are relatively bigger by volume and mass (table 1);

b) the kidneys are located relatively lower and have a physiological agility;

c) the insufficient development of the mechanism of kidneys fixation frequently promotes pathological agility and nephroptosis.

Kidneys of newborn and early age babies keep the lines of embryo structure. The kidneys have 10-20 lobules, which are separated one from another by furrows of different depth.

Lobular structure of the kidneys disappear by 2-4 years of life, if the lobular structure of kidneys keeps later, it is **spanging kidneys**.

The renal tissue of the newborns and one year old babies is ontogenetic immature with insufficient development of substantia corticalis. In the newborn the thickness of the cortic layer is about 1/4 of medular one, in adult 1/2. Correlation such in adults becomes to 7 years. The medulla layer of kidneys in children is expressed well.

Glomerulogenesis is finished by the 34th week of the gestation, but formation of new nephrons are completed by the 20th day of the postnatal life. By the moment of birth the nephron has a number of features of morphologic immaturity:

– -at birth time glomerul are differentiated incompletely and they have a little diameter, which is 85 mk, where as in adult is 200 mk;

– -the epithelium, covering the glomerulus, is cylindrical, in adult – flat epithelium;

– -boumenov capsule consist of high epithelial cells, which do not penetrate between the vessels of the glomerulus.

Among the signs of immaturity of children's kidney there is unfinished development of the tubules renales:

- tubules renales have a little length and width;
- convolved tubule renales are twice narrow, than in adults;
- henle loops of tubule renales bad-developed and look like a little bend.

Kidney interstitial tissue of the newborn and the children of early age is bad-developed and consists of numerous reticular fibrils and cells, which look like fibroblasts. So, the interstitial affection of the kidneys, as interstitial nephritis, in children of early age appears rarely.

So, histological peculiarities of the renal tissue of the children are not absolute differention of nephron separate structures and the interstitial tissue. It has great importance for the functional immaturity.

Anatomical and histological peculiarities are typical for building of children's kidneys and urinary tract.

The renales pelvises of newborns and babies are relatively bigger and its walls are bad-developed hypotonic because of the muscular and elastic fibers. That's why the forms of the pelvises may be different such as ampularis, branchies, sac-like. The renal pelvis is finally formed by 12 months of life. The weak development of the muscular and elastic fibers, relatively bigger size of the pelvises and wall hypertension promotes retention of the urine and development of the inflammatory process.

The children up to 5 years have intrarenal localization of the pelvises that are characterized by the location of the pelvis inside the renal sinus. During the next years, in the process of opening of the renal sinus the pelvises settle down externally (outside the renal sinus).

There are some peculiarities of the children's ureters: they are relatively longer and wider than in adults. Ureters are twisty and have bends, especially in the places of crossing with the iliac vessels and transition in to the bladder wall. Ureters muscularis layer are expressed bad, that's why they seem atonical and contract badly and have insufficient interaction of the muscular contraction of separate 1 functions pronding of the ureters – cystoids that provide the passing of the urine to distal direction. The elastic layer of the ureters is absent, that promotes to atonical condition of the ureter walls. The mucous membrane has typical folds, which disappear at the end of first year of life.

Peculiarities of the ureters structure such as wide crack of the ureter, twisty, folds of the tunica mucosa, bends and atony of the wall's predispose to retention of the urine and formation of microbial inflammational diseases of the kidneys and urinary tract.

I want to pay your attention to else one peculieritie of the ureters structure in children that has important clinical meaning. Distal part of the ureter is intramural segment in the submucous membrane of the bladder. It is short in newborns, not higher than 0,5 cm. By 10-12 years this segment has maximum length – 1,5 cm.

The short interbladder segment leads to development of the reflux id est it leads to reverse of the bladder contents up to ureter. The babies have the bladder with some peculiarities, which change during the development:

– the bladder of the babies is located higher than the bladder of the children and adults. The bladder is in the suprapubical area. So, we can palpate it later it is gradually lowing into the cavity of pelvis minor;

– the muscles layer and elastic fibers of the bladder are bad-developed. The longitudinal muscular layer of the bladder is developed best of all. The circular layer is expressed poorly; the muscular layer develops actively since 6 years old;

– the muscular fibers are bad-developed around the mouth of the ureter, that's why they have the form of the small pitt and gape. These peculiarities promote to the appearance of bladder-ureter reflux;

– tunica mucosa of the bladder consists of the connective tissue, which is thick and developed better than in adults. But it is friable, tender and vascularized very well that leads to development of the inflammation process often.

Both the girls and boys in early age have the short ureter with weakly manifested folds of mucosa, which have a little connective tissue fibers of the elastic tissue. External ostium of uretra in girls is oval. Its anterior border is smooth and the posterior border is covered by villi, which prevent the invasion of infection from the anus.

So, such anatomical-histological peculiarities of the kidneys and urinary tract structure predispose to frequent infection of the urinary tract and renal pelvis, wich results in to bacterial-inflammatary diseases of the urine system in children.

Besides, anatomo-histological peculiarities of the kidneys and urinary tract make condition on the peculiarities of functions of the urine system in children.

Peculiarities of kidneys function.

Embryo kidneys begin to function relatively early. The urine is formed on the 9-10 weeks of embryogenesis, but excretory function does not matter. The excretion of products of metabolism is carried out by placenta. Kidneys' function during antenatal period means only to training of organs and it preparation to function in postnatal ontogenesis.

After birth kidneys carry out some functions, directed to guarantee of homeostasis. The kidneys take part:

1) guarantee of the constant, concentration of osmotically active substance of blood and other fluids of the organism (osmoregulation);
2) regulation of blood volume and extracellular fluids (volumoregulation);
3) ionic composition of the blood, acid-based condition (ABC);
4) the excretion of the products of azotic metabolism foreign substance;
5) metabolism of proteins, carbohydrates, fats and regulation of the arterial pressure, erythropoesis, vitamin D, prostoglandins and so on.

Processes in the nephrons and parenchyma provide these kidneys function by filtration of fluid in glomerulus, reabsorption and secretion in tubules, synthesis of new combinations.

Glomerular filtration (Gf) of the newborns and babies is lower than adults and equals 30-50% from corresponding value of the healthy people. The lower level of Gf in newborns and during first year of life is conditioned by lower arterial pressure, smaller diameter of abducting vessels and relatively little total filtration surface of the glomerulus, which is five times less than adult ones.

Gf becomes like in adult only by the end of the 2 years of life. Gf of the children at one year is 50-60 ml/min, and in the children and adults is 80-120 ml/min (table 2,3).

The tubules reabsorption of the newborns and babies of first year of life is flawed.

The function of kidneys stabilizing the acid -based condition in newborns is underdeveloped too. Therefore babies susceptible to food and endogenal acidosis.

The water-secreting function of babies is underdeveloped. Therefore mimit diuresis of babies is large.

In newborn diuresis is 200-300 ml and in the first year it's 600 ml. In babies from one year till ten years diuresis calculates by empirical formula: 600+100(n-1), where n is quantity of years of life.

The children over ten years excrete 1700-2000 ml urine during the day, and the night, like adults.

High diuresis and quick water metabolism lead to great necessity in water.

So, newborn necessity in water of 180-200 ml/kg, at six months – 120-130 ml/kg, at two-three years needs 90 ml/kg, the adult needs 40-50 ml/kg.

Frequency of urination from birth till 6 months is 20-25 times a day and it is 10-15 times a day at one year, and 6-8 times a day at school age.

The volume of urination is increased. Volume of urination of the babies till six months is 30 ml, one-year babies – 60 ml, at 3-5 years – 90-100 ml, at 7-8 years – 150 ml, at 10-12 years – 250 ml.

Concentration function of the kidneys of newborns is limited. The kidneys cannot form hypertonic urine. During the period to 3 months of life kidneys secret hypotonic urine relatively blood plasma.

The concentration ability of the kidneys is significantly perfected by 4-6 months of life, but it reaches the concentration ability of the adult by 9-12 months only.

The low concentration ability of child's kidneys influences the index of relative urine density: in newborn 1,006-1,012, in 2-5 years babies – 1,009-1,016, in 10-12 years – 1,012-1,025, like the adult one.

In conclusion, I take your attention these anatomical-histological and physiological peculiarities of the kidney of newborns and the babies testify about morphological and functional underdevelopment of the kidneys. Any diseases can lead to disturbance of kidneys function.

COMPLAINTS

Oedemata of the body (general or local) can take place with glomerulonephritis, nefritic syndrome, pyelonephritis, pyelonephrosis. It is necessary to pay attention to the time of their formation, their size, location, lability.

With inflammation in the kidneys. Oedema of the lids appears chiefly in the morning an during the first days of illness. And when the disease is progressing the oedemata appear also on the extremities and on the trunk.

Due to accumulation of liquid in the abdomen it can be enlarged. It is necessary to clear up how quickly the oedemata grow or under what circumstances they diminish.

Pain in the small of the back is usually constant, dull, pulling, with the acute and chronic glomerulonephritis, pyelonephritis, pigback kidney, when it is due to gallstones in the kidneys the pain is acute, paroxysmatic, lasts for some hours, often is accompanied with reflex anuria and vomiting. It is called renal stones disease.

A child can feel acute pain in the small of the back with a renal pelvis spasm or an ureter spasm, or due to an ureter's thrombosis with a clot of blood and pus.

Patients, suffering of renal stones disease, can provoke the paroxysms of pain if they jump run, ride or drive, change position of the body. After the paroxysms of pain sometimes red urine can occur or it can look like milk, or be turbid.

Paroxysmatic colic often occur in children after emotional stress, overstrain or sometimes after becoming too cold.

Pain in the lower part of abdomen (over the pubis) with irradiation into perineum is typical for the urinary bladder inflammation. In such case pain grows at the end of urination or just after it.

Urethritis gives great burning pain or colic in the lower part of abdomen. Urination is accompanied with the growth of painful sensation. Sometimes urination system inflammation causes pain of indefinite location.

Headache is a constant sign of hypertension syndrome: it takes place with the inborne and acquired kidney diseases (hypoplasia and displasia of renal tissue, abnormal vessels, polycystosis of kidneys, acute glomerulonephritis, chronic renal deficiency). When the arterial hypertension is steady, a headache is sharp, accompanied by giddiness, pain in the heart, short breath, paleness.

Distorsion of nitrogen excreting function due to chronic kidney disease leads to kidney contraction. Complaints: dryness and bitter taste in the mouth, sickness, vomiting, hard feeling above the stomach.

Chronic renal deficiency causes complaints of progressive flesh loosing up to dystrophy, constant thrist (babies often demonstrate adversion to any liquid), dyspepsia (it causes lasting diarrhea, convulsions of separate muscle groups, sometimes euphoria.

On the final stage of the disease oliguria appears. If uremia is progressing, a child becomes sleepy, sad, dull and very inert. Often smell of urine is produced from the mouth, diarrhea with particles of blood and mucus.

On the late stage of chronic nephritis retinopathology is developing, and due to that vision weakens.

Vision weakening up till blindness can sometimes be a symptom of the acute nephritis and takes place due to angiospasmus of retina.

ANAMNESIS OF THE DISEASE

For inborn and inherited nephropathy constitute about 15 per cent of all the kidney diseases in the infants, it is very important to find out if the parents or other relatives suffer of renal system pathology.

Acute and chronic infections, poisoning, cooling, arterial hypertension can provoke renal system disease.

It is necessary to bear in mind, that kidney pathology can appear due to transfusion of incompatible blood, injections of nephrotoxic antibiotics, sulphanilamides and some other medicines due to vaccinations, injections gammaglobulins and serum injections. It is of use to get data of previous urine analyses and other laboratory investigations, including X-ray and general clinical investigations of the patient.

ANAMNESIS OF LIFE

It is necessary to pay attention to the information on the family inherited diseases, that can directly or indirectly influence nephropathy in the child.

The data on the physical and mental development of the child during his life are very important as well as adaptive and resistance abilities of his organism, peculiarities of his reaction to different diseases suffered earlier. One should not fail to take into account that the regular overcooling of an infant can be the cause of nephritis, that’s why it is necessary to wear proper clothes according to the season during walking or playing outdoors, going in for skating, skiing etc. In winter.

EXAMINATION OF A PATIENT

One of the most characteristic signs of a renal pathology are oedemata very well seen during examination of a patient. Oedemata can be small and vivid only on the face and extremities of a patient, local (under the eyes) or general when liquid is gathered in the cavities.

Genesis of oedemata can be various and it depends on the kind of renal disease. The oedemata can appear due to heightened penetrability of capillary, enlarged re-absorbtion of water and sodium in the canals, accumulation of sodium in tissues, hypoprotheinemia (lowering of oncotic pressure), arterial hypertension, oliguria and anuria. Oedematic skin is pale and glossy. With general oedemata (anasarka) the imprints of linen are seen on the skin; the imprints can be produced by pressing a finger upon the skin. If a patient lies on his back, liquid accumulates in the sacrum region.

Diffusive affection of kidneys leads to general oedemata, accompanied with ascytis, hydrothorax, hydropericarditis. Sometimes the oedemata are not vivid, they can be hidden (they can be found only after the hydrotesting, regular weighing the patient, measuring of the all-day dieresis and comparing it with the amount of the taken liquid.

During the examination the localization and size of the oedemata can be determined. Glomerulonephritis is characterised by the oedemata of the face, shin, ankle, waist, genitals, on the stomach.

Hydrothrax leads to swelling of one side of the chest, limitation of the inhaling movement of the chest, that can be seen during attentive examination. Ascytis enlarges the stomach, sometimes umbilical hernia occurs.

When examining a patient with a tumor of the kidney it is possible to notice swelling of the waist.

For the patients suffering of renal diseases, pale skin and mucous membranes is usual due to spasm of the skin vessels and anemia. In the children with pyelonephritis pale complexion is accompanied with the blues under the eyes.

In a baby suffering of pyelonephritis, anemia, sometimes anxiety, sunken eyes, oedematic eyelids, dry skin, yellowish paleness, sometimes convulsions are the main symptoms. A tumor of the kidney is often accompanied with widening of the blood vessels on the stomach side and in the boys, besides, the veins of seminal canal are also widened. When the urinary bladder is sagged, some swelling is seen in the lower part of the stomach. Pathology of renal system can be visually determined due to signs of hypospadias or epispadias, ulcerous and inflammatory process in the urinary canal and other symptoms.

A patient suffering of uremia has typical appearance. He is languid, apathetic, pupils of the eyes are contracted and don't react to the light. Sometimes some groups of muscles twitch, but convulsions are not excluded as well. The skin is pale, yellowish, dry. On the skin the scratches are seen.

Due to large amount of the urinary acid and NaCl secretion small particles of them are seen on the skin and even on the face. Some patients have small hemorrhage spots on the mucous membranes. Besides, when examining a patient, general oedemata is seen (at the final stage of disease it is diminished). Breath is deep, rare, sometimes noisy, arythmic, it is so-called Chain-Stocks breath. Unpleasant smell from the mouth is felt, it is like urine's smell.

Palpation

Kidneys can be palpated, when they are enlarged (hydronephrosis, perinephritis, tumor, etc.) or shifted down.

Normal and Pathological Quantity/Quality Data of Urination
in Children Suffering of Kidney Disease

Quantity of urine produced by kidneys affected by different diseases varies greatly.
Diminishing of daily diuresis (oliguria) to 100-250 ml takes place with acute and chronic renal deficiency (glomerulonephritis, nephrotic syndrome, inborn anomalies of urethral system).
Oliguria on the background of renal deficiency appears due to a number of pathological alterations: deterioration of glomerular filtration, aggravation of the re-absorption in the renal channels, spasm and thrombosis of artery, partial destruction or not full development of the renal tissue.
Especially stubborn oliguria can take place with a contracted kidney, inborn defect of the kidneys, nephritic syndrome, amyloidosis, erythematosus and other system vasculitises.
Quantity of urine can diminish when eating dry food, loss of liquid due to diarrhea, perspiration, vomiting, great blood losses, heart deficiency, burns, fever, etc.
Anuria - full absence of urine excretion throughout a day (or very small amount of urine - 30/50 ml a day) can take place due to a renal or other kind of disease, for example, acute nephritis, nephritic syndrome, uremia, eclampsia, shock, poisoning (with mushrooms or different chemical poisons), botulism, peritonitis, incorrect blood transfusion, urinary stone disease and other mechanical obstacles in the urethra. Renal anuria is caused by the ceasing of urine excretion, non-renal - by the obstacles for the already produced urine on its way from the pelvis of the kidney.
Pathological enlarging of the urine amount (polyuria) is reported with diabetes mellitus and diabetes insipidous, diencephalic syndrome, phychonosema and phsychoemotional affection, after taking some diuretic or cardiac medicine and rather often at the end of acute nephritis (polyuretic phase of renal deficiency), genetic dysfunction of renal channels (Albright syndrome, renal glucousuria, etc.)
As a rule, polyuria is accompanied by pollariuria, that is, frequent urination. With at hydronephrosis polyuria and oliquria alternate.
Normally the daytime volume of urine in a child is twice as much as at night. Contrary situation can be with chronic pyelonephritis, circulation of the blood deficiency and other diseases.
Color of urine. Depending of the kind of disease the color of urine can differ greatly. Thus, with the insipideous urinoexhaustion urine is absolutely colorless, with a contracted kidney urine is light yellow due to insufficient oxidation of chromogenes. With the circulation of blood deficiency urine is dark yellow (miocarditis and heart valves deficiency). Glomerulonephritis renal calculus disease, tumors of renal system give meat slops color of urine (macrohematturia).
Great percentage of phosphates in urine gives grayish-milky color, and urates give brick-red color. With viral hepatitis and other parenhematic jaundices urine looks like beer due to great amount of hepatic pigments in it. Urine color also changes after taking some medicines and after taking medicines and after poisoning.
Analgin, amidopirin, carbolic acid give red or pink color in some children, after taking phenolphtalein or red phenol - purple color; poisoning with phenol, cresol, phenilhydzasine and melanosarcome give dark (almost black) color. 5-NOK gives bright-yellow colour of urine, while indican gives dark bravn one.
Carrot and red beet give their color to urine.
Transparency of urine.

Transparency of urine can be distorted due to great amount of different salts (urine acids, phosphorous, casbonics, bacterial, due to particles of blood (cylinders, fats, etc).

Smell of urine.

Smell of urine is of no diagnostic significance. It is like smell of spoiled apples due to large amount of acetone, which is characteristic of diabetes. Urine in children can have unpleasant odour due to eating garlic or other species.

Chemical reactions of urine.

Due to long-lasting starvation renal deficiency, diabetes, leucosis, tubeculosis of kidney and renal system, acidosis treating with ammonium chlorides, acid in urine grows.

When a patient is suffering of different infections of renal system, vomiting, oedemata drinking of alkaline mineral waters reaction shifts to alkaline reaction. Under the influence of bacteria CO_2 is formed that leads to alkalization of urine.

Relative density of urine.

If insufficient quantity of liquid comes to organism, relative density of urine grows. With hard renal deficiency, pyelonephritis, polycystosis, diabetes it diminishes as well as with polyuria of different genesis.

Microscopic investigation of urine sediments.

Urine sediments are divided into two groups:

1. Unorganized sediment (different alkaline, organic inclusions, medicine);
2. Organized sediments (epithelium, erythrocytes, leukocytes, cylinders).

1. Unorganized sediments of urine. A kind of sediments depends on urine reaction (it can be acid, alkaline or amphotheric). In the acid urine sediments are formed of urinal acid, alkaline (urates), calcium phosphate, calcium sulfate (gypsum), hippuric acid, calcium oxalate.

If a large number of urinal acid crystals are present in the urine sediments, it is a sign of lithic diathesis. To identify urinal acid crystals it is necessary to conduct a microchemical reaction (murexidie probe). In the first days of life there are many crystals of urinal acid and urates in a baby's urine. Due to that urine acquires brick-red color (urinal acidic infarction). When it is oxalatic diathesis, there are many crystals of calcium oxalate in the urinal sediments (excretion of oxalates is 2-3 times as much as normal, that is up to 30 mgr / % and even more). Such level can be found with neuro-arthritic, diathesis, diabetes mellitus, dystrophy, tuberculosis, after the hard diseases. Cystitis, alkaloses give amorphous phosphates and crystals of trypelphosphats.

2. Organized sediments of urine (erythrocytes, leukocytes, epithelial cells, cylinders) found during the laboratory investigation can help in diagnostics of renal diseases.

There exists false and real hematuria. The second is connected with pathology of renal system, the first - with excretion of blood from the genitals. With real hematuria it is necessary to clear out if excretion of erythrocytes takes place from the kidneys or from some other point of the renal system.

Acute nephritis with diffusive bleeding gives urine of brown-reddish (not bright-red) color.

Blood increments in the urine is a sign of local bleeding in the kidney pelvis or urinary bladder.

Small quantity of erythrocites in urnine is called microhematuria, large quantity of erythrocires (they cover all the visual field in microscope) is macrohematuria.

Macrohematuria gives red or brown urine. Macrohematuria gives ordinary color.
Hematuria is characteristic for nephritis and pyelonephritis, inborne anomalies (hydronephrosis, polycystosis, etc.) anaphylactoid purpura (Schonlein-Henoch disease) collagenous nephropathy, urolitiasis, tumour of kidney and renal system, trombosis of renal veins, tuberculosis of kidney, acute hemorragic cystitis, kidney trauma, medicine nephropathy, etc.
Leukocyturia is characteristic for inflectional diseases of kidney, pelvises, urinary bladder, urine excretion channel. Glomerulonephritis gives not many leukocytes in urine (10-30 in the visual field). Chronic glomerulonephritis gives still less quantity or no leukocytes at all.
Pyuria is characteristic for purulent affection of renal system on any level. It is typical for pyelonephrites of different ethnology. It is necessary to bear in mind possibility of a «false» pyuria in children, when pus increments in urine take place due to its excretion from the genitals (in the girls) or from abscesses on the organs, laying near the renal system. In such a case consultation of a surgeon or gynecologist is necessary.
Quality investigation of leukocytes is based on coloring of alive leukocytes, existing in urine during pyelnephritis. These leukocytes flow to urine from the regions of renal parenchyma inflammation and due to physical and chemical changes in urine (lowering of its osmotic pressure) leukocytes turn into so-called Sternheimer-Malbin cells. «Activ» leukocytes let water through their membranes quite well and enlarge their dimensions 2-3 times as much, but they can't be coloured well and have light gray or light blue color under the microscope. Those cells are named Sternheims-Malbin cells. They have multi-sectional nucleus, and protophlasma grams at the certain stage begin Brown movement. This is the reason of special appearance of the cells: they are bright sparkling, and can be of round, oval or pear-like form. «Active» leukocytes,turned into Sternheims-Malbin cells are placed among the common («unactive») cells of leukocytes, that are well-colored and don't change their form and dimensions.
It is proved that with hypostenuria. «Activ» leukocytes can turn into Sternheims-Malbin cells in the different parts of renal system-from nephrone to usinary bladder. Optimal conditional for such transformation is osmotic pressure of urine between 100 and 200 mosmol/l or density of urine should less than 1010.
If osmotic pressure of urine rises, Sternheims-Malbin cells turn back into the «activ» leukocytes. Finding of «activ» leukocytes (Sternheims-Malbin cells) is one of real laboratory criteria of pyelonephritis, though Sternheims-Malbin cells cannot be absolute sign of pyelonephritis, as they can be found in the other cases as well. (Sometimes in the healtly children).
Epithelium in urine.
With different pathologies the process of epithelium desquamation can quickly grow, that's why quantity of epithelium cells in urine sediments also grows. The process is characteristic for inflammation and degeneration of the renal system, trauma of the mucous membrane by a descending urinary stone or by a tumor.
Kidney epithelium can be found with nephritis and pyelonephritis. Large amount of urinary bladder mucous membrane cells are found with cystitis in the urine sediments.
In the girls vagina and outer genitals epithelium cells can be found in the urine sediments.
Cylinders in the urine sediments look like elongated particles of coagulated albumen of cells. The cylinders are «moulds» of urinary channels. More often the cylinders are formed in the acidous urine, and in the alkaline urine they quickly dissolve and cannot be found.

There are real and false cylinders. Real cylinders are gyalinic, grainy, waxy, epithelial, bloody and cylindroids.
Group of false cylinders includes cylinders consisting of leukocytes, bacteria, urates, fat, amorphous phosphates and mucus.
Conducting clinical estimation of «cylinderuria» it is necessary to bear in mind as follows. Presence of small number gyalinic cylinders in the urine is not a sign of a renal disease, as it can be due to great physical stress, fever, etc, though if there is a great number of the gyalinic cylinders (especially covered with kidney epithelium and with erythrocites) - it is a sure sign of renal pathology.
Grainy cylinders in the urine sediments is a sign of dangerous degenerative processes in the kidney canals.
Waxy cylinders are formed in the canals of relatively big diameters and have yellowish colour. They can be seen with hard kidney affections (often chronic diseases), especially affection of the canals epithelium.
Epithelium cylinders are the reliable symptom of the hard degenerative changes of the kidney canals due to acute diffusive and chronic glomerulonephritis large while kidney. Epithelium cylinders are the layers of the canals' epithelium.
Protheinuria
Renal protheinuria occurs due to: (a) distortion of kidney ball filter and excretion of normal plasma albumin; (b) diminishing of reabsorption of the albumin back onto the canals; (c) excretion of albumin by the canals' epithelium and tubulorrhexis; (d) destruction of kidney epithelium cells.
According to pathogenesis protheinuria can be functional and organic; functional protheinuria drmonstrates only occasional distortion of kidney function , but organic changes are those, which show diffusive or limited pathomorphological changes of kidney parenchyma.
Group of functional protheinuriae includes: physiological protheinuriae of a new-born child, orthostatic protheinuria, one caused by nervous and physical stresses, hyperthermia, exicosis and toxicosis, haemostasis due to cordial diseases, allergy. Functional protheiuria is short-lasting and not vivid. They disappear together with a provoking factor. Organic protheinuriae are long-lasting, with vivid symptoms, have the other pathological signs (leukocyturia, hematuria, etc.), and occur with glomerulonephritis, tuberculosis, polycystosis of kidneys, thrombosis of renal vessels and other organic affection of renal system.
Bacteriuria
Cystitis and pyelitis are characterized by staphylococcosis and streptococcal infection in urine (urine reaction is low acidic, sometimes low alkalotic). Salmonellosis and enteric fever give a lot of bacteria of the kind in urine, petlonephritis gives intestinal bacilli, white or golden staphylococcus, hemolytic streptococcus.
Bacteriouria caused by plasmocoagulating staphylococcus vulgar protheus, entherococcus, green-blue pus basilli is very dangerous.
Measuring of ball filtration allows to indicate the grade of its diminished at pyelonephritis, glomerulonephritis and other renal diseases. Considerable deviation from the norm can be registered at the other diseases (contracted liver, hard toxicosis, blood circulation deficiency, etc).
Results of Zimnitsky probe can be interpreted as follows.

1. Equal volume of separate portions of urine is a sign of lowered accommodation of the kidneys to constantly changing conditions of life and nutrition.
2. High relative density of urine (1025-1030) can be a sign of initial acute glomerulonephritis.
3. When the daytime diuresis is equal to the nightly one or nightly diuresis is greater, it is supposed that concentration ability of kidneys is lowered and possibly the deficiency of blood circulation also takes place.

Biochemical Inxestigation of Blood

Quantity of urea in the blood serum can heighten when lating a lot of albuminous food, liver affection and diseases giving intensive katabolismes of albumin and other pathological processes, though the urea level heightens not for a long time and moderately.
In the patients
With renal deficiency concentration of creatininum in blood serum considerably grows.
Creatininum does not almost reabsorb in the canals and that's why creatininum probe is used to estimate functioning of the balls.
Pathological deviation from the normal concentration of electrolytes take place with chronic renal deficiency, especially at the final stage. The same can be with the acute renal deficiency, happened due to serious affection of kidney parenchyma. Hypernatriemia and hypercaliemia can appear at the polyuretic stage of the acute and chronic renal deficiency, renal diabetes insipidus, that is characterized by the considerable uncompensated loss of water.
Lowering of natrium and calcium concentration, often accompanied with cerebral and cordial disorder can occur due to long-lasting strict diet, vomiting, diarrhea, chronic pyelonephritis or glomerulonephritis with renal deficiency.
Hypophospkatemia is characteristic for renal tubular acydosis with nephrocalcynosis, phosphat-diabetes, hypophospkatemia and oliguria- for the final stage of chronic renal deficiency. Constant hypocalciemia is a cause of osteoporosis and osteodistrophy.

General Albumine and Albumine

Fractures of Blood

Patients, suffering of the acute glomerulonephritis and pyelonephritis show a moderate level of hypoprotheinemia caused by excretion of albumin with urination and the albumine limiting diet.
With nephritic syndrome hypoprotheinemia is clearly seen.
With glomerulonephritis, pyelonephritis and especially nephrotic syndrome disprotheinemia often occurs: quantity of α -2 globulin together with γ - globulin and at the same time quantity of albumin fall down. Albumin/globulin ratio normally is less than 1, and with nephritic syndrome it equals 0,3 or 0,4.

Excretory (Intravein)

Unorgraphy

Reliable symptom of enlarging or contraction of the kidney is its 20% deviation to positive or negative side.
Kidney grows in its size with a tumor, hydronephrosis, polycystosis, urolithiasis; it diminishes with hypoplasia, nephrosclerosis, vessel anomalies.
Obstruction of renal channels with hydronephrosis, renal stone disease bladder- urethric reflex is accompanied with considerable widening of the neck of the pelvic, its enlarging and deformation.

Thin parenchyma of a kidney is characteristic for the secondarily contracted kidney. The shadow of a kidney can be absent in the urogram when it is inborn aplasy of the kidney.
It is important to play attention to the contours of the kidneys. Normally they are smooth, but with polycystosis, sclerosis of kidney parenchyma with its contraction the contours are wary, though in the early age a wavy outline can be due to segmental form of a juvenile kidney.
Excretory urography is used for finding the defects in development of renal system.

Renal Angiography

Angiography allows to find localization of stenosis or occlusion of renal arteria, as well as their stage and length. When there is thrombosis of renal asteria or its branch, the sympthom of stump appears, for the contrast liquid cannot penetrate farther than thrombus.

Mixture cystourethrography
(cystophluorography)

Mixture cystophluorography allows to get precise data on pathomorphlogical changes during urinary bladder and urinary excretion channel disease, especially to diagnose defects of their development, to clear out the cause of the urine excretion from the bladder obstacles, to find out bladder/urethra reflux, etc.

Radioisotopic renography

Normally configuration of the left and the right kidney does not much differ. With clinical estimation of the isotopic renography it is important to pay attention to the following:
The most vivid changes can be seen on the renogramma when only one kidney is affected (hydronephrosis, stenosis of renal asternia, etc.): on the back-ground of the normal kidney renogramma of the ill kidney looks low and delayed in time.
Elongation and flatness of all the segments of renogramma of both kidney can occur due to chronic glomerulonephritis and double-sided pyelonephritis (on the stage of renal deficiency). A «deaf» kidney gives straight line («plateau»), that means there are no or very small excretion of urine from the kidney.

Scanning of the Kidneys

With renal disease, for example, polycystosis on the scanogramme there are signs of the kidney enlarging, their outline is illegible, there are also many defects of filling, coinciding with the cysts. If a kidney is affected with a tumor, it has a distorted form with a defect of filling of the some kidney segment on the scanogramme. «Deaf zones» can be due to a cyst or abscess of a kidney. With chronic glomerulonephritis scanogrammes are symmetric, though even diminishing of a nuclide filling is seen and besides when the disease takes a bad turn, the dimensions of the kidneys decrease. With pyelonephritis the affected kidney decreases its outline is illegible, distribution of nuclide in the parenhyma is uneven and weak.

Syndrome of Acute Renal Deficiency (ARD)

ARD - is a sudden total failure of kidney functioning, accompanied with the water/electrolytic balance and acidic/basic state of blood failure, disarray of the blood pressure regulation, growing of isothermia and development of uremia.

ARD of extrarenal origin:

- shock
- obstruction of urine excretion
- thrombosis of renal vessels

ARD of renal origin:

- acute glomerulonephritis
- hemolytic - ureic syndrome

■ renal cortical necrosis
■ acute tubular necrosis

The main cause of ARD is a temporary ischemia of a kidney, when cortical blood flow is affected.

Clinical development of the disease:

■ initial stage (from some hours to some days)
■ inure or hard oliguria stage (from some hours to 5-7 days)
■ stage of enlarged diuresis with renal function deficiency
■ early polyuretic (about 5 days)
■ late polyuretic (6-9 months)

Initial stage of ARD:

■ shock
■ oliguria
■ dehgdratation (dry skin, thirst, hypotension, heightening of haematocritis)
■ hypercaliemia

Oliguretic stage:

■ inure
■ acidosis
■ azotemia (headache, sleepyness, vomiting, ache in the stomach, diarrhea, liver and spleen enlarged, high temperature)
■ lowering of creatininum and urea in the blood
■ cellular dehydratation and outcellular hyperhydratation

Early polyuretic stage:

-enlargement of urine excretion with low relative density

Late polyuretic stage:

■ excretion of large amount of urea, nitrogen, calcium, magnesium
■ dehydratation with hypernatriemia and hyperchloremia

Rehabilitation of renal function goes very slowly.

Syndrome of Chronic
Renal Deficiency (CRD)

CRD is an irreversible deterioration of homeostatic functions of the kidneys due to hard progressing renal disease.

Causes of CRD:

1. Inborn kidney pathology
2. Glomerulonephritis
3. Pyelonephritis

Clinical signs	Reasons
Delay of growing and development	Lack of albumen, energy, vitamins, acidosis, azotemia
Asthenia, Psycho-neurological diseases	Azotemia, anemia, hypoxia, hypertension
Anorexia	Azotemia, affection of the

	stomach tract
Lack of albumine (hypoprotheinemia, lowering of muscular tissue tonus)	High catabolismus Ration of albumin assimilation
Hypertension	High secretion of renine, deterioration of water/electrolytic
Oedemata	Protheinuria, hypovolemia, hypoprotheinemia
Anemia	Lack of albumine, ferrum, *erythropoetinek*
Hamorrhagic Syndrome	Enlarging of time of bleeding under the influence of urea
Osteodistrophy	Deterioration of active metabolites of vitamine «D» production, lack of Ca^{2+}, hyperparatireoidism
Azotemia	Delay of nitrogenic metabolites in the blood due to phylotration; deterioration of toleration to common albuminous diet
Hypercaliemia	Deterioration of filtration function, high catabolismus, acidosis
Hyponatriemia	Deteriaration of intrarenal transportation of Na^{+}
Acydosis	Deterioration of filtration and aminoacydogenesis

Materials to activate the students during lecture:

Questions :

1. Anatomical and physiological features of urinary system in children.
2. Semiotics of most wide-spread diseases of urinary system in children (pyelonephritis, glomerulonephritis, cystitis etc.).
3. Semiotics of microscopic changes of urinary deposit (proteinuria, erythrocyturia, leucocyturia,cilindruria etc.).

4. Syndrome of acute and chronic renal insufficiency.

Materials of methodical software of the lecture:

- Auditory
- Software
- Illustrations

The materials for self study of students. (Literature for the student.)

o) For the topic of the current lecture
Literature:
1. Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 621 – 670; 779 – 780.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

p) For the topic of the next lecture
Literature:
1. Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – P. 671 – 684; 785 – 787.
2. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.

Literature used by lector during preparation the lecture:

64. Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – 808 pp.
65. Partha,s Fundamentals of Pediatrics. Ajanta offset &Packagings Ltd., New Delhi.-2013.- 782 pp.
66. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.
67. Vicky R. Bowden, Cindy S. Greenberg. Pediatric nursing procedures. - Lippincott Williams & Wilkins. - 2011. - 822 pp.
68. Pediatric Nursing Procedures. Vicky R. Bowden, Cindy S. Greenberg. - Wolters Kluwer Health.- 2015 -728pp.
69. Nelson. Essentials of Pediatrics. Sixth Edition. Canada: 2011. - 831p.
70. Robert M. Kliegman. Nelson Textbook of Pediatrics 19th ed. – W.B. Saunders Company, 2011. – 2680 pp.
71. Pocket Book of Hospital Care for Children. – World Health Organization, 2013. – 438 pp.
72. Nelson Textbook of pediatrics / R. M. Kliegman, B. F. Stanton, J. W. St Geme III, N. F. Schor ; ed. by R. E. Behrman. – 20th ed. – Canada : ELSEVIR, 2016. – 5315 p.

Lecture № 10

Topic: «Anatomical and physiological features of blood system in children of different age groups. Clinical and hematological semiotics of main syndromes (anemic, hemolytic, hemorrhagic etc.) and diseases of blood system in children (anemia, acute and chronic leucosis, hemorrhagic vasculitis, trombocytopoenic purpura and hemophilia etc)» – 2 hours.

The actuality of the topic: Knowledge of Anatomical and physiological features of blood system in children of different age groups, clinical-hematological semiotics of the basic

syndromes (anaemic, hemolytic, haemorrhagic, etc.) and blood diseases in children (anaemia, acute and chronic leukosis, hemorrhagic vasculitis, thrombocytopenic purpura, hemophilia etc), ability to investigate a patient is an important part of diagnostics and treatment. To make the diagnosis should be able to correctly estimate the data during the investigation of a patient, data of laboratory analyses and then to make clinical analyses of the semiotic of diseases.

Aims of the lecture

Practical (training) aims:

- To acquaint students with anatomical and physiological features of blood system in children of different age groups, clinical-hematological semiotics of the basic syndromes (anaemic, hemolytic, haemorrhagic, etc.) and blood diseases in children (anaemia, acute and chronic leukosis, hemorrhagic vasculitis, thrombocytopenic purpura, hemophilia etc),
- to teach the students the methods of investigation and nursing children with blood system diseases.

Educational aims:

- Training students in current clinical thinking,
- Ensuring the development of students leading domestic value of clinical, scientific and educational schools in the development of the problems of the lectures,
- Emphasize the need to fight for further reducing child morbidity and mortality from digestive diseases,
- Skills to teach ethics and medical ethics.

Plan and structure of the lecture :

№№	**The main steps of the lesson**	**The aims in the levels of learning of the material**	**Materials of methodical software**	**Time (in minutes or %) of a hole lesson**
1	**2**	**3**	**4**	**5**
I.	*The preparatory level*	1	The presentation, Auditory	5%
1.	The defining the purpose			
2.	Formation of the positive motivation			
II.	*The basic level.*	*II.*		85-90%
3.	The presentation of the lecture by plan 1. Anatomical and physiological features of blood system in	*II.*		

	children of different age groups. 2. Clinical and hematological semiotics of main syndromes (anemic, hemolytic, hemorrhagic etc.) 2. Clinical and hematological semiotics of main and diseases of blood system in children (anemia, acute and chronic leucosis, hemorrhagic vasculitis, trombocytopoenic purpura and hemophilia etc).	*II*. *II*.	Illustrations Literature, questions, tasks.	
III.	Final level.			5%
4.	The conclusion of the lecture.			
5.	Answering questions.			
6.	The task for self-control			

The content of the lecture

Anatomical and physiological features of blood system in children of different age groups

The following lecture discusses the science of blood – hematology. Haemopoesis is the processes of formation and the following maturation of the formed blood elements in the haemopoietic organs. Three periods of embryonic haemopoeisis are differentiated:

1) embryonic or mesoblastic – duration first 3-5 weeks of gestation;
2) hepatic or extramedullary – duration from the 6^{th} week to 5^{th} month;
3) bone-narrow or medullar – duration from the 4^{th}-5^{th} month of gestation to baby's birth.

All stages of haemopoeisis follow each other gradually, there are no limits of time interval between these periods.

I period. The first focuses of haemopoieisis are found out in the 19^{th} day of the human embryo in the blood islands of the yolk-sac; mesenchymal cells surrounding the embryo start producing the first primitive blood cells – megaloblasts.

It is period of the primitive erythropoiesis. By the end of the 1st month haemopoeisis comes through almost everywhere, extravasculary, but is soon restricted by the liver.

II period. Begins from the 6th week and reaches a maximum by the 5th month. Megaloblasts are gradually replaced by the erythroblasts, forming the first neutrophils and megakaryocytes. By the 3rd-4th month of the embryonic life, haemopoeisis occurs in the spleen, realizing granulocyto-, erythrocyto-, megakaryocytopoiesis. From the 5th month hepatic haemopoeisis gradually decreases and almost completely stops by the birth. Active lymphocytopoiesis occurs a little later in the spleen from the end of the 7th month of gestation.

III period. Starts from the 4th-5th month of the embryonic life and gradually becomes basic. The medullar is the only organ of haemopoeisis up to the birth. The medullar bones are red in the prenatal period.

According to the three periods of haemopoeisis there are three forms of haemoglobin: primitive (embryonic) HbP, fetal HbF and adult HbA.

Primitive haemoglobin is found out in the earliest stages of embryonic development in the megaloblasts (8-10 weeks).

The main differences in the composition of the forming elements of the embryonic blood are following: gradual increasing in the number of erythrocytes, content of haemoglobin, number of leukocytes. In the first half of gestation, the blood contains many unformed elements, while the next half is dominated by the matured forms. A schematic diagram of the stages of embryonic haemopoeisis is shown in dia 1.

Already by the 8-10th week of gestation, HbF consists of 90-95% of the total haemoglobin in the erythrocytes; other 5-10% is HbA.

During birth HbF varies from 45% to 90%, gradually replacing by HbA: by 1 year HbF composes only 15% and by 3rd year - not more than 2%.

These types of haemoglobin differ from each other by the amino-acid composition and functional potentials. HbA and HbF have high affinity for oxygen (are oxyphilic), which is important for the oxygenation of embryonic blood.

By the end of the intrauterine period, haemopoeisis occurs only in the bone medulla.

The bone medulla of the newborn completely fills up all the flat and tubular bones. The bone medulla of the newborn weighs almost 40gms, thus composing almost 1,4% of the total body weight. The bone medulla of the adults weights 3000 gms (5%).

The transformation of the red bone medulla to yellow starts from the 6th month; which increases between ages 4-6. By 12-15 years haemopoeisis occurs in the bone medulla of the flat bones, ribs, corpuses vertebrae, and proximal parts of the tubular bones (like in adults).

Differentiation of blood cells goes in the following phases. Each following phase of maturation decreases the rate of universality of the cells and its ability for self supporting.

The following phase of differentiation in the line of mylopoiesis is arising of the cells-predecessors of myeloid haemopoeisis. This forms a row of bio-potent cells: predecessors of granulomono-, granuloerythro-, erythromegakaryocytopoiesis. After this process unipotent cells are formed: granulocyto-, eosino-, basophilopoiesis and corrulent cells, erythropoiesis, megakaryocytopoiesis.

In the last stages morphological differences on the myelogramme interspaces and matured cells occur in all phases of bone medullar haemopoeisis.

In lymphoid haemopoeisis we can't distinguish generic cells for T- and B-lymphopoesis with the following universal differentiation: there are predecessor cells or pre-B- and pre-T-

lymphocytes, early B- and T-lymphocytes and mature B- and T-lymphocytes.

The intensity of formation of all cells is determined by the action of humoral regulators: stimulators (poietins) or inhibitors.

Leukopoietic function is carried out by different colonystimulating factors. Inhibitors of granulocytopoiesis are lactoferin and prostaglandins.

Stimulators of erythrocytopoeisis are erythropoietin and burstobrazic factor; for thrombocytes-thrombopoietin; for T-lymphocytes- thymosin and T-growing factor.

All phagocytes of the organism are the derivatives of the hemapoitic cells and are descendants of monocytes.

The younger the child's age, the more functional liability and running down of bone medulla. Some unfavourable factors (infection, intoxication, anaemia, leukosis, etc.) in babies, blood lead to return of the embryonic type of haemopoeisis.

The composition of the peripheral blood in children, from the first day of life undergoes considerable changes.

On the first day of life in newborn there is more haemoglobin (180-240gms/l) and erythrocytes (7,2-5,38 T/l) because of placental transfusion and haemoconcentration. Already by the end of the first day this amount decrease, which by the 7-10th day reach normal smallest data. Colour index in the first day of life is about 1-1,3.

Qualitative differences of a newborn is as follows:

1) anisocytosis – increasing of erythrocytes size like megalocytes (13-15mk) macrocytes (8-12mk) or decreasing to microcytes(4-6mk);

2) polychromatophilia (polychromasia) – deviation from the normal staining of erythrocytes. Basophilic erythrocytes appear, which loose the ability to stain with eosin but stain with methyl blue to blue colour;

3) poikilocytosis – deviation from the normal forms of erythrocytes (pear shaped, semicircular).

Appearance of normoblasts (young, immature erythrocytes) – polychromatophils and reticulocytes (8-13% to 42% in the blood of a newborn) with moderate anisocytes; they reflect increasing of the haemopoietic activity of the bone medulla in a newborn.

Change of the form and staining of the red blood bodies to microcytes, anisochromia are signs of depressing function of the bone medulla, a sign of degeneration.

Usually, the red blood of a new- born represents normocytes, and within some days may appear erythroblasts; but they appear in single forms.

The peripheral blood is composed of big number of erythrocytes and haemoglobin, young and immature forms that testify about the intensive haemopoeisis and underlines the increased necessity for oxygen in newborns. Embryonic erythrocytes are prone to haemolysis and live for a short time (up to 12 days), which is 5-6 times less in children than in adults.

There is a difference in the erythron composition (system: erythrocytes and reticulocytes of blood + erythrocytic shoot in a haemopoietic apparatus) of newborn and children. As soon as external breathing is formed, hypoxemia changes to hyperoxemia and the intensity of erythropoiesis decreases. The peripheral blood of the 10 days newborn contains the same number of erythrocytes and haemoglobin, as in adults.

Blood groups.

In the present time antigens of erythrocytes unite the 15 systems of blood groups. Antigens of erythrocytes, combining within the groups and also between groups, form hundreds of variants. There are no two people with the same erythrocytic antigens. For

practical purposes, we use system ABO, Rh –(+), MN (SS). In ontogenesis antigens A and B form in the embryonic erythrocytes by 2-3 mths., but reach the greatest activity by 10-20 years.

Agglutination of erythrocytes in newborn is 1/5 th of adults one. Antigens R and H form only after birth; and are activated by one year old like adults.

Antigens M and N appear by the end of the 3rd month of intrauterine life, and quickly develop fully by 5^{th} month of embryogenesis.

The Rhesus antigen system forms by the 8^{th}-10^{th} week of intrauterine life.

Peculiarities of white blood.

In the peripheral blood of a newborn during 5 days of life the number of leukocytes is 18-$20*10^9$/l, neutrophils compose 60-70% of white blood (like in adults), the leukocyte formula shifts to the left because of the increased number of stab neutrophil and juvenile forms, may appear an isolated myelocyte, especially in the premature newborn.

The changes of the leukocytic formula start from the first day of life: the number of neutrophils decreases while the number of lymphocytes increases. On the 5^{th} day of life their number becomes equal (decussation or first cross-over) and composes 40-44%. Subsequently the number of lymphocytes increases; which by the 10^{th} day composes 55-60%, while at the same time the number of neutrophils decreases up to 30%. Gradually the formula shift to the left dissapears (stab neutrophil – 3%, juvenile – not more than 1%, myelocytes fully disappears from the blood).

This special characteristic correlation between neutrophils and lymphocytes is kept during the first year of life, subsequently the number of neutrophils again gradually increases, while the number of lymphocytes - decreases. At 4-5 years a second crossover takes place in the leukocyte formula (the number of neutrophils equals lymphocytes).

From 5 years the number of lymphocytes decreases and neutrophils - increases and by 10-12 years the leukocyte formula in children is like in adults. Number of thrombocytes in newborn is 150-400 g/l, anisocytosis is possible

The duration of bleeding by Duke's method is 2-4 mint.

The coagulation period in newborns may be prolonged, especially during jaundice.

The physio-chemical and morphological features of blood in children change according to age, sexual and constitutional peculiarities.

The blood volume and its parts in children isn’t constant. Apart of the circulating blood, there is the depot in the liver, spleen, sub-cutaneus capillaries, plexus etc.

Haematocrit in newborns is 54%, in children 39%.

ESR according to Panchenkov's method: in a newborn is 2mm/hr, 1-12mths. – 4,8 mm/hr, in children – 4-10mm/hr.

Information about coagulating blood system.

The process of haemostasis is supported by three links: vascular, plasmal and thrombocytic.

The vascular haemostatic link by birth is well developed morphologically. The increased brittleness and permeability of the capillaries and decreased contracting ability of the pre-capillaries is characteristic of babies.

The plasmal haemostatic link (the different coagulating factors) differs by the following specialities:

1. Contains the V, VIII, XIII factors in newborns are active like in adults.
2. Activity of vit. K-depending factors on the first day of life, II factor (prothrombin), VII

(proconvertin), IX (anti-haemophilic globulin B), X (Stuart -Prower factor) and XI + XII (contact factors) decreased. After the 3rd day of life; the synthesis of vit. K in the intestinal flora and the protein-synthesis function of the hepatocytes increases and possibility of thrombosis disappears.

The thrombocyte activity decreases because of functional immaturity of the thrombocytes (ability of aggregation decreases). Quantity of thrombocytes is the same as in adults.

Specialities of the anti-coagulating system of blood.

Activity of this system in children is not well learnt. In newborns level of heparin and fibrinolitic activity of the blood are increased.

Activity of the tissue and plasmatic anti-thromboplastin, antithrombin III, anti-activating XI and X factors, plasminogen are decreased.

The decreased or low activity of almost all factors of coagulation in newborns is a physiological phenomenon, protecting the newborn from thrombosis, which may appear with tissue injury (especially of the brain) during labour and entering of the tissue thromboplastin in the blood.

By the end of the first year, exponents of the coagulating and anti-coagulating system becomes like adults till period of hormonal reorganization (pre- and pubertal age).

Haemopoietic pecularities of immature newborn.

The sources of extra-medullar haemopoesis in liver and spleen continue in immature newborn.

Red blood cells are characterized by increased number of normocytes (juvenile form of erythrocytes containing nucleus), quantity of HbF more, than in mature newborns.

These higher indexes of red blood cells decrease quickly, not gradually, which lead to anaemia by 1,5-2 months because of fast increasing volume of blood and weight of the body and insufficient formation of erythrocytes. By 4-5 months the concentration of Hb starts decreasing because of iron deficit.

White blood cells are characterized by the increased concentration of juvenile cells.

Clinical-hematological semiotics of the basic syndromes (anaemic, hemolytic, haemorrhagic, etc.) and blood diseases in children (anaemia, acute and chronic leukosis, hemorrhagic vasculitis, thrombocytopenic purpura, hemophilia etc)

General complaints.

Weakness, fatigue, dizziness, dyspnea after physical exercises, palpitation, increasing fragility of the hair and nails can be an appearance of anemia, leukosis, aplasia of haemopoesis.

Itchy skin can be a manifestation of lymphogranulomatosis, erythremia, and chronic leukosis.

Temperature increasing: anemia, leukosis, and lymphogranulomatosis.

Loss of appetite and growing thin is well expressed in patients with cancer, leykosis, lymphogranulema, and lymphosarcomatosis.

Pathological changes of sense of smell and taste can be a characteristic of iron-deficiency anemia (children start eating clay, chalk, soil etc.)

Pain in flat bones can be a characteristic of leukosis.

Pain in the right and subcostal regions can be a manifestation of hemolytic anemia, cancer of haemopoetic organs.

Anamnesis.

There are two hereditary hematological diseases: hemophilia and thrombocytopathy.

Visual examination.

Pale skin is a characteristic of anemia. “Cherry-like” red skin, more expressed on the neck, face and wrists can be a sign of erythremia.

Different kinds of skin and mucous membrane rash are typical for thrombocytopenic syndrome and hemorrhagic syndrome.

Thrombocytopenic syndrome: it is a characteristic of thrombocytopenic purpura, when a numerous hemorrhagic spots of different size and shapes (from small petechie to large ecchymosis) appear on the skin and mucous membranes.

At first these spots have red color, then it gradually changes to “cherry-like” color, to blue and finally to yellow. This happens because of metabolism of hemoglobin to biliverdin and to bilirubin. After being pressed such spots don’t disappear. As an additional symptom, a blood analysis shows thrombocytopenia.

Hemorrhagic syndrome: it is a characteristic of hemorrhagic vasculitis. It can be noticed by papullar hemorrhagic rash on the external unbended symmetrical surfaces of the joints, rash also can be seen on the buttocks. Papules usually rise above skin level and don’t leave pigmentation after their healing. Blood analysis shows anemia.

Anemic syndrome: there are several symptoms of anemia: pale and dry skin, desquamation of skin epithelium, dry fragile hair, fragile flat nails with transversal folds on them, atrophic glossitis, progressing caries of the teeth, inflammation of mucous membranes around teeth necks, cutaneous and subcutaneous fat edema, signs of growth retardation.

Enlargement of peripheral lymph nodes is a possible symptom of leucosis, lymphogranulomatosis, infectious mononucleosis.

Palpation.

During diseases of blood system and haemopoetic organs lymph nodes enlarge because of increasing mass of myeloid and lymphoid tissue in them. Enlargement of the lymph nodes can be symmetrical, single-sided, isolated or widespread. Lymph nodes are enlarged in case of leucosis, lymphogranulomatosis, and lymphosarcoma.

In case of leucosis peripheral lymph nodes are not connected to the surrounding tissues, they are round, smooth, elastic, painless; skin above them is unchanged, they never form pus.

Lymphogranulomatosis starts with the enlargement of the back neck lymphatic nodes. They are painless, not connected to the surrounding tissues, movable. Skin above them is without visible changes, they don’t form puss. Further metastases appear around the primary focus, in time they join in groups and these conglomerates become motionless.

Lymphosarcoma is characterized by infiltrative growth: lymphatic nodes connect with each other and with the surrounding tissue, lymph nodes become motionless, they are painless and skin above them is unchanged.

Spleen enlarges in case of hemolytic anemia, thrombocytopenic purpura, leucosis. In case of hemolytic anemia and leucosis spleen consistence is thick, its surface is smooth and it is painless.

Liver enlargement is connected with leucosis and lymphogranulomatosis. In these cases front liver edge is elastic, thick, painless, its surface is smooth.

Percussion.

Percussion of the tubular and flat bones reveals pain in case of leucosis, pain can be explained by the growth of tumour in the haemopoetic system.

Semiotics of laboratory methods of diagnostics

Changes of red blood.

Anaemia (decrease of the amount of RBC and hemoglobin in one volumetric unit) can be true and false.

True anaemia can be noticed during hypofunction / hypoplasia of red bone marrow, hematological organs' tumour, increase of erythrocyte destruction. Combination of anemia with increased blood levels of bile pigments can be a sign of increasing RBC destruction (haemolysis).

False anaemia can be seen as paleness of skin, that doesn't depend of erythrocyte and haemoglobin levels. False anaemia can be considered in children with capillaries, localized deep in the skin and in case of vegetative blood vessel distony.

Hyper chromic erythrocytes (CI>1) is a sign of hemolytic and B_{12} deficient anemia.

Normochromic erythrocytes (CI=0,8-1,0) is a characteristic of early physiological anaemia, acute posthaemorrhagic anaemia, hypoplastic anaemia.

Hypochromic anaemia (CI < 0,8) is noticed during chronic blood loss and it is a result of iron, protein and protoporphirin deficiency.

Anisocytosis is appearance differently sized erythrocytes in blood circulation. Macrocytes can be seen in newborns, microcytes can be found during anemia.

Poikilocytosis – presence differently shaped erythrocytes (oval, pear-shaped) in blood. In general, poikilocytosis is a sign of erythrocyte degeneration.

Basophilic granules appear as a result of coagulation of basophilic stroma on polychromatofilic erythrocytes, it happens in case of anaemia, malaria, congenital syphilis, mercury and lead intoxication. Erythrocytes with nucleus remnants (Jollie particles, Kabo circles) indicate acute anaemia.

Presence of megaloblasts indicates the embryonic erythropoetic cycle. Megaloblasts can appear in children in case of alimentary and infectious anemia.

Erythroblasts and normoblasts are immature nucleated forms of erythrocytes. In small numbers they can be found in newborn's blood. Their appearance in blood of elder children can indicate red bone marrow hyperfunction due to specific pathological processes.

Polychromatophils appear in peripheral blood in case of increased production of blood elements.

Reticulocytes are young RBC forms, their presence indicates the regeneration process in red bone marrow. If red bone marrow erythropoietin is suppressed, reticulocyte number decreases, in case of red bone marrow hyper function their number increases.

Increased number of erythrocytes (polyglobulia) can be either true or false.

True polyglobulia is normal in newborns, but can be noticed in several other cases: in infants with congenital "blue type" heart defects, after living in high altitude areas, during polycytemia, in case of tumour metastases in bone marrow, or can be a hereditary character.

Increase of erythrocyte number is followed by quick growth of myeloid tissue in bone marrow, liver and spleen. In children it is seen by hyperemia of the skin, mucous membranes have cherry-like or bluish tint, children complain of headaches and dizziness.

False and temporary polyglobulia is a result of blood clotting because of considerable loss of liquid as a result of toxic dyspepsia , dysentery or other intestinal infections; because of increased sweating or insufficient liquids uptake.

Changes of white blood.

Leucocytosis is an increase of leucocyte number. Physiological leucocytosis can be seen in newborns. Pathological leucocytosis can appear because of local or general infections, leucosis, leucemoidal reactions.

False leucocytosis is seen because of blood density increase, unequal blood distribution in capillaries because of crying, heat effects or eating (digestive leucocytosis).

Neutrophiliosis can be a result of child's crying, emotional stress, psychological overload, and physical effects.

Pathological neutrophiliosis appears during most part of infectious diseases, pus formation, sepsis, pneumonia.

Appearance of large number of young leucocyte forms (blast cells) in peripheral blood can be a sign of acute leucosis, large number of undifferentiated cells is a sign of a chronic one. Besides the general amount of leucocytes a great role plays evaluation of the neutrophils' nuclear shift. Left shift (increase of the number of young forms) indicates acceleration of leucocytes' formation.

Lymphocytosis is physiological in early childhood.

Pathological increase of lymphocyte number is characteristic for rubella and whooping cough. Also it can be seen during infectious mononucleosis, typhoid fever (in the peak period and reconvalescence period), lymphoblastic leucosis.

Lymphocytosis along with leucopoenia is seen during acute viral respiratory infections. Monocytosis is a sign of malaria, measles, tuberculosis. Monocytosis together with lymphocytosis is characteristic for infectious mononucleosis.

Eosinophilia is a sign of allergic diathesis, serum disease, bronchial asthma, anaphylaxis, scarlet fever, leucosis, worm invasion. Eosinophilia during acute infectious process can be considered a good prognostic sign.

Basophilia is seen in case of chronic leucosis, lymphogranulomatosis, carcinogenic anemia (malignant anemia), polycytemia.

Leucopoenia is a specific symptom for typhoid fever in the beginning of reconvalescence phase, measles, rubella in rash stage, AVRI (Acute viral respiratory infection). False leucopoenia occurs because of unequal blood distribution in capillaries, because of overall temperature fall etc.

Neutropenia follows leucopoenia and indicates a complex development of sepsis and several other diseases.

Lymphopenia is noticed in some infectious diseases, followed by neutrophilic leucocytosis. Absolute lymphopenia is a sign of lymphogranulomatosis.

Monocytopenia is seen during complex septic infectious processes, lymphadenitis, leucosis.

Eosinopenia is a sign of typhoid fever, measles, pneumonia, sepsis, tuberculosis, rheumatic fever, change of chronic diseases to acute ones in the peak of the pathological process.

Aneosinophilia is an indication of a hard process and is connected with malaria and leishmaniosis. Qualitative changes of white blood (like toxic leucocyte granularity) are triggered by toxins, which change chemical processes of the cells.

Appearance of plasmocytes indicates irritation of haemopoetic organs and happens in infants with measles, rubella, pneumonia, meningitis.

Thrombocytosis is a typical phenomenon for pneumonia and rheumatism.

Thrombocytopenia is a sign of acute anemia, leucosis, thrombocytopenic purpura.

Erythrocyte sedimentation rate (ESR) increases because of acute infectious processes, anemia, malignant formations. ESR can remain unchanged in whooping cough.

Low ESR together with worsening patient's state is a bad prognostic sign.

Myelogram.

Examination of red bone marrow puncture material plays a significant role in correct evaluation of haemopoesis state. Decrease in erythroid cells' level is a sign of hypo- and aplastic anemia, its increase characterizes high regeneration rate that also indicates anemia.

Evaluation of different WBCs' levels is also of great importance. Significant increase of myeloid cells with blast cells prevailing is seen during myeloleucosis. This is accompanied by depression of erythroid and lymphomonocytic growth.

Bone marrow puncture reveals infectious agents of malaria, leushmaniosis, etc.

Semiotics of the blood clotting system.

Changes of blood clotting system factors are often seen in grout of hemorrhagic diatheses, to which following states are related: hemorrhagic vasculitis, thrombocytopenic purpura, hemophilia.

Time of blood clotting is prolonged in case of increased anticoagulation blood activity or decrease of procoagulants' concentration. Time of blood clotting can be decreased because of raised thrombus formation tendency.

The longest period of time (up to several hours) is needed for blood clotting in case of hemophilia A.

The duration of bleeding increases in case of thrombocytopenic purpura. In this case the retraction of blood clot is also changed.

Capillaries' permeability increases in case of hemorrhagic vasculitis, Randew-Ossler disease.

Hematological syndromes.

Syndrome of anemia.

Anemia is decreased peripheral blood level of hemoglobin (less then 110g/l) and erythrocytes (less then 4,0T/l).

Anemic hypoxia is characterized by pale skin and mucous membranes, loss of appetite and activity, weight loss, sleep disturbance, dizziness, noise in the ears, emotional lability, decreased performance in school studying.

Sideropenia can be characterized by dry skin and mucous membranes, fragility of hair and nails, angular stomatitis, picochlorotics (distorted appetite – child eats chalk, clay, soil etc.), pagophagy (eating of cold food, such as ice, icicles etc).

Hemogramm:

– decreased hemoglobin level (less than 110g/l for 6 year-old children and younger and less than 120g/l for children elder than 6 years);

– hypochromia of erythrocytes (CI – color index - less than 0,8);

– anisocytosis;

– microcytosis;

– poikilocytosis;

– increased common iron-linking ability (CILAS) and latent iron-linking ability of blood serum;

– lowered amount of serum iron (less then 12-14 mkm/l);

– lowered coefficient of saturation of transferring by iron (less then 17%);

$$K = \frac{serum\ iron}{CILAS} \times 100\%$$

Hemorrhagic syndrome.

It is an increased hemorrhage frequency that can be seen by nose bleeding, bleeding from mucous membranes, appearance of skin and joint hemorrhages and intestinal hemorrhages.

There are five main types of bleeding:

1. Hematomic type – general hemorrhages, profuse bleeding – traumatic, postoperational, rarely spontaneous, non adequate to trauma. Typical for hemophilia A and B.

2. Microcirculatory type, which is characterized by petechia-macular rash, that appears as:

- petechia, ecchymosis on skin and mucous membranes;
- localized chaotically, look like "leopard skin";
- have no definite localization;
- doesn't rise above the skin level;
- disappears after being pressed on;
- doesn't desquamate;
- doesn't leave pigmentation;
- during healing process color changes from purple to yellowish-green;
- possible spontaneous nose or gum bleeding.

Typical for thrombocytopenia, thrombocytopathy, hypo- and disfibrinogenemia, deficiency of X, V, II factors of blood coagulation system.

3. Microcirculatory-hematomic (mixed) type combines two types listed before. Is common for Willerandt disease, Willerandt-Jurgenc syndrome, DIC syndrome, overdose of anticoagulants.

4. Vasculitic-purpuric type develops because of eccudative–inflammatory phenomenon in microvessels as a result of immune-allergic and infectious toxic reactions.

Rash is characterized by papillo-haemorrhagic type, located on the extense surface of big joints, on buttocks, its symmetric, rises above the skin level, doesn't disappear after being pressed, doesn't desquamate, leaves temporary pigmentation, can be accompanied by pain in abdomen and lumbar region.

Typical for hemorrhagic vasculitis (Schönlein-Genoch disease).

5. Angiomatous type is characterized by different forms of telangiectasia (Rendue-Osler disease). Main symptoms are: frequent bleeding from nose, gums, lips, digestive tract and respiratory tract.

Care of children with blood system diseases.

Children with increased haemorrhages, depending on its etiology, should be strictly in bed, in regular nurses control. Feeding in the room, transport to the investigation cabinet in a stretcher, should themselves do the toiletteries and move around in the wards only after the doctors instructions, not to use sharp, rusted, cutting instruments, which may cause trauma and fail.

Pre-medical help during haemorrhage.

During nasal haemorrhage (after trauma, during Verlgoffs disease, haemorrhagic vasculitis, haemophilia, leukosis, hypovitaminosis C, K, during measles, whooping cough, influenza, sepsis, polyps, adenoids, foreign body, hypertonic crisis (it is necessary to carry the child, make him sit, support the head, on the nostrils apply ice or gauze, douched in cold water. May press the nose against the nostrils (for 3-5 min), breathing should be done

through the mouth.

In the absence of any effect, it is necessary to tampon the nasal cavity: keep cotton tampons about 50 cm, douched in hydrogen peroxide or thrombin. On the occipital put the bladder with ice, in case of absence of any effect is given calcium chloride, vit. C and K. The tampon inside the nose should not be kept for more than 2 days after its removal, without fall grease the nose with oil.

During haemorrhage after removing teeth, press a tampon which the patient lightly presses with his teeth. Eat approximately after 2 hrs, use cold semi-liquid food.

During haemorrhage from the ears: cotton is to be placed in the ear. Bandage is to be placed on the top.

During pulmonnal haemorrhage (vomit of blood) is necessary: to calm the patient, place in half-sitting position, without unnecessary exercise, give oxygen. With ice, give medicine to lessen cough, it's necessary to treat the main process in the lungs.

Fresh, red blood appears in the faeces during haemorrhage of the large intestine, black faeces (melena) and coffee-ground vomiting appears during haemorrhage from the stomach and small intestine. The child should be in bed, should be calm, lie on the back, bladder with ice is to be placed on the upper part of the abdomen, small pieces of fresh ice may be given, and no food for the first 24 hrs. Then later is given cold and liquid intake (milk, cream, egg white, oil, vegetable puree with minced meat or fish), may given after 2-3 days − diet No A. The child should not get up, should use a “pot”.

During uteral haemorrhage: strict bed rest, cold on the lower part of the abdomen, regularly clean the child.

During renal haemorrhage; strict bed rest, check on the urine colour (should urinate in a urinary vessels).

During hematoma and haemorrhage in joints: strict bed rest, use stretcher, immobilize the joint or extremity (shaft, lancet, plaster).

Care during anemia: long stays in fresh air, full diet with vitamins, microelements (fresh fruits, vegetables, meat and liver) and iron tablets.

In case of any haemorrhage it is necessary to take medical help (after pre-medical help).

Materials to activate the students during lecture:

Questions :

1. Anatomical and physiological features of blood system in children of different age groups.
2. Clinical and hematological semiotics of main syndromes (anemic, hemolytic, hemorrhagic etc.) in children.
3. Clinical and hematological semiotics of main diseases of blood system in children (anemia, acute and chronic leucosis, hemorrhagic vasculitis, trombocytopoenic purpura and hemophilia etc).

Materials of methodical software of the lecture:

- Auditory
- Software
- Illustrations

The materials for self study of students.

8. Literature used by lector during preparation the lecture:

73. Kapitan T. Propaedeutics of children's diseases and nursing of the child: Textbook for the students of higher medical educational institutions. – Vinnitsa: The State Cartographical Factory, 2010. – 808 pp.
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75. Pediatric Physical examination/ Karen G. Duderstadt.- 2nd ed.- 2014.- 366 pp.
76. Vicky R. Bowden, Cindy S. Greenberg. Pediatric nursing procedures. - Lippincott Williams & Wilkins. - 2011. - 822 pp.
77. Pediatric Nursing Procedures. Vicky R. Bowden, Cindy S. Greenberg. - Wolters Kluwer Health.- 2015 -728pp.
78. Nelson. Essentials of Pediatrics. Sixth Edition. Canada: 2011. - 831p.
79. Robert M. Kliegman. Nelson Textbook of Pediatrics 19th ed. – W.B. Saunders Company, 2011. – 2680 pp.
80. Pocket Book of Hospital Care for Children. – World Health Organization, 2013. – 438 pp.
81. Nelson Textbook of pediatrics / R. M. Kliegman, B. F. Stanton, J. W. St Geme III, N. F. Schor ; ed. by R. E. Behrman. – 20th ed. – Canada : ELSEVIR, 2016. – 5315 p.